Chapter 3
Acrylamide

Yuan Yuan and Fang Chen

3.1 Occurrence, Exposure Assessments, and Toxicity

3.1.1 Occurrence in Foods

Acrylamide (AA), $CH_2CHCONH_2$ (Fig. 3.1), is a low-molecular-weight vinylic compound with a molecular weight of 71.09 g/mol. AA is a chemical monomer that is widely used in polyacrylamide synthesis and a variety of other chemicals in industry. AA has been classified as "probably carcinogenic to humans" by the International Agency for Research on Cancer on the basis of sufficient evidence for carcinogenicity in experimental animals and mechanistic considerations [1].

The Swedish National Food Administration (SNFA) and the University of Stockholm reported that AA was present in wide varieties of consumed foods for the first time in 2002 [2]. After that, several countries had analyzed the AA contents in foods and verified the results found by Swedish scientists. Since 2003, data on the occurrence of AA in food commodities had been submitted to the Joint Research Center (JCR) of the European Commission by member states both from authorities and food industries [3]. The Food and Drug Administration (FDA) published data about AA concentrations in foods and confirmed that AA concentration in foods was in the range of 0–2510 μg/kg in 2004 [4]. Food categories that contained higher levels of AA were listed as follows: potato products (French fries, oven-baked chips, potato crisps), different cereal-based foods such as breakfast cereals, cookies, bis-

Y. Yuan
College of Food Science and Engineering, Jilin University, Changchun, China

F. Chen (✉)
College of Food Science and Nutritional Engineering, China Agricultural University, Beijing, China

S. Wang (ed.), *Chemical Hazards in Thermally-Processed Foods*,
https://doi.org/10.1007/978-981-13-8118-8_3

cuits, bread (especially toasted bread), coffee, and other coffee substitutes, chocolate, baby rusks, and various other baby foods. Table 3.1 showed the mean levels of AA in different food categories from 2010 to 2015 [5].

Fig. 3.1 The structure of AA

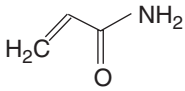


Table 3.1 Distribution of AA in different food categories from 2010 to 2015 by EFSA

Food categories	Mean (μg/kg)
1 Potato fried products (except for potato crisps and snacks)	
French fries and potato fried, fresh or precooked	
1.1 / sold as ready-to-eat	308
1.2 /sold as fresh or precooked, analyzed as sold	367
1.3 /sold as fresh or precooked, prepared as consumed	244.5
1.4 /sold as fresh or precooked, preparation unspecified	368
1.5 Other potato fried products	606
2 Potato crisps and snacks	
2.1 Potato crisps made from fresh potatoes	521
2.2 Potato crisps made from potato dough	327
2.3 Potato crisps unspecified	519
2.4 Potato snack other than potato crisp	283
3 Soft bread	
3.1 Wheat soft bread	38
3.2 Other soft bread	113.5
3.3 Soft bread unspecified	40
4 Breakfast cereals	
4.1 Maize, oat, spelt, barley, and rice-based products	122.5
4.2 Wheat- and rye-based products	160
4.3 Bran products and whole grains cereals	165
4.4 Breakfast cereals, unspecified	124
5 Biscuits, crackers, crisp bread, and similar	
5.1 Crackers	231
5.2 Crisp bread	279
5.3 Biscuits and wafers	201
5.4 Gingerbread	407
6a Coffee (dry)	
6a.1 Roasted coffee (dry)	510
6a.2 Instant coffee (dry)	695
6b Coffee substitutes (dry)	
6b.3 Substitute (dry), based on cereals	510
6b.4 Substitute (dry), based on chicory	2942
6b.5 Substitute (dry), unspecified	415

(continued)

Table 3.1 (continued)

Food categories	Mean (μg/kg)
7 Baby foods, other than processed cereal based	
7.1 Not containing prunes	18
7.2 Containing prunes	107
7.3 Plum content unspecified	33
8 Processed cereal-based baby foods	
8.1 Biscuits and rusks	106
8.2 Other processed cereal-based foods	53.5
8.3 Processed cereal-based foods unspecified	19.5
9 Other products based on potatoes, cereals, and cocoa	
9.1 Porridge	19
9.2 Cake and pastry	66
9.3 Savory snacks other than potato	171
9.4 Other products based on cereals	68
9.5 Other (non-fried) products based on potatoes	108
9.6 Other products based on cocoa	104
10 Other products not based on potatoes, cereals, coffee, and cocoa	
10.1 Roasted nuts and seeds	93
10.2 Black olives in brine	454
10.3 Prunes and dates	89
10.4 Vegetable chips	1846
10.5 Paprika powder	379
10.6 Other	68

3.1.2 Exposure Assessments

JECFA had assessed the AA dietary exposure and the risk of human cancer from the consumption of AA-contaminated foods [6]. The most recent analysis of human food consumption and an AA dietary exposure assessment for eight countries were evaluated at a JECFA meeting held in early 2010. For the general adults, the mean dietary exposures at the national level ranged from 0.2 to 1.0 μg/kg b.w. per day. For adult consumers at the high (95th–97.5th) percentile, the estimates of dietary exposure ranged from 0.6 to 1.8 μg/kg b.w. per day. Based on the few data available for children, it was noted that the dietary exposures of AA were about twice of those for adults when expressed on a body weight basis. The major foods contributing to the total mean dietary exposures for most countries were fried potatoes (in the United States, French fries) (10–60%), potato crisps (in the United States, potato chips) (10–22%), bread and rolls/toast (13–34%), and pastry and sweet biscuits (in the United States, cookies) (10–15%). Generally, other food items contributed less than 10% of the total dietary exposure. Table 3.2 showed the levels of AA exposure in diet (mean) from different regions in recent years.

Table 3.2 Exposure of AA (mean) in diet from different regions and countries

Origin of data	Dietary exposure	Group	References
Poland	0.16 μg/kg b.w./day	Women	[7]
China	0.319 μg/kg b.w./day	Population	[8]
Lebanon (from potato/corn chips)	0.204 μg/kg b.w./day	Population	[9]
Spain (from potato chips)	0.035 μg/kg b.w./day	Population	[10]
Poland	0.09 μg/kg b.w./day 0.13 μg/kg b.w./day	Girls Boys	[11]
Hong Kong	0.213 μg/kg b.w./day	Adults	[12]
Croatia	0.122 μg/kg b.w./day	Female	[13]
Turkey	1.43 μg/kg b.w./day	Toddlers	[14]
Canada	0.29 μg/kg b.w./day	Adolescents	[15]
Western diet	0.441 μg/kg b.w./day	Teenagers	[16]

3.1.3 Toxicity of AA

The metabolic kinetics of AA on human, mouse, and rat had been widely reported in recent years. After digestion and absorption, AA can rapidly distribute throughout the body, which can be detected in most organs, such as thymus, liver, heart, kidney, brain, and even in placenta and breast milk [17–19]. This indicated that AA could pass through the placental barrier, from pregnant mother to the fetus, posing a potential threat to the development. The two main metabolic pathways of AA in the body were shown in Fig. 3.2. Firstly, AA is oxidized to glycidamide (GA) by cytochrome P450 CYP2E1 after the absorption by the body. Secondly, AA can be combined with glutathione and form a copolymerized co-dicarboxylic acid salt. Toxicology experiments showed that 60% of the absorbed AA was excreted from urine, 86% of AA were excreted in the form of thiol and urate, and the unconverted AA accounted for only 4.4% [20]. N-Acetyl-S-(2-ethyl ester)-L-cysteine sulfoxide (AAMA-sulfoxide) was a specific metabolite of the human body and had not been detected in animals. It had been proved to be nephrotoxic and bladder toxic [21]. The study on rodents showed that as a result of the metabolic reactions, AA was transformed to GA, which then induced the formation of DNA adducts, resulting in mutagenesis and the development of cancers [22–24]. Most studies had pointed out that AA was neurotoxic to animals and humans, and AA was also known for its reproductive toxicity, genotoxicity, teratogenicity, mutagenicity, and carcinogenicity in animals.

3.1.3.1 Neurotoxicity

Neurotoxicity was firstly found in highly exposed occupational workers. The main mode of infection was through inhalation of the nose, mouth, and dermal absorption. The main manifestations of infection were ataxia, neuralgia, and numbness of limbs [25]. Early studies reported that peripheral nerve atrophy induced by AA was the

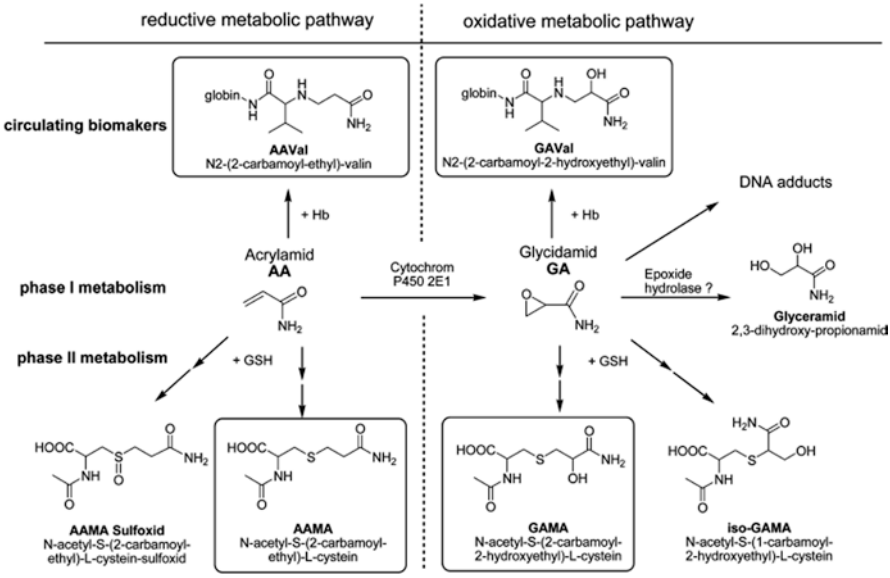


Fig. 3.2 Proposed metabolism of AA

major cause of neurotoxicity. Morphological examination of peripheral nerves revealed that this phenomenon occurred only at low doses of AA [26, 27]. Subsequently, there was a study confirming that AA toxicity also caused peripheral nerve endings by silver staining [28–30]. The combination of morphological, electrophysiological, morphological, and chemical results illustrated that toxicity of AA would damage the nerve transmission. As nerve endings were the primary targets of AA, it would result in damages to the central and peripheral nervous system synapses in a series [31]. With the development of mass spectrometry and nuclear magnetic resonance technology, AA was regarded as an electrophilic reagent, and it was easy to react with DNA, protein, and other macromolecules to form adducts. For example, toxicity induced by AA can attack the residue Cys342, thereby inhibiting the Na^+-dependent presynaptic dopamine transporter function [32]; tandem mass spectrometry results showed that AA at low doses could react with the active site of GADPH and Cys152, while AA at high doses would add a site to Cys156 and Cys247 to inhibit the normal physiological role [33]. As a typical 2-olefin structure, AA can easily perform soft ionization and attack sulfhydryl groups in proteins and DNA. The attack of AA on some proteins can cause transmission disorders, which resulted in significant damages of ending synaptosome [32]. The nerve ending without transmission and translation ability would not have self-protection function. Therefore, it needed other cells to help to achieve its own regulation, and the injured part was difficult to be repaired. This caused insufficient signal supply and led to neurotoxicity [34–36]. Although the maximum dose of AA to cause neurotoxicity was found to be between 0.2 and 10 mg/kg b.w. in the rat model, there was no significant neurotoxicity observed below this range of doses [6]. However, some researchers had pointed

out that neurotoxicity induced by AA might have chronic accumulation effect. Although the average daily exposure amount was far below the maximum dose which had no effects, the risk of accumulation of AA due to long-term dietary exposure to high doses of it still should not be ignored [25]. 2-Olefins at low doses may contribute to the development of stroke, atherosclerosis, Alzheimer's disease, diabetes mellitus, and spinal cord injuries because of the intrinsic oxidative stress [37–41]. Further studies had shown that oxidative stress would lead to highly active 2-olefin derivatives, such as acrolein and HNE (Hydroxynonenal), which might be an important pathological progression of Alzheimer's disease [42–44]. And in Alzheimer's disease, the initial manifestation of pathophysiology was the damage of nerve endings, which occured prior to neurodegeneration and was similar to what AA was induced in animal models [45–47]. Based on a common structure and a similar toxic phenotype, oxidative damage caused by AA may be a potential cause of neurotoxicity and should be paid attention to.

3.1.3.2 Hepatotoxicity

The liver lesion can lead to metabolic detoxification, which causes systemic reactions, and is one of the major target organs that AA attacked after entering the body [48]. As the primary metabolic organ, metabolic activity in the liver was active after exposing to AA. Zhang L et al. reported that AA at 50 mg/kg b.w. could induce liver damage in mice by inducing excessive production of ROS (Reactive oxidative species), consuming GSH and breaking the redox balance [49]. Similarly, Sen et al. also pointed out that 2.5 mM AA on liver cell model for 24 h significantly increased the expression of GST and cytochrome P450-related genes such as deethylase, demethylase, and hydroxylase as well as the protein levels [50]. And a higher dose of AA could cause the death of liver cells [50]. Zhao M et al. reported that 50 mg/kg b.w. AA could cause DNA damage in liver cells of mice and lead to excessive ROS production. The activities of antioxidant reductase GST, GPx (glutathione peroxidase), and SOD (superoxide dismutase) were also changed, leading to break liver cellular redox balance and liver injuries [51]. AA also produced an electron leak by altering the enzyme activity in mitochondrial complexes, stimulating excessive amounts of ROS, and causing damage to liver mitochondrial function [52]. Liver injuries induced by AA were mainly caused by excessive ROS production, resulting in redox imbalance, but the pathway and mechanism on liver toxicity between ROS and AA still needed further investigation.

3.1.3.3 Carcinogenicity

AA had been shown to cause carcinogenicity in several organs in animals and caused tumor formation in the lungs, uterus, dermis, breast, and brain tissues [53]. Studies were carried out on F344 rats and B6C3F1 mice to study the carcinogenicity induced by AA. The results suggested that AA might cause breast fibroadenoma or malig-

nancy in female rats and lead to the formation of the scrotum sheath tumor and the thyroid adenoma in male rats. Conversely, no tumor was found in the target organs of the AA, including the livers, kidneys, lungs, and lacrimal glands [54–56]. Most studies had suggested that the carcinogenicity of AA was caused by the metabolic product GA in vivo. In vivo and in vitro studies illustrated that GA was more mutagenic and genotoxic than AA. Adducts formed by DNA and AA, GA were considered to be the key factor to induce carcinogenesis [57, 58]. Studies had suggested that the combination of AA and GSH led to the depletion of glutathione in vivo, breaking the original redox balance as well as resulting in oxidative stress and thereby affecting gene expression [59]. It was also reported that AA/GA inhibited mitotic or meiotic regulating proteins, and the disruption induced by this may be the cause of genotoxicity and carcinogenicity [60, 61]. In addition, the abnormal hormone levels induced by AA are also a possible reason for gonadal cancer and thyroid cancer [6]. Many references linked the carcinogenicity of AA with its genetic damage and genotoxicity. However, the exact relationship between genotoxicity and carcinogenicity induced by AA had not been established. The United States National Cancer Research Center (NCTR) treated newborn mice and rats with drinking water containing AA/GA for a period of 2 years. The results showed that in the carcinogenic mechanism induced by AA, GA contributed to the distortion in DNA directly [56]. At the same time, AA could cause tumors in a variety of different sites, and there was specificity related to species in mice and rats. Moreover, researchers had found tumors in neonatal mice, which might be related to the expression of cytochrome P450 2E1, a key enzyme in AA transformation to GA, in early embryonic development. Therefore, the harm of AA to infants was worrisome [56].

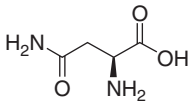
3.2 Formation Mechanisms

In 2002, two articles had been published in *Nature* about AA formation mechanisms. The Maillard reaction between asparagine (Asn) and reducing sugar, represented by glucose (Glc) and fructose (Fru), is the main route for AA formation [62, 63]. In addition, the triacylglycerol oxidation pathway of acrolein can also form small amounts of AA.

3.2.1 Asparagine Pathway

The Maillard reaction is considered as the main AA formation pathway. Stadler and Mottram [62, 63] heated individual amino acids at a certain time above 100 °C, and only a small number of amino acids (such as Asn, glutamic acid, tryptophan, and methionine) produced a small amount of AA. When Asn and reducing sugar were mixed, the amount of AA formed was 500 times higher than that of amino acids heated alone. Zyzak et al. [64] proved that nitrogen atom and carbon atom of AA

Fig. 3.3 Asn structure



from Asn by isotope tracing method (^{15}N and ^{13}C). Asn can be directly converted to AA by the removal of carboxyl and amino group under heating conditions without the addition for reducing sugars from the structure of Asn (Fig. 3.3). However, the results obtained in actual experiments showed that the pyrolysis products of Asn were mainly maleimide, and the reasons were owing to the rapid intramolecular cyclization of Asn molecules under heating conditions, thus inhibiting the formation of AA [65]. While in the presence of reducing sugars, after the formation of a reaction of N-glycosylated intermediates by Asn and reducing sugar, the energy of the decarboxylation pathway was reduced, thereby bypassing intramolecular cyclization and promoting the generation of AA [65].

The beginning of the asparagine pathway was the initial stage of the Maillard reaction (Fig. 3.4). When the Schiff base intermediate was formed in equilibrium with the N-amino acids, two different reaction routes led to the formation of AA.

The first pathway for AA formation was the N-glycosidic pathway (Fig. 3.5). Under the neutral condition, Schiff base intermediates formed by Asn and Glc generate oxazolidinone intermediate by intramolecular cyclization. This intermediate can decarboxylate to Azomethine ylide even at room temperature [66, 67]. It could also form the decarboxylation of Amadori products, and then the C-N bonds were cleavaged to generate AA. The limited step of this process was the formation of AA between the C-N bonds through the elimination reaction [65]. Under the alkaline conditions, Schiff base can also be directly decarboxylated to decarboxylation Schiff base. The decarboxylation Schiff base and the Amadori product directly generated AA or by 3-amino amide pathway. In the N-glycoside pathway, the 3-amino amide was a key intermediate for the formation of AA [64, 68]. Since this decarboxylate reaction was a mild reaction, it could occur at room temperature, and more AA could be produced as compared to the Strecker pathway [69].

The second pathway for AA formation was the Strecker pathway (Fig. 3.6). Schiff base generated by Asn and Glc would produce Amadori products and then dehydrate ammoniagenesis. Asn could be degraded to AA by Strecker degradation in the presence of carbonyl molecule [62]. This pathway suggested that two carbonyl compounds played a crucial role in the formation of AA.

3.2.2 Acrolein Pathway

In addition to the asparagine pathway, AA can also be produced by acrolein or acrylic acid in the high-fat systems (Fig. 3.7) [70]. Yasuhara [71] found that Asn and three oleic acid glycerin could generate more AA after mixing and heating in the model systems. Ammonia and acrolein were heated at 180 °C to produce a consider-

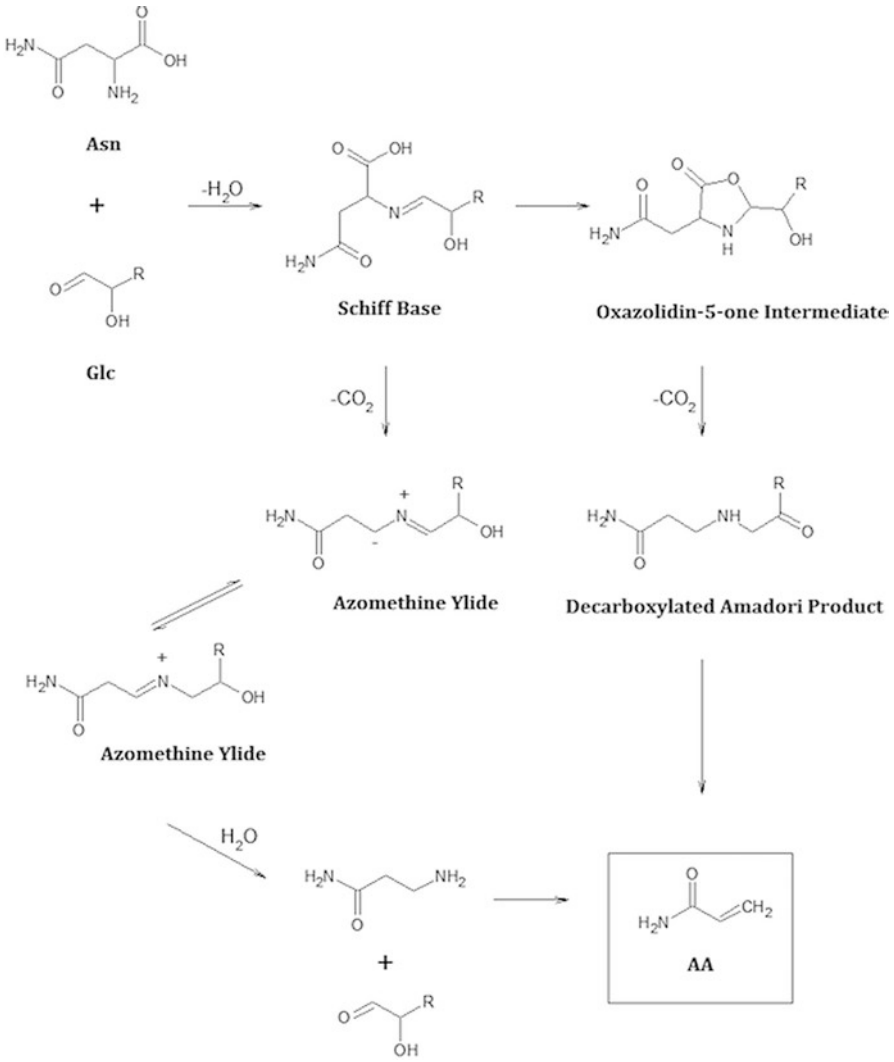


Fig. 3.4 Formation mechanism of AA via the asparagine pathway

able amount of AA. When ammonia and acrylic acid react under the same conditions, more AA would be produced. The study also found that ammonia and acrolein could react at various temperatures, and even at room temperature. The findings suggested that some low-temperature processed foods also had the potential to be contaminated by AA.

Acrolein came from a wide range of sources, and three major nutrients could produce acrolein during food processing. The thermal degradation of monosaccharide which occurred in the heating process of high-temperature protein and carbohydrate decomposition reaction would produce a large number of small molecular

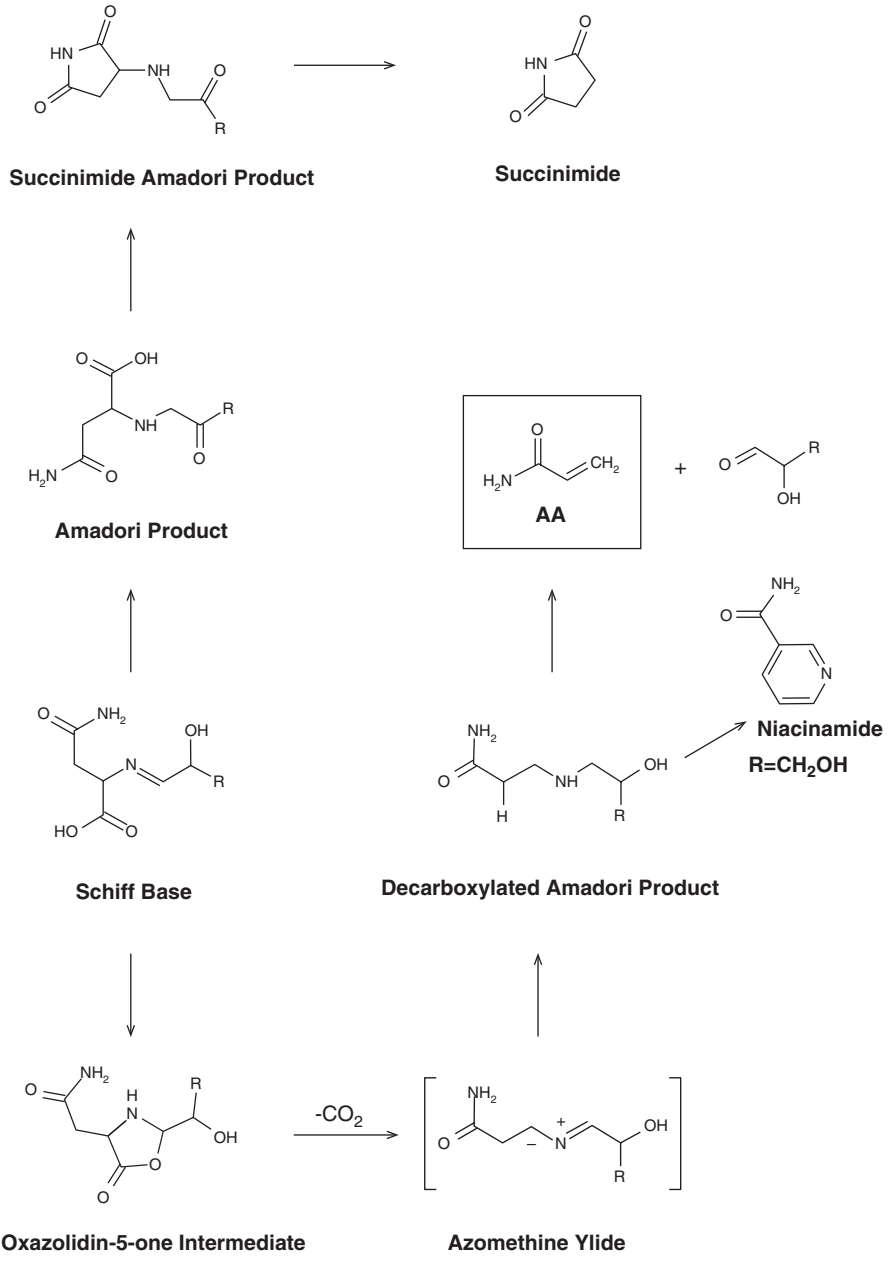


Fig. 3.5 AA formation by N-glycoside pathway

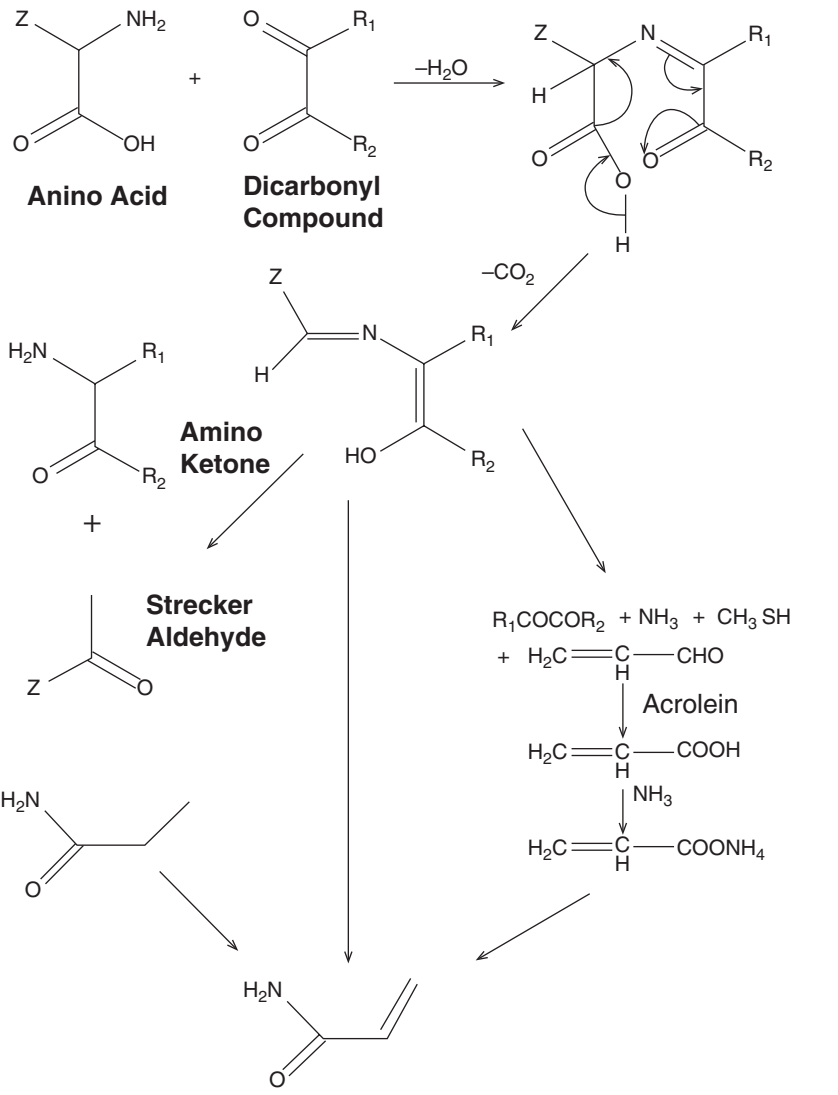


Fig. 3.6 AA formation by Strecker pathway

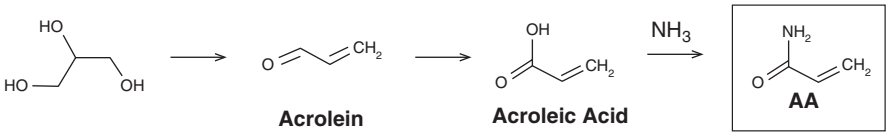


Fig. 3.7 Formation of AA from acrolein

aldehydes (such as formaldehyde, acetaldehyde, and so on). These aldehydes can form acrolein under suitable reaction conditions and then produce AA by the acrolein pathway [72]. The release of oil at high temperature in the heating process of glycerol three vinegar can be decomposed into glycerol and fatty acids, then further dehydration of acrolein produced glycerol [73], and fatty acids and glycerol were generated by oxidation of acrolein and acrylic acid, which provided precursors for the formation of AA. Protein and amino acids in foods, such as aspartic (Asp), can be degraded into acrylic acid. Alanine and arginine heated at high temperature can be converted into acrylic acid. Serine and cysteine generated acrylic acid by pyruvic acid and lactic acid under the condition of heating [69]. In meat products, carnosine can release β-alanine by hydrolysis, after deaminate to produce acrylic acid [74]. Although acrolein was widely used in food processing, the amount of AA produced by acrolein via the acrolein pathway was much smaller than that of the asparagine pathway [75] because of the presence of free ammonia in the acrolein pathway.

3.3 Analysis Methods

3.3.1 Pretreatments for AA Analysis

3.3.1.1 Extraction Technology

The initial focus in the analysis of AA was to obtain a representative homogenous sample. The whole portion or serving of food should be homogenized thoroughly before the extraction and analysis steps. The greatest differences between the analytical methods were the extraction and cleanup step [76]. In recent years, some new extraction technologies were used in the extraction of AA for further analysis. Abd and Albishri presented the simultaneous online preconcentration and separation technology for AA, Asn, and Glc using analyte focusing on ionic liquid micelle collapse capillary electrophoresis (AFILMC) [77]. The coupling of AFILMC with IL-based ultrasonic-assisted extraction (ILUAE) was successfully applied to the efficient extraction and determination of AA, Asn, and Glc in bread samples. Furthermore, an eco-friendly and costless coupling of the HPLC-UV with ionic liquid-based ultrasonic-assisted extraction (ILUAE) was developed to determine the AA content in food samples. ILUAE was proposed for the efficient extraction of AA from bread and potato chips samples [78]. Sun et al. demonstrated a facile MS-free method to determine AA content in microwaved and conventional heated popcorn and rice, using activated carbon-packed extraction column coupling with simple GC analysis [79]. Gökmen et al. studied the effects of single- and multiple-stage extraction procedures on the extraction yield of AA for various cereal- and potato-based thermally processed foods [76]. The extractability was an exponential function which could be used to optimize the multiple extraction conditions during the analysis of foods for AA. In general, the aqueous extraction using 10 mM formic acid was found to be more effective than the methanol extraction.

3.3.1.2 SPE and SPME Methods

For the cleanup of AA from complex samples, accelerated solvent extraction (ASE), liquid-liquid extraction (LLE), and solid-phase extraction (SPE) were used alone or in combination with other purification steps to extract AA. ASE and LLE with conventional organic solvent were time-consuming and labor-intensive methods for AA extraction and cleanup. Furthermore, these methods can easily lead to loss of AA in the scavenging process and require large amounts of organic solvents. The SPE method is simple and stable, has easy automation methods, and has been developed and applied in the cleanup of AA. Can and Arli developed an HPLC method and indicated AA levels in some traditional and non-traditional foods consumed in Turkey [80]. AA content of samples was determined using HPLC coupled to photodiode array detection. Samples were prepared by utilizing Oasis HLB (Waters) cartridges for cleanup and analyzed with no further preconcentration steps. Zhang W et al. researched a rapid and effective HPLC method, using tetraazacalix arene triazine-modified silica gel (NCSi) as SPE sorbent, which was developed for the purification and determination of trace AA in starchy foodstuffs [81]. The improved method was simple, rapid, accurate, and promising for the determination of trace AA in starchy foods with a complex matrix. Zhao H et al. synthesized a functionalized material by immobilizing an ionic liquid onto an activated silica gel surface [82]. Using this material as a sorbent, a method of SPE coupled with HPLC (SPE-HPLC) for analysis of AA in foods was developed. And this method was successfully applied to quantitative detection of AA in bread crust and cracker samples. Bortolomeazzi et al. developed a rapid and reliable purification method based on a single mixed SPE column, for the determination of AA in roasted coffee by LC-MS [83]. The results showed that the SPE method could be used in the pretreatment of AA in food. For SPE, the most important effect on the enrichment efficiency was the adsorption performance of the sorbent. Thus, a functionalized material with high adsorption ability was highly desirable.

The analysis of AA in food always required several pretreatments and cleanup steps. Conventional extraction techniques used SPE to purify crude sample extracts prior to the analysis. However, due to the multiple SPE steps, these methods were often time-consuming and achieving unsatisfactory limits of detection (LOD) [84]. Over the past few decades, solid-phase microextraction (SPME) had become a popular extraction technique in food analysis, due to its exceptional simplicity and ease of use. SPME was an equilibrium-based extraction/preconcentration technique that enabled the consolidation of sample preparation, cleanup, and sampling into one simple step. Compared to SPE, SPME was a non-exhaustive extraction technique that used considerably smaller sample volumes and was not susceptible to analyte breakthrough [85]. Qu et al. developed an SPME fiber coated with SWC-NTs/Ppy coupled to GC with electron capture detection (ECD) method for the detection of AA in different food samples [86]. Ghiasvand and Hajipour found a rapid, easy, and low-cost HS-SPME-GC-FID method to determinate the AA in foodstuffs (chips and French fries), using GC-flame ionization detection (GC-FID) system after its direct trapping in the upper atmosphere of samples by headspace SPME (HS-SPME) [87]. Chen L et al. established a new approach for the determination of AA in foods by SPME-GC

after derivatization [88]. The 2-bromoacrylamide (2-BAM), transformed from AA, was extracted by a commercial SPME fiber, 75 μm Car/PDMS fiber, for GC detection. The presented method was applied to the determination of AA in fried foods.

3.3.1.3 MSPD Methods

MSPD was a sample extract technology based on the traditional SPE method. In recent years, a number of applications of MSPD in food analysis had been reported [89–92]. Zhao H et al. synthesized and characterized the chitosan-grafted multiwalled carbon nanotubes using Fourier transform infrared spectroscopy, transmission electron microscopy, X-ray diffraction, thermogravimetric analysis, and static and kinetic adsorption experiments [93]. And it could be used as a sorbent for development of MSPD extraction coupled with HPLC for the determination of trace AA in foods. Xu X et al. found a quantitative method for the simultaneous determination of 3-MCPD and AA in food by GC-MS/MS. The analytes were purified and extracted by MSPD technique with Extrelut NT [94]. Soares and Fernandes described the development of an optimized MSPD procedure for the analysis of AA in a variety of food matrices (cereal products, chocolates, and baby foods) [92].

3.3.1.4 Derivatization Methods

Bromination was often used to determine the AA content in water or foods. Although bromination was a well-known and widely used derivatization technique, it needed a large number of reagents. In addition to this bromination method, some derivatization reagents such as 2-mercaptobenzoic acid [95, 96], dansulfinic acid [97, 98], and N,O-bis (trimethylsilyl) trifluoroacetamide (BSTFA) [99] had been applied for the determination of AA.

Cengiz and Boyaci Gündüz developed a method involving defatting with n-hexane, extraction with water, precipitation of proteins, bromination, extraction into ethyl acetate, and injection into a GC-MS system to detect AA of 62 different cereal-based baby foods [100]. The method was more eco-friendly and less expensive because it consumed very little solvent relative to other methods using bromine solutions and ethyl acetate. Surma et al. reported the development of a rapid and simple sample preparation method for AA determination in cocoa powder through extract purification by dispersive SPE and derivatization with BSTFA [101]. The developed method had been successfully applied to the determination of AA in 9 cocoa powder and 30 chocolate (dark, milk, and white) samples purchased at a Polish supermarket. Molina-Garcia et al. developed an AA derivatization technique using xanthydrol and analyzed by GC-MS in selected ion monitoring (SIM) mode in order to analyze AA in fried potatoes [102]. This method was applied to diverse foodstuffs and water, and it was claimed to be more environmentally friendly. A series of drawbacks that occurred with bromination derivatization were avoided when xanthydrol was used.

3.3.1.5 QuEChERS Methods

QuEChERS (quick, easy, cheap, effective, rugged, safe) was the latest rapid sample pretreatment technology developed by the US Department of Agriculture in 2003. QuEChERS had the advantages of high recovery rate, good accuracy, a wide range of analytical pesticides, fast analysis speed, and easy operation. In 2006, the QuEChERS method was developed and optimized by Mastovska and Lehotay as a high-throughput extraction method for AA analysis in various food matrices using LC-MS/MS or GC-MS [103]. The method involved the extraction with acetonitrile, liquid-liquid partition by salting out with sodium chloride and magnesium sulfate, and the cleanup step with the dispersive solid-phase extraction (dSPE) such as a primary secondary amine (PSA) [104].

The usefulness and effectiveness of QuEChERS method combination followed by AA silylation had been proven. Modification of the QuEChERS sample preparation method for the determination of AA in coffee and coffee substitutes via silylation had been reported. A sample of instant coffee was used to develop an analytical method in the optimization experiment, which involved the selection of the most suitable sorbents for dSPE cleanup. The AA level after conversion to N,O-bis-(trimethylsilyl)-AA (BTMSA) was determined with GC-MS in a single ion monitoring mode (GC-SIM-MS). The usefulness of the method had been verified based on the recovery ratio of AA (fortified samples analysis). The chosen method was successfully applied for the AA determination in 17 roasted, 10 instant, and 8 coffee substitute samples [105]. And the method for AA extraction and determination in dried fruits (dried prunes and raisins) and edible seeds (almonds, hazelnuts, peanuts, pine nuts, pistachios, and walnuts) using more efficient QuEChERS approach was set up. The study described a quick and easy method for AA determination by QuEChERS approach coupled to LC-ESI-MS-triple quadrupole technique with a “one-pot” sample preparation. Linearity, sensitivity, accuracy, and precision of the method were satisfactory [106]. A sample preparation method based on modified QuEChERS with aluminum oxide (Al_2O_3) as dSPE material and HPLC LTQ-Orbitrap MS was established. The performance of two analytical columns namely Kinetex C_{18} and Rezex ROA-organic acid was compared for AA separation. The proposed Al_2O_3 dSPE method was successfully applied to the analysis of AA in real food samples. And the Kinetex C_{18} column can be used for routine analysis of AA in a huge number of food samples, and ROA-organic acid column can be used for analysis of samples containing low contents of AA [107]. The use of the QuEChERS method, being nowadays one of the most widespread techniques in food analysis, can provide the ability to analyze various food contaminants at the same time.

3.3.2 Instrument Analysis

Hitherto, LC and GC coupled to MS appear to be acknowledged as the most useful and authoritative methods for the determination of trace AA.

3.3.2.1 HPLC Technology

HPLC with liquid mobile phase separated the different polarity of a single solvent or different proportions of mixed solvents. Xu L et al. reported a sensitive method of HPLC based on SPE using a reversed phase C_{18} column, which was characterized by adsorption experiments with good adsorption ability and rapid adsorption dynamic toward AA [108]. And the developed method was applied to the extraction and determination of AA in deep-heated food samples with a good accuracy. Jiang et al. successfully synthesized a novel AA molecularly imprinted material based on silica microparticles [109]. The molecularly imprinted polymers (MIP) on the surfaces of silica microparticles were characterized using infrared spectroscopy and scanning electron microscopy, and the amount of AA adsorbed on the MIP was measured using HPLC and equilibrium binding experiments. HPLC as one of the most widely used technologies, coupled with MS detection, was the most preferred.

The use of HPLC for the analysis of AA was another way to avoid derivatization steps, which were often laborious and resulted in variable yields. HPLC analyses of AA that employed UV-detection suffered from high detection limits (3–10 μg/L) [110]. Wang H et al. studied on a simple and cost-effective method using HPLC-UV technology to quantify the content of AA in baked and deep-fried Chinese foods [111]. HPLC-UV methodology was equipped with a vacuum degasser, a binary pump, a diode array detector, and a temperature-controlled column oven. And the AA contents were found to be 86.3–151 μg/kg. A fast and cost-effective method using HPLC-UV had been developed for the determination of AA in deep-fried flour-based leaven dough foods in Hong Kong [112]. AA was detected at UV wavelengths of 210 and 225 nm. The amounts of AA in eight food samples (stuffed glutinous rice ball, "oxtongue" fritter, sesame ball, deep-fried sesame cookies, glutinous rice sesame ball, fried sweet dumpling, fried egg pastry, and sweet potato crisps) were 27–198 μg/kg when 1 g samples were analyzed. Paleologos and Kontominas developed a method using normal phase with UV detection for the analysis of AA and methacrylamide [113]. This method was applied for the determination of AA and methacrylamide in spiked food samples without native AA yielding recoveries between 95% and 103%.

More recently, HPLC-MS methodology is used due to its high selectivity and sensitivity. Solid-phase cleanup coupled to LC-MS detection was nowadays the gold standard procedure for AA quantitation owing to the high reproducibility, good recovery, and low relative standard deviation. Backe et al. developed a simple and sensitive analytical method to quantify levels of AA in environmental and drinking waters by HPLC-MS [110]. The method detection limit and reporting levels were 2.4 and 17 ng/L, respectively. Troise et al. researched an LC method for AA determination using HRMS detection. It was proved to be solid and robust with LOD of 2.65 ppb and LOQ of 5 ppb. The method was tested on four AA-containing foods: cookies, French fries, ground coffee, and brewed coffee [114].

LC-MS/MS showed high sensitivity, strong anti-interference ability, and high qualitative and quantitative accuracy in the multi-residue trace detection. Recently,

HPLC-MS/MS and UHPLC-MS/MS had been developed to simultaneously analyze AA, Asn, and sugars in the Maillard reaction. Lim and Shin developed an LC-MS/MS method to determine the amount of AA in foods after derivatization with D-cysteine [115]. The method was successfully applied to determine the amount of AA in potato chips, French fries, and coffee. Zhang Y et al. developed an automated microwave digestion labstation (MDL) combined with UHPLC-MS/MS method which was successfully validated and applied to the determination of AA and its precursors and intermediates during Maillard reactions and kinetic elucidation [116]. Zhang C et al. employed UPLC-MS/MS and QuEChERS dispersion to establish the detection method for four AA compounds (AA, methacrylamide, N-methylolacrylamide, N-(Methoxymethyl)methacrylamide) in food-contact paper products and food simulants, which could provide reference for the detection and specific migration study of related compounds in foods [117].

3.3.2.2 GC Technology

GC methods have generally measured the derivatization of AA with potassium bromate/potassium bromide to improve the GC properties. A recent GC method employing a high-resolution time-of-flight mass analyzer was developed for direct analysis (no derivatization) of AA in various heat-processed foodstuffs [118]. Another GC method was based on derivatization of the target analytes with bromination and detection by an electron capture detector (ECD) [119]. Qu et al. developed an SPME coupled to GC with ECD method for the detection of AA in food samples, which possessed good extraction efficiency of AA [86]. The linearity range between the signal intensity and the AA concentration was found to be in the range 0.001–1 μg/mL, and the coefficient of determination was 0.9985. Notardonato et al. proposed a new method for quantitative analysis of AA in cereal-based foods and potato chips [120]. This method used reaction with trifluoroacetic anhydride and analyzed the resulting derivative by GC-ECD. Under the optimum conditions, good retention and peak response were achieved for the AA derivative. The analytical method was fully validated by assessment of LODs and LOQs.

A European interlaboratory study was conducted to validate two analytical procedures for the determination of AA in bakery ware (crisp bread, biscuits) and potato products (chips), within a concentration range from about 20 μg/kg to about 9000 μg/kg. The two methods were based on GC-MS of the derivatized analyte and on HPLC-MS/MS of native AA [121]. A sample preparation technique based on MSPD was applied to the determination of AA in potato chips. After bromination of the extract, the samples were analyzed by GC-MS in selected ion monitoring mode. The method presented good recoveries, and the LOD and LOQ were 12.8 and 38.8 μg/kg, respectively [122].

A GC-MS/MS method was used to measure AA in aqueous matrices extracted from French fries and potato crisps using direct immersion SPME without derivatization. The concentrations of AA detected in French fries and potato crisps were 1.2 and 2.2 μg/g, respectively [123]. Another GC-MS/MS method was developed for separa-

tion, detection, identification, and quantification of AA in bread, biscuits, and similar products, which showed good precision with values lower than 6%. A good sensitivity was achieved for bread with 2.41 (LOD) and 7.23 μg/kg (LOQ), respectively, while for biscuits, LOD and LOQ were 4.63 and 13.89 μg/kg, respectively [124].

In addition, GC-nitrogen phosphorus detector (GC-NPD), GC-MSD, and other technologies could be used in the analysis and detection of AA. Kim et al. developed an improved analytical method for the quantification of AA using a GC-NPD, which showed reasonable recovery, sensitivity, and accuracy in monitoring AA levels [125]. A new protocol combining GC-NPD and LC-tandem mass spectrometry (MS/MS) could be applied to analyze AA accurately and precisely, contributing to the development of a prescreening tool to analyze AA in the food industry. A direct GC-MS method for quantitative determination of AA in several foods was described. The use of diatomaceous earth as sorbent was in combination with ethyl acetate as desorbing solvent. This allowed us to make use of a mass selective detector (MSD), which was cheaper, and available in more laboratories than tandem mass spectrometers [126].

3.3.2.3 Capillary Electrophoresis (CE) Technology

CE had the merits of high efficiency, fast analysis, and low reagent consumption and became an effective alternative for the analysis of a great variety of target substances, but its stability is poor [108]. A non-aqueous CE method was developed for the quantitative determination of AA in the processed food like potato chips and French fries. The AA amount of these samples was found to be 2.95 ± 0.11 mg/kg. A field-amplified sample stacking technique with the non-aqueous capillary electrophoresis method was introduced for the online concentration of AA to increase the poor sensitivity of UV detection at 210 nm by diode array detection [127]. The application of the sample stacking was performed on potato chips and almond extracts. The AA content in almond extract and some potato chips was found to be 95.5 ± 7.6 and 4.3 ± 1.5 μg/kg, respectively [127]. Chen Q et al. [128] detected AA levels in potato crisps by CE with quantum dot-mediated LIF detection. The method had been successfully applied in the determination of sub-parts per million levels of AA or 2-propenamide in a complex sample such as potato crisps.

3.3.3 Rapid Detection Technologies

Since the industries and factories are trying to control the levels of AA in food, there is a need for fast and routine quality control methods for the detection of AA. The current techniques used for AA detection had the following steps: aqueous extraction of AA from food, the concentration of the sample, and analysis by either GC/LC-MS or HPLC. The whole process took a considerable amount of time and required both sophisticated equipment and trained personnel. For this reason, there was a need for a simple, robust, and fast detection method for AA [129]. LC-MS/

MS and GC-MS methods as standard detection methods showed high sensitivity, selectivity, stability, and repeatability. However, these methods required expensive instruments, skilled technicians in laboratories, industries and factories, and high testing costs, and could not meet the needs for real-time and online detection of AA in foods. Therefore, rapid detection methods had developed based on the similar sensitivity and selectivity, but required less time and cost in comparison with standard methods for AA detection.

3.3.3.1 Elisa Methods

Immunochemical methods such as enzyme-linked immunosorbent assays (ELISA) had become a popular and useful screening tool for deleterious compound residues because the methods are of low cost, high selectivity, and specificity. Moreover, ELISAs were able to simultaneously analyze a large number of samples with a simple extraction process. Zhu Y et al. coupled the AA derivative (4-mercaptobenzoic acid) to a carrier protein such as bovine serum albumin and ovalbumin [130]. The conjugates were used as the immunogen and coating antigen. A rapid and sensitive indirect competitive enzyme-linked immunosorbent assay (icELISA) against AA-4-mercaptobenzoic acid was obtained by optimizing the experimental parameters. The monoclonal antibody had no specificity for AA or 4-MBA, but had a high affinity for AA-4-MBA, with an IC_{50} of 32 ng/mL and LOD of 8.87 ng/mL. The quantitative working range was 8.87–112.92 ng/mL (IC_{20} to IC_{80}). Cross-reactivity with other analogs was lower than 10%. These results indicated that the developed icELISA was a fast and efficient method for the detection of AA in food [131].

An enhanced chemiluminescence ELISA had been developed to quantitate AA in food products, such as potato chips, instant noodles, cookies, and cakes [132]. As AA was easily reacted with 4-MPA at high derivation yield, a competitive indirect enzyme-linked immunosorbent assay (ciELISA) for AA via a pre-analysis derivatization was developed. The derivatization and ELISA conditions were fully optimized for AA assay, and the IC_{50} of the method was 2.86 μg/kg, LOD was 0.036 μg/kg, and linear range was 0.25–24.15 μg/kg. This method was thus deemed suitable for routine AA screening in food samples at low cost [133]. Singh et al. [134] raised the polyclonal antibodies against a hapten derived from AA and 3-mercaptobenzoic acid. An indirect competitive ELISA was developed to rapidly quantify AA in complex food matrices and water. Good recoveries for AA were observed in all matrices tested, and the results using this method were comparable to those obtained from mass spectrometry methods.

3.3.3.2 Sensor Methods

Gezer et al. tested a biodegradable zein-based sensor with surface-enhanced Raman spectroscopy (SERS) platform as a potential tool to detect AA for the first time [129]. This was a proof-of-concept study to investigate the potential of a SERS sensor, and it was shown that it could be potentially used with a careful design of AA

extraction from food samples. Liu X et al. successfully constructed a sensitive molecularly imprinted electrochemical sensor [135]. It was applied to the detection of AA in potato chips. HPLC analysis was also conducted to detect AA in the same samples to demonstrate the applicability of the electrochemical molecularly imprinted polymer sensor. The electrochemical sensor had received increasing attention in many fields due to their low cost, small size, fast response time, the possibility of achieving low detection limits, and strong operability [136, 137].

3.3.3.3 Nondestructive Methods

Conventional methods of AA identification in food items were time-consuming, expensive, and might need specialized manpower. It was necessary to propose some nondestructive methods for identifying the presence of AA in food.

Dutta et al. proposed a computer vision work-based nondestructive method to identify the presence of AA in potato chips [138]. The proposed method was based on analysis and classification of the discriminatory features of the image in the spatial domain. The potato chips were automatically segmented from the image followed by statistical and texture features extraction from the segmented image in the spatial domain. These statistical features were then analyzed for identification of AA content using support vector machine (SVM) classifier. The experimental results had shown accuracy over 94% and sensitivity of 96% indicating that this method could be explored for viable commercial use.

While, in 2016, Dutta et al. presented an efficient image processing-based nondestructive method for identification of the presence of AA from potato chips based on statistical analysis and classification of the image features in the wavelet domain [139]. This proposed work involved segmentation of the area of the potato chip from the background of the image followed by feature extraction and strategical analysis in the wavelet domain. Wavelet features had discriminatory properties for identification of the presence of AA in food items. The experimental results of 97% accuracy and 100% sensitivity could be explored for commercial use.

Adedipe et al. investigated the ability of near-infrared spectroscopy (NIRS) to predict AA content in French fried potato [140]. Reflectance spectra (400–2500 nm) of each freeze-dried sample were captured on a Foss XDS Rapid Content Analyzer-NIR spectrometer. NIRS could accurately detect AA content as low as 50 μg/kg in the model potato matrix. The findings indicated that NIRS could be used as a screening tool in potato breeding and potato processing research.

3.4 Inhibition Methods of AA Formation in Foods

As AA widely existed in different food categories, the control and prevention of AA intake along human had become a hot topic within the food safety field in recent years. Because of the obvious matrix effects on the food system and various AA

formation mechanisms, several factors were needed to be considered for reducing the content of AA in foods. According to the mechanisms of AA formation, the widely used methods of inhibiting AA were mainly in the following three ways: the first one was to reduce or eliminate the AA formation precursors to decrease the content of AA formed in the end-products. The second was to optimize the processing technology, such as adjust the processing mode, processing temperature, processing time, pH values, moisture content, and other factors. The third was to add exogenous compounds to inhibit the formation of AA.

3.4.1 Reducing the AA Formation Precursors in Food Systems

According to the formation mechanisms of AA, Asn and reducing sugar (mainly Glc and Fru) were the major precursors for AA formation. Reducing the substrate content of the reactions was found to be an effective way to reduce the AA levels in the food products. Screening the crop varieties and cultivation conditions can control the content of AA precursors [141] from the first beginning. Postles et al. found that Asn was the main amino acid in potato tubers, and Asn concentrations were significantly different among different varieties [142]. Asn concentrations in the three tested samples were between 1621 and 3512 mg/kg, while Glc was between 985 and 1781 mg/kg; the low sugar level tubers had the lowest AA yield which was only 58% of the high sugar level variety. The limited factors in the formation of AA in potato tubers were mainly relevant to sufficient content of Asn and the low concentration of Glc [143], so the use of low-sugar varieties can effectively reduce the AA content in the food products. For cereal products, Asn was a limiting factor for the formation of AA, and low Asn content crop varieties can reduce the AA content of cereal products effectively [142]. These results showed that an AA mitigation strategy focused on developing potato cultivars with low Asn and reducing sugars was likely to be an effective and sufficient strategy. Gene silence technology was used to minimize the AA contents by Zhu et al. [144]. They generated a large number of silenced lines of potato cultivar *Russet Burbank* by targeting the vacuolar invertase gene *VInv* and the asparagine synthetase genes *StAS1* and *StAS2* with a single RNA interference construct. The transcription levels of these three genes were correlated with reducing sugar and Asn content in tubers. Fried potato products from the best VInv/StAS1/StAS2-triple silence lines contained only one-fifteenth of the AA content compared to the control groups.

The cultivation conditions had important effects on the content of amino acids and reducing sugar in the crops, and the AA content of the final products could be reduced by controlling the cultivation conditions of the crops. The use of sulfur fertilizer in the process of potato cultivation can significantly affect the ratio between free amino acid and sugar, as well as free Asn and total amino acids in potato tubers. By reducing the amount of sulfur fertilizer, the ratio of free Asn occupied in total amino acids could be also lowered [145, 146], which resulted in a decrease in the amount of AA produced during subsequent thermal processing. The amount of

nitrogen applied was closely related to the Asn content in rye. Excessive fertilization will lead to a significant increase in the amount of Asn, thereby increasing the AA content in rye [142].

The storage conditions of the crop also affected the free Asn and reducing sugar content. In the low-temperature storage conditions, the potato tubers will undergo a phenomenon called "low-temperature saccharification," which led to the metabolism of carbohydrates in the direction of reducing sugars. A large amount of reducing sugar was beneficial to the formation of AA in the final products. Reducing sugar content in potato tubers can be effectively reduced by selecting the appropriate storage temperature and storage conditions. The AA in the final product could be reduced by up to 16 mg/kg [147, 148] when the potato tubers were stored at 8 °C compared to the low-temperature stored potato tubers.

3.4.2 Optimizing the Processing Technologies

The Maillard reaction is the main way to form AA in food. By adjusting the heating temperature, heating time, pH value, water content of food systems, and other factors during processing, the degree of Maillard reaction and AA formation could be effectively controlled. In addition, during the heating process, we can modify or add certain processing techniques to reduce the production of AA, such as using vacuum baking instead of conventional baking methods [149], pretreatment, altering the oil used for frying [150], blanching [151], fermentation [152], and other processes.

3.4.2.1 Controlling the Processing Temperature and Time

The Maillard reaction is a typical reaction of temperature and time. It was beneficial to reduce the accumulation of AA in the product by reducing the heating temperature and reducing the heating time [153]. In the general food systems, AA could be formed when the heating temperature was above 120 °C, and the amount of AA would increase along the heating temperature and time. But when AA reached a certain amount range, AA contents began to decrease as the reaction time prolonged [62]. Truong found that the frying time significantly affected the AA content in the fried sweet potato fries, and the AA yields were 124.9, 255.5, and 452.0 ng/g (fresh weight) when frying 2, 3, and 5 min, respectively [154]. Tuta et al. pretreated frozen fries by microwave and fried at 190 °C for 2.5 min [155]. AA content in these fries reduced by 64% compared to the traditional fried whose frying time was 3.5 min.

3.4.2.2 Adjusting the pH Values

The Maillard reaction is a pH-depended reaction. Under different pH values, the Maillard reaction route will change accordingly. The potato powder model system (containing 41% potato powder, 38% water, and 21% oil) with different initial pH

values was heated at 170 °C for 6 min, and the largest AA content was found when the pH value was between 7 and 7.5 [156]. For AA, the most proper pH value range for AA formation was between 7 and 8 [153]. Reducing the pH value of the system can limit the nucleophilic addition of Asn and reducing sugars, thereby preventing the formation of Schiff base [153, 157, 158]. Thus, the accumulation of AA in the final product can be limited by the addition of an acidifying agent in the food processing process [159]. After lowering the pH of the dough, the AA content of the flour was significantly reduced, but the level of 3-chloro-1,2-propanediol, another hazardous substance, was significantly increased [159]. Therefore, in the actual food processing, it was necessary to take into account not only the qualities of the final products but the formation of other harmful substances.

3.4.2.3 Adopting New Processing Methods

In recent years, new processing technologies had been widely used in food processing. Banchero et al. used a supercritical fluid extraction process to extract AA from coffee, which would reduce the AA contents by 79% without affecting the caffeine content in the coffee [152]. With the vacuum frying method, the fried temperature can be reduced from 180 to 165 °C, and the AA content in the end product can be reduced by 51% [160]. Wei invented a novel microwave-infrared combined baking process, which could reduce the baking time by 27% and the energy consumption by 85% compared to the traditional baking method [161]. The AA content in the cookies produced by this method was reduced by 66.7%, and the sensory quality of the cookies was not significantly different from that of the conventional baking method [161].

3.4.2.4 Using of the Asparaginase

AA was considered to form from the reaction of Asn and reducing sugars contained in foods, so the free asparagine was a key limiting factor for AA formation. The content of AA in the final product can be reduced by treating with asparaginase. Asparaginase can decompose Asn in raw materials to inhibit AA production effectively [162–164]. Onishi et al. treated sliced potatoes with *Bacillus subtilis* L-asparaginase II (BAsnase; 4 U/g potato), and approximately 40% of L-Asn in the sliced potatoes was converted into L-aspartic acid (L-Asp), resulting in the hydrolysis of approximately 90% of L-Asn to L-Asp [165]. The AA content of BAsnase-treated fried potato chips decreased to below 20% compared to BAsnase-untreated fried potato chips.

Some new types of L-asparaginase showed even better performance in inhibiting AA than the commercial enzymes. *Escherichia coli* type L-asparaginase (EcAII) was a commercial enzyme trademarked Elspar (R) that was widely used for medical applications. Dias et al. produced and purified an L-asparaginase from native *Aspergillus oryzae* CCT 3940 with high potential for AA mitigation [166]. The

results indicated the AA concentration of the fried potato treated with the L-asparaginase from *A. oryzae* CCT 3940 and treated with commercial enzyme reduced 72% and 92%, respectively, compared to control sample [122]. Moreover, the L-asparaginase from *A. oryzae* CCT3940 showed no L-glutaminase activity, while commercial enzyme promoted a decrease in the L-glutamine content (25%). Sun et al. reported the identification of a novel bacterial type II L-asparaginase, abASNase2, from *Aquabacterium* sp. A7-Y [167]. Compared to the untreated potato strips (AA content: 0.823 +/− 0.0457 mg/kg), 88.2% AA was removed in the abASNase2-treated group (AA content: 0.097 +/− 0.0157 mg/kg). Bhagat et al. purified an extracellular asparaginase from an endophytic bacteria *Pseudomonas oryzihabitans* isolated from *Hibiscus rosa-sinensis* [168]. Addition of purified asparaginase (2.8 U/g dried potato), 90% AA was reduced in fried potatoes. Shi R et al. cloned and expressed a novel L-asparaginase gene (PbAsnase) from *Paenibaeillus barengoltzii* CAU904 in *Escherichia coli* [169]. They found that pre-treatment by PbAsnase significantly decreased the AA contents in potato chips and mooncakes by 86% and 52%, respectively [169]. All these results indicated that the novel L-asparaginase could be a potential candidate for applications in the food processing industry.

3.4.2.5 Blanching

Blanching was another important strategy to control the formation of AA in food systems. Potato chips or fries could be blanched to reduce the sugar and Asn on the surface. After the potato chips were soaked in distilled water for 90 min and then fried at 150 °C, 170 °C, and 190 °C, the amount of AA was reduced by 27%, 38%, and 20%, respectively. After the blanching treatment, Glc and Asn contents in the material were significantly reduced [170].

Zuo et al. developed an extremely thermostable L-asparaginase which could be used in the reduction of AA level through blanching [171]. The purified enzyme displayed the maximum activity at pH 8.5 at 90 °C, and the temperature was the highest ever reported. When potato samples were treated with 10 U/mL of L-asparaginase at 80 °C for only 4 min, the AA content in the final French fries was reduced by 80.5% compared with the untreated group. This result showed that a combination of different inhibition methods could be used in the further processing industry.

3.4.2.6 Fermentation

Fermentation treatments were an effective way to reduce AA formation in fried potatoes without influencing product quality. Using lactic acid bacteria in rye dough, the AA in rye bread was reduced by 40–59.4% [172], and the reason was related to the activity of starch decomposition reduction caused by lactic acid bacteria, accompanied by pH reduction in the food system.

Potato chips can also reduce the amount of AA by yeast fermentation. Zhou et al. found that the optimum conditions for decreasing reducing sugar contents were a 1:4 solid-liquid ratio and a fermentation temperature of 37 °C with 0.5% yeast [173]. Potato strips treated under optimum conditions had 70% less AA than the control group chips.

The effect of the fermentation process by four lactic acid bacteria and yeast on an industrial scale was studied on AA reduction in bread [174]. AA content in the control fermented by yeast contained the highest amount of reducing saccharides (239.12 mg/kg). After being treated with four lactic acid bacteria, fermented bread with *Lactobacillus paracasei* showed the lowest amount of AA (131.06 mg/kg) due to its lower pH of sourdough (3.51) and glucose content (5.44 mg/g).

Bartkiene studied AA reduction technology in wheat flour biscuits supplemented with lupine and flaxseed treated by solid state (SSF) and submerged (SMF) fermentations by *Lactobacillus sakei*, *Pediococcus pentosaceus,* and *Pediococcus acidilactici* strains, respectively [175]. The most effective AA reduction in biscuits (78% and 85%, respectively) was reached by using *P. acidilactici* for flaxseed (SMF) and lupine (SSF). Using *L. sakei* for SSF of flaxseed and SMF of lupine, the average AA reduction rate was 83.4%.

3.4.3 Adding Exogenous Compounds

The changes of the raw materials of the food ingredients and processing conditions will affect the food quality, which is difficult to be accepted by manufacturers and consumers. Nowadays, some additives are used to inhibit the formation of AA in food systems and show good effects.

3.4.3.1 Adding Metal Ions

Studies showed that the addition of the metal ions could effectively inhibit the formation of AA. The addition of sodium chloride (NaCl) in food reduced AA formation significantly [176–178]. However, the inhibitory effect of NaCl on AA was not consistent with the concentration. Studies had shown that AA formation was reduced by 32%, 36%, and 40%, respectively, after adding 1%, 5%, and 10% NaCl in equimolar Asn/Glc model systems [179, 180]. In industrial production, the addition of 1% NaCl had a large application prospect, since 1% NaCl was the salt content actually required in many food products [181]. Friedman and Levin proposed the addition of Na^+ could alter the ionic strength of the reaction environment, thus affecting the combination of amino groups of amino acids and AA [182]. In addition, using DSC and infrared spectroscopy technologies, Kolek et al. found that NaCl could catalyze the polymerization of AA to polyacrylamide [176, 179, 180]. The study also demonstrated that the addition of potassium ferrocyanide ($K_4[Fe(CN)_6]$) and potassium iodate (KIO_3) had better performance in AA polymerization than using

NaCl. However, as a commonly used leavening agent in baking, ammonium bicarbonate (NH_4HCO_3) could significantly promote the formation of AA, and it was because of the promotion of NH_4HCO_3 on the formation of α-dicarbonyl compounds and the reaction with NH_3 to form glycosylamine [182]. When sodium bicarbonate ($NaHCO_3$) replaced NH_4HCO_3, the AA in the final product reduced by 70% [183]. Other metal ions, such as K^+, Ca^{2+}, Mg^{2+}, Zn^{2+}, and VO^{2+}, had also reported the abilities to inhibit the formation of AA [156, 184–187]. The current studies had shown that metal ions inhibited the formation of AA by affecting the formation of Schiff base [185, 186, 188] and the pH values of the solutions [156, 187, 189, 190].

3.4.3.2 Adding Vitamins

Zeng et al. found that water-soluble vitamins could inhibit the formation of AA in the Maillard model systems and the food system effectively, while the inhibitory effect of fat-soluble vitamins was weak [191]. The inhibitory effect of water-soluble vitamins such as biotin, pyridoxine, pyridoxamine, and vitamin C on the Asn/Glc model systems was up to 50% (170 °C, heating for 30 min). Except for VB_5 and pyridoxal, the total inhibitory rate of all the water-soluble vitamins in the food system was lower than that of the model system. The phenomenon might be due to the final functional groups (hydroxyl groups, amino groups, and aldehydes) of the side chains of the compounds, which played a reductive role in the formation of AA. In the low-moisture system, the addition of pyridoxamine could effectively inhibit the formation of AA, but had no influence on the degradation of AA precursors Glc and Asn, which indicated the inhibitory effect of pyridine on AA formation did not occur at the beginning of the Maillard reaction. It could be concluded as pyridoxamine could capture the dicarbonyl cyclides and sugar fragments in the Maillard reaction through the phenolic groups and aminomethyl groups on the pyridine ring to achieve the effect of inhibiting AA formation [192].

Kamkar et al. studied the efficiency of vitamins B_3, B_6, and the autolyzed yeast on the decrease of AA formation in potato chips [193]. Potatoes were soaked in different concentrations (0.1%, 0.5%, and 1%) of vitamin B_3, B_6, and the autolyzed yeast treatments. Results indicated a 58%, 50%, and 33% of the reduction, respectively, in AA formation in final products without affecting the flavor of the chips when the addition levels were 1 g/100 g. Although the inhibitory effects of on AA have been reported many times, there is no uniformity on its inhibition mechanism, and further study is still needed.

3.4.3.3 Adding Antioxidants and Plant Extracts

In recent years, some plant extracts were found to be able to affect the amount of AA in food and model systems. Cheng et al. found that apple extracts demonstrated potent inhibition on AA formation [194], whereas blueberry, mangosteen, and lon-

gan extracts did not have a significant impact on AA reduction. Column chromatography technology showed that the proanthocyanidin-rich subfraction played a key role in mediating the inhibitory activity. The inhibitory activity can be corroborated in fried potato crisps. Zhu et al. provided a possible use of crude aqueous plant extracts to mitigate AA formation in the Maillard reaction [195]. Effects of 35 crude aqueous extracts from dietary plants (spices, fruits, tea, beans, and herbs) and 11 phenolic compounds on the mitigation of AA in an Asn/Glc model system were investigated. Extracts of mint, cumin seeds, and star anise caused the greatest reduction in AA formation of 75%, 73%, and 69% respectively, but Huanggang Ku-Jing-Cha extract facilitated AA formation with increases of 18%. Of the phenolic compounds, *p*-coumaric acid caused the most significant reduction (53%), whereas hesperetin increased AA levels by 9%. Zhang et al. optimized the method and the frying processing parameters of the addition antioxidant from bamboo leaves to fried chicken wings and also compared the relationship between the contents of total flavonoids in three extracts (EBL971, EBL972, and antioxidant from bamboo leaves) [196]. The results showed that nearly 57.8% and 59.0% of AA in fried chicken wings were reduced when the antioxidant from bamboo leaves addition ratios were 0.1% and 0.5% (w/w), respectively. The maximum inhibitory rate was achieved when antioxidant from bamboo leaves was chosen as the additive with a total flavonoid content of 32% compared with other two extracts, and antioxidant from bamboo leaves mixed with flour was selected as the method of addition.

Using plant polyphenols in inhibiting the formation of AA also became a focus of food safety field. The polyphenol was a potent antioxidant widely contained in fruits and vegetables [197, 198]. The results showed that polyphenols had a good inhibitory effect on the formation of AA in food and model systems, and the uses of some typical polyphenols on AA inhibitions were shown in Table 3.3. But the effects of plant polyphenols on AA were complicated; some polyphenols such as t-butylhydroxyanisole and dibutylhydroxytoluene even promoted the formation of AA [199]. The results indicated that the inhibitory effect of polyphenols on AA was not depended on its antioxidant properties [70, 72, 200, 201], but the detailed inhibition mechanisms of polyphenols were still not clear.

3.4.3.4 Adding Amino Acids and Proteins

As the components of food, amino acids and proteins can also inhibit the formation of AA in the model systems and food systems [153, 208, 209]. Previous studies had shown that glycine (Gly) was effective in inhibiting the formation of AA [210, 211]. The α-amino group on Gly can directly link to the double bond of AA as Michael addition, or Gly can be oxidized and react with AA to eliminate [210]. The kinetic data showed that there was a relative competition inhibition mechanism between the reactors, and Gly and AA elimination reaction was the main mechanism of inhibition of AA formation [131]. The addition of amino acids could significant reduce AA formation while the color and flavor of the final product would change as well [156, 190, 212]. At the same time, some non-protein amino acids, such as alliin and

Table 3.3 Summary of studies and findings on the reduction of AA formation by antioxidant

Matrix	The type of antioxidant	Inhibitory effect	References
Cookie	Eugenol and cinnamaldehyde	The maximal inhibitory rates of eugenol and cinnamaldehyde were 42.3% and 34.0%	[202]
	Tert-butylhydroquinone	The inhibition rate was 54.1%	[203]
Emulsification system	Protocatechuic acid and gallic acid	The maximum inhibitory rates of protocatechuic acid and gallic acid were 70.0% and 50.0%.	[204]
Potato model system	Epicatechin (EC), epigallocatechin gallate (EGC), epicatechin gallate (ECG), apigenin, celeryin, vitexin, isovitexin, luteolin, lute 7-O-glucoside, luteolin-4′-O-glucoside, orientin, alfalfin, kaempferol, kaolin-3-O-glucoside, quercetin, quercetin-3-glucose Glycosides, rutin, myricetin, daidzin, daidzin, genistein, genistin	All the antioxidants can inhibit the produce of AA, the inhibition differs from different concentrations, the best AA inhibition rates of various substances were between 25.3% and 71.6%.	[205]
model systems	Epigallocatechin gallate (EGCG)	The maximal inhibition rate is 91.9%	[205, 206]
	Ferulic acid	The maximum inhibition rate is 47.1%	[207]
	Iso-orientin	The maximum inhibition rate of 61.8%	[205]

taurine, were also found to have the ability to inhibit AA formation [212–214]. In fact, in addition to amino acids, protein could also achieve the purpose of inhibiting the formation of AA in food. Amaranth protein could inhibit the formation of AA in the model and food systems. The inhibition rate in the model system was 35%–40%, in the cookie system was 89%, and in the fried corn flakes was 51%. When frying potato chips were wrapped with Chickpea batter, it was found that AA content reduced by about 30% compared to the unpackaged chips [72]. The addition of soy protein hydrolyzate to fries could also reduce AA formation [215]. Not only the plant but the animal proteins could inhibit the formation of AA in food, and it was deduced as the protein nucleophilic group (-SH or -NH_2) could react with AA and finally eliminate the formation of AA [153].

3.4.3.5 Other Additives

Sansano et al. found that the addition of chitosan might be an excellent alternative for reducing the formation of AA because of their richness in amino groups [216]. The results showed that chitosan was capable of inhibiting the formation of AA in

model systems and in fried batters. In model systems, the reduction in AA levels ranging from 49% to 85% was achieved for only 1% of chitosan. In the fried batter, AA was mitigated by 59 ± 6% with a chitosan concentration of 0.27% in batter formulations.

3.5 Conclusion and Outlook

In recent years, there have been significant improvements in the AA field including the detection methods, the formation mechanisms, and mitigation strategies. Several researchers have investigated many rapid and sensitive AA analysis methods. The developments of some pretreatments technologies gave us more choices for different food matrix. But the complexities of food matrixes also gave us more challenges for AA detections. Rapid detection methods played important roles in the detection of AA, including computer vision, ELISA, electrochemical biosensors, and fluorescent methods with the advantages of being low cost, simple, easy to handle, and portable for detecting AA in thermally processed foods compared with standard methods such as LC-MS/MS and GC-MS. These rapid detection methods have the ability to satisfy the need for food industries, regulatory agents, and customers. However, they still need further improvements to make themselves more accurate, sensitive, repeatable, reproducible, and/or portable to achieve online and real-time detection of trace AA. Besides, simplified pretreatment is essential in the detection of AA using a rapid (except for computer vision method) method.

Various AA mitigation methods have been successfully tested in the lab, but seldom applied in food industries at a commercial scale, partially because those current practical measures to reduce AA in foodstuffs are taken voluntarily [108]. Further researches in this area should focus on the following aspects: development of standardized quantitative methods to facilitate the proper and accurate evaluation of AA levels in foods, building up a corresponding AA database worldwide, and the development of new technologies to successfully reduce AA in food at a commercial scale.

References

1. Lyon F (1994) IARC monographs on the evaluation of carcinogenic risks to humans. In: Some industrial chemicals, vol 60. IARCPress, Lyon, pp 389–433
2. Swedish N (2002) Information about acrylamide in food. Swedish National Food Administration 4:24
3. Joint Research Centre's Institute for reference materials and measurements. Acrylamide monitoring database. European Commission. 2006
4. Survey data on acrylamide in food: total diet study results. FDA. 2004–2006
5. EFSA CONTAM Panel (2015) Scientific opinion on acrylamide in food. EFSA J 13(6):4104

6. World Health Organization (2005) Summary report of the sixty-fourth meeting of the Joint FAO/WHO Expert Committee on Food Additive (JECFA). The ILSI Press International Life Sciences Institute, Washington DC, Rome
7. Mojska H et al (2016) Estimation of exposure to dietary acrylamide based on mercapturic acids level in urine of Polish women post partum and an assessment of health risk. J Expo Sci Environ Epidemiol 26(3):288
8. Gao J et al (2016) Dietary exposure of acrylamide from the fifth Chinese Total Diet Study. Food Chem Toxicol 87:97–102
9. Hariri E et al (2015) Carcinogenic and neurotoxic risks of acrylamide and heavy metals from potato and corn chips consumed by the Lebanese population. J Food Compost Anal 42:91–97
10. Mesías M, Morales FJ (2015) Acrylamide in commercial potato crisps from Spanish market: Trends from 2004 to 2014 and assessment of the dietary exposure. Food Chem Toxicol 81:104–110
11. Wyka J et al (2015) Estimation of dietary exposure to acrylamide of Polish teenagers from an urban environment. Food Chem Toxicol 75:151–155
12. Wong WW et al (2014) Dietary exposure of Hong Kong adults to acrylamide: results of the first Hong Kong Total Diet Study. Food Addit Contam A 31(5):799–805
13. PERIŠ TKD (2014) Quick estimation of dietary exposure to heterocyclic aromatic amines and acrylamide in a Croatian female population. J Food Nutr Res (ISSN 1336-8672) 53(3):251–259
14. Cengiz MF, Gündüz CPB (2013) Acrylamide exposure among Turkish toddlers from selected cereal-based baby food samples. Food Chem Toxicol 60:514–519
15. Normandin L et al (2013) Dietary exposure to acrylamide in adolescents from a Canadian urban center. Food Chem Toxicol 57:75–83
16. Katz JM et al (2012) Comparison of acrylamide intake from Western and guideline based diets using probabilistic techniques and linear programming. Food Chem Toxicol 50(3-4):877–883
17. Sörgel F et al (2002) Acrylamide: increased concentrations in homemade food and first evidence of its variable absorption from food, variable metabolism and placental and breast milk transfer in humans. Chemotherapy 48(6):267–274
18. Schettgen T et al (2002) Hemoglobin adducts of ethylene oxide, propylene oxide, acrylonitrile and acrylamide–biomarkers in occupational and environmental medicine. Toxicol Lett 134(1-3):65–70
19. Abramsson-Zetterberg L et al (2005) Acrylamide tissue distribution and genotoxic effects in a common viral infection in mice. Toxicology 211(1-2):70–76
20. Fennell TR et al (2005) Metabolism and hemoglobin adduct formation of acrylamide in humans. Toxicol Sci 85(1):447–459
21. Ramu K et al (1995) Acrolein mercapturates: synthesis, characterization, and assessment of their role in the bladder toxicity of cyclophosphamide. Chem Res Toxicol 8(4):515–524
22. Wang R-S et al (2010) Mutagenicity of acrylamide and glycidamide in the testes of big blue mice. Toxicol Sci 117(1):72–80
23. Capuano E, Fogliano V (2011) Acrylamide and 5-hydroxymethylfurfural (HMF): a review on metabolism, toxicity, occurrence in food and mitigation strategies. LWT – Food Sci Technol 44(4):793–810
24. Besaratinia A, Pfeifer GP (2005) DNA adduction and mutagenic properties of acrylamide. Mutat Res/Genetic Toxicol Environ Mutagen 580(1):31–40
25. LoPachin RM (2004) The changing view of acrylamide neurotoxicity. Neurotoxicology 25(4):617–630
26. Lehning EJ et al (1998) Biochemical and morphologic characterization of acrylamide peripheral neuropathy. Toxicol Appl Pharmacol 151(2):211–221
27. LoPachin RM et al (1992) Acrylamide disrupts elemental composition and water content of rat tibial nerve: I. Myelinated axons. Toxicol Appl Pharmacol 115(1):21–34
28. Lehning EJ et al (2002) Acrylamide neuropathy: II. Spatiotemporal characteristics of nerve cell damage in brainstem and spinal cord. Neurotoxicology 23(3):415–429

29. Lehning EJ et al (2002) Acrylamide neuropathy: I. Spatiotemporal characteristics of nerve cell damage in rat cerebellum. Neurotoxicology 23(3):397–414
30. Lehning EJ et al (2002) Acrylamide neuropathy. III. Spatiotemporal characteristics of nerve cell damage in forebrain. Neurotoxicology 24:125–136
31. LoPachin RM et al (2002) Nerve terminals as the primary site of acrylamide action: a hypothesis. Neurotoxicology 23(1):43–59
32. Barber DS et al (2007) Proteomic analysis of rat striatal synaptosomes during acrylamide intoxication at a low dose rate. Toxicol Sci 100(1):156–167
33. Martyniuk CJ et al (2011) Molecular mechanism of glyceraldehyde-3-phosphate dehydrogenase inactivation by α,β-unsaturated carbonyl derivatives. Chem Res Toxicol 24(12):2302–2311
34. Zhang L et al (2011) Role of the Nrf2-ARE pathway in acrylamide neurotoxicity. Toxicol Lett 205(1):1–7
35. Lin RC, Scheller RH (2000) Mechanisms of synaptic vesicle exocytosis. Annu Rev Cell Dev Biol 16(1):19–49
36. Barber DS, LoPachin RM (2004) Proteomic analysis of acrylamide-protein adduct formation in rat brain synaptosomes. Toxicol Appl Pharmacol 201(2):120–136
37. Luo J et al (2007) Mechanisms of acrolein-induced myocardial dysfunction: implications for environmental and endogenous aldehyde exposure. Am J Physiol-Heart Circ Physiol 293(6):H3673–H3H84
38. Wang G-W et al (2008) Acrolein consumption exacerbates myocardial ischemic injury and blocks nitric oxide-induced PKCε signaling and cardioprotection. J Mol Cell Cardiol 44(6):1016–1022
39. Conklin DJ et al (2010) Acrolein consumption induces systemic dyslipidemia and lipoprotein modification. Toxicol Appl Pharmacol 243(1):1–12
40. Ismahil MA et al (2011) Chronic oral exposure to the aldehyde pollutant acrolein induces dilated cardiomyopathy. Am J Physiol-Heart Circ Physiol 301(5):H2050–H2H60
41. Srivastava S et al (2011) Oral exposure to acrolein exacerbates atherosclerosis in apoE-null mice. Atherosclerosis 215(2):301–308
42. Ansari MA, Scheff SW (2010) Oxidative stress in the progression of alzheimer disease in the frontal cortex. J Neuropathol Exp Neurol 69(2):155–167
43. Singh M et al (2010) Role of by-products of lipid oxidation in Alzheimer's disease brain: a focus on acrolein. J Alzheimers Dis 21(3):741–756
44. Sultana R, Butterfield DA (2010) Role of oxidative stress in the progression of Alzheimer's disease. J Alzheimers Dis 19(1):341
45. LoPachin RM et al (2008) Type-2 alkenes mediate synaptotoxicity in neurodegenerative diseases. Neurotoxicology 29(5):871–882
46. LoPachin RM et al (2009) Molecular mechanisms of 4-hydroxy-2-nonenal and acrolein toxicity: nucleophilic targets and adduct formation. Chem Res Toxicol 22(9):1499–1508
47. LoPachin RM et al (2009) Synaptosomal toxicity and nucleophilic targets of 4-hydroxy-2-nonenal. Toxicol Sci 107(1):171–181
48. Grattagliano I (2009) Current concepts of mechanisms in drug-induced hepatotoxicity. Curr Med Chem 16(23):3041
49. Zhang L et al (2012) Protective effect of allicin against acrylamide-induced hepatocyte damage in vitro and in vivo. Food Chem Toxicol 50(9):3306–3312
50. Sen A et al (2012) Diverse action of acrylamide on cytochrome P450 and glutathione S-transferase isozyme activities, mRNA levels and protein levels in human hepatocarcinoma cells. Cell Biol Toxicol 28(3):175–186
51. Zhao M et al (2015) Blueberry anthocyanins extract inhibits acrylamide-induced diverse toxicity in mice by preventing oxidative stress and cytochrome P450 2E1 activation. J Funct Foods 14:95–101

52. Zhao M et al (2015) The chemoprotection of a blueberry anthocyanin extract against the acrylamide-induced oxidative stress in mitochondria: unequivocal evidence in mice liver. Food Funct 6(9):3006–3012
53. Rice JM (2005) The carcinogenicity of acrylamide. Mutat Res/Genetic Toxicol Environ Mutagen 580(1):3–20
54. Johnson KA et al (1986) Chronic toxicity and oncogenicity study on acrylamide incorporated in the drinking water of Fischer 344 rats. Toxicol Appl Pharmacol 85(2):154–168
55. Friedman MA et al (1995) A lifetime oncogenicity study in rats with acrylamide. Fundam Appl Toxicol 27(1):95–105
56. National Center for Toxicological Research (NCTR) cancer bioassays for acrylamide and its mutagenic metabolite, glycidamide. NCTR. 2008
57. Besaratinia A, Pfeifer GP (2007) A review of mechanisms of acrylamide carcinogenicity. Carcinogenesis 28(3):519–528
58. Mei N et al (2008) Genotoxic effects of acrylamide and glycidamide in mouse lymphoma cells. Food Chem Toxicol 46(2):628–636
59. Lamy E et al (2008) Ethanol enhanced the genotoxicity of acrylamide in human, metabolically competent HepG2 cells by CYP2E1 induction and glutathione depletion. Int J Hyg Environ Health 211(1):74–81
60. Sickles DW et al (2007) Acrylamide effects on kinesin-related proteins of the mitotic/meiotic spindle. Toxicol Appl Pharmacol 222(1):111–121
61. Chatzizacharias NA et al (2008) Disruption of FAK signaling: a side mechanism in cytotoxicity. Toxicology 245(1):1–10
62. Mottram DS et al (2002) Acrylamide is formed in the Maillard reaction. Nature 419:448
63. Stadler RH et al (2002) Acrylamide from Maillard reaction products. Nature 419:449
64. Zyzak DV et al (2003) Acrylamide formation mechanism in heated foods. J Agric Food Chem 51(16):4782–4787
65. Yaylayan VA et al (2003) Why asparagine needs carbohydrates to generate acrylamide. J Agric Food Chem 51(6):1753–1757
66. Grigg R et al (1989) X=Y-ZH compounds as potential 1,3-dipoles. Part 231,2 mechanisms of the reactions of ninhydrin and phenalene trion with ∝-amino acids. X-ray crystal structure of protonated ruhemann's purple, a stable azomethine ylide. Tetrahedron 45(12):3849–3862
67. Manini P et al (2001) An unusual decarboxylative Maillard reaction between l-DOPA and d-glucose under biomimetic conditions: factors governing competition with pictet−spengler condensation. J Org Chem 66(15):5048–5053
68. Granvogl M et al (2004) Quantitation of 3-aminopropionamide in potatoes-a minor but potent precursor in acrylamide formation. J Agric Food Chem 52(15):4751–4757
69. Yaylayan VA, Stadler RH (2005) Acrylamide formation in food: a mechanistic perspective. J AOAC Int 88(1):262
70. Becalski A et al (2003) Acrylamide in foods: occurrence, sources, and modeling. J Agric Food Chem 51(3):802–808
71. Yasuhara A et al (2003) Gas chromatographic investigation of acrylamide formation in browning model systems. J Agric Food Chem 51(14):3999–4003
72. Vattem DA, Shetty K (2003) Acrylamide in food: a model for mechanism of formation and its reduction. Innov Food Sci Emerg Technol 4(3):331–338
73. Gertz C, Klostermann S (2002) Analysis of acrylamide and mechanisms of its formation in deep-fried products. Eur J Lipid Sci Technol 104(11):762–771
74. Yaylayan VA et al (2004) The role of creatine in the generation of n-methylacrylamide: a new toxicant in cooked meat. J Agric Food Chem 52(17):5559–5565
75. Mestdagh F et al (2008) Importance of oil degradation components in the formation of acrylamide in fried foodstuffs. J Agric Food Chem 56(15):6141–6144
76. Gökmen V et al (2009) Multiple-stage extraction strategy for the determination of acrylamide in foods. J Food Compost Anal 22(2):142–147

77. Abd El-Hady D, Albishri HM (2015) Simultaneous determination of acrylamide, asparagine and glucose in food using short chain methyl imidazolium ionic liquid based ultrasonic assisted extraction coupled with analyte focusing by ionic liquid micelle collapse capillary electrophoresis. Food Chem 188:551–558
78. Albishri HM, El-Hady DA (2014) Eco-friendly ionic liquid based ultrasonic assisted selective extraction coupled with a simple liquid chromatography for the reliable determination of acrylamide in food samples. Talanta 118:129–136
79. S-y S et al (2012) A facile detection of acrylamide in starchy food by using a solid extraction-GC strategy. Food Control 26(2):220–222
80. Can NO, Arli G (2014) Analysis of acrylamide in traditional and nontraditional foods in turkey using HPLC–DAD with spe cleanup. J Liq Chromatogr Rel Technol 37(6):850–863
81. Zhang W et al (2014) Determination of trace acrylamide in starchy foodstuffs by hplc using a novel mixed-mode functionalized calixarene sorbent for solid-phase extraction cleanup. J Agric Food Chem 62(26):6100–6107
82. Zhao H et al (2014) Preparation and application of immobilised ionic liquid in solid-phase extraction for determination of trace acrylamide in food samples coupled with high-performance liquid chromatography. J Sci Food Agric 94(9):1787–1793
83. Bortolomeazzi R et al (2012) Rapid mixed mode solid phase extraction method for the determination of acrylamide in roasted coffee by HPLC–MS/MS. Food Chem 135(4):2687–2693
84. Cagliero C et al (2016) Matrix-compatible sorbent coatings based on structurally-tuned polymeric ionic liquids for the determination of acrylamide in brewed coffee and coffee powder using solid-phase microextraction. J Chromatogr A 1459:17–23
85. Cagliero C et al (2016) Determination of acrylamide in brewed coffee and coffee powder using polymeric ionic liquid-based sorbent coatings in solid-phase microextraction coupled to gas chromatography–mass spectrometry. J Chromatogr A 1449:2–7
86. Qu Y et al (2013) Electropolymerization of single-walled carbon nanotubes composited with polypyrrole as a solid-phase microextraction fiber for the detection of acrylamide in food samples using GC with electron-capture detection. J Sep Sci 36(24):3889–3895
87. Ghiasvand AR, Hajipour S (2016) Direct determination of acrylamide in potato chips by using headspace solid-phase microextraction coupled with gas chromatography-flame ionization detection. Talanta 146:417–422
88. Chen L et al (2009) Determination of acrylamide in foods by solid phase microextraction-gas chromatography. Food Sci Biotechnol 18(4):895–899
89. Kristenson EM et al (2006) Recent advances in matrix solid-phase dispersion. Trends Anal Chem 25(2):96–111
90. Barker SA (2007) Matrix solid phase dispersion (MSPD). J Biochem Bioph Methods 70(2):151–162
91. Bogialli S, Di Corcia A (2007) Matrix solid-phase dispersion as a valuable tool for extracting contaminants from foodstuffs. J Biochem Bioph Methods 70(2):163–179
92. Soares CMD, Fernandes JO (2008) MSPD method to determine acrylamide in food. Food Anal Methods 2(3):197
93. Zhao H et al (2015) Preparation and application of chitosan-grafted multiwalled carbon nanotubes in matrix solid-phase dispersion extraction for determination of trace acrylamide in foods through high-performance liquid chromatography. Food Anal Methods 8(5):1363–1371
94. X-m X et al (2013) Simultaneous determination of 3-monochloropropane-1,2-diol and acrylamide in food by gas chromatography-triple quadrupole mass spectrometry with coupled column separation. Anal Chim Acta 760:93–99
95. Bagdonaite K et al (2008) Determination of acrylamide during roasting of coffee. J Agric Food Chem 56(15):6081–6086
96. Shi Z et al (2009) Ultrasonic-assisted precolumn derivatization-HPLC determination of acrylamide formed in Radix Asparagi during heating process. J Pharm Biomed Anal 49(4):1045–1047

97. Alpmann A, Morlock G (2008) Rapid and sensitive determination of acrylamide in drinking water by planar chromatography and fluorescence detection after derivatization with dansulfinic acid. J Sep Sci 31(1):71–77
98. Alpmann A, Morlock G (2009) Rapid and cost-effective determination of acrylamide in coffee by planar chromatography and fluorescence detection after derivatization with dansulfinic acid. J AOAC Int 92(3):725–729
99. Lagalante AF, Felter MA (2004) Silylation of acrylamide for analysis by solid-phase microextraction/gas chromatography/ion-trap mass spectrometry. J Agric Food Chem 52(12):3744–3748
100. Cengiz MF, Boyacı Gündüz CP (2014) An eco-friendly, quick and cost-effective method for the quantification of acrylamide in cereal-based baby foods. J Sci Food Agric 94(12):2534–2540
101. Surma M et al (2016) Development of a sample preparation method for acrylamide determination in cocoa via silylation. Anal Methods 8(29):5874–5880
102. Molina-Garcia L et al (2015) Acrylamide in chips and french fries: a novel and simple method using xanthydrol for its GC-MS determination. Food Anal Methods 8(6):1436–1445
103. Mastovska K, Lehotay SJ (2006) Rapid sample preparation method for LC− MS/MS or GC− MS analysis of acrylamide in various food matrices. J Agric Food Chem 54(19):7001–7008
104. Oracz J et al (2011) New trends in quantification of acrylamide in food products. Talanta 86:23–34
105. Surma M et al (2017) Optimization of QuEChERS sample preparation method for acrylamide level determination in coffee and coffee substitutes. Microchem J 131:98–102
106. De Paola EL et al (2017) Determination of acrylamide in dried fruits and edible seeds using QuEChERS extraction and LC separation with MS detection. Food Chem 217:191–195
107. Omar MMA et al (2015) Determination of acrylamide in Sudanese food by high performance liquid chromatography coupled with LTQ Orbitrap mass spectrometry. Food Chem 176:342–349
108. Longhua X et al (2012) Determination of trace acrylamide in potato chip and bread crust based on SPE and HPLC. Chromatographia 75(5):269–274
109. Jiang D et al (2012) Preparation and application of acrylamide molecularly imprinted composite solid-phase extraction materials. Analytical Methods 4(11):3760–3766
110. Backe WJ et al (2014) The determination of acrylamide in environmental and drinking waters by large-volume injection – hydrophilic-interaction liquid chromatography and tandem mass spectrometry. J Chromatogr A 1334:72–78
111. Wang H et al (2013) HPLC-UV quantitative analysis of acrylamide in baked and deep-fried Chinese foods. J Food Compost Anal 31(1):7–11
112. Wang H et al (2008) SPE/HPLC/UV studies on acrylamide in deep-fried flour-based indigenous Chinese foods. Microchem J 89(2):90–97
113. Paleologos EK, Kontominas MG (2005) Determination of acrylamide and methacrylamide by normal phase high performance liquid chromatography and UV detection. J Chromatogr A 1077(2):128–135
114. Troise AD et al (2014) Quantitation of acrylamide in foods by high-resolution mass spectrometry. J Agric Food Chem 62(1):74–79
115. Lim H-H, Shin H-S (2014) A new derivatization approach with d-cysteine for the sensitive and simple analysis of acrylamide in foods by liquid chromatography–tandem mass spectrometry. J Chromatogr A 1361:117–124
116. Zhang Y et al (2011) Ultra high-performance liquid chromatography−tandem mass spectrometry for the simultaneous analysis of asparagine, sugars, and acrylamide in maillard reactions. Anal Chem 83(9):3297–3304
117. Zhang C et al (2016) Isotope internal standard method for determination of four acrylamide compounds in food contact paper products and food simulants by ultra-high performance liquid chromatography tandem mass spectrometry. Food Anal Methods 9(7):1895–1903
118. Dunovská L et al (2006) Direct determination of acrylamide in food by gas chromatography–high-resolution time-of-flight mass spectrometry. Anal Chim Acta 578(2):234–240

119. Zhang Y et al (2006) Rapid determination of acrylamide contaminant in conventional fried foods by gas chromatography with electron capture detector. J Chromatogr A 1116(1):209–216
120. Notardonato I et al (2013) Validation of a novel derivatization method for GC–ECD determination of acrylamide in food. Anal Bioanal Chem 405(18):6137–6141
121. Wenzl T et al (2006) Collaborative trial validation study of two methods, one based on high performance liquid chromatography–tandem mass spectrometry and on gas chromatography–mass spectrometry for the determination of acrylamide in bakery and potato products. J Chromatogr A 1132(1):211–218
122. Fernandes JO, Soares C (2007) Application of matrix solid-phase dispersion in the determination of acrylamide in potato chips. J Chromatogr A 1175(1):1–6
123. Lee MR et al (2007) Determination of acrylamide in food by solid-phase microextraction coupled to gas chromatography–positive chemical ionization tandem mass spectrometry. Anal Chim Acta 582(1):19–23
124. Negoiță M, Culeţu A (2016) Application of an accurate and validated method for identification and quantification of acrylamide in bread, biscuits and other bakery products using GC-MS/MS system. J Braz Chem Soc 27(10):1782–1791
125. Kim SH et al (2011) Analysis of acrylamide using gas chromatography-nitrogen phosphorus detector (GC-NPD). Food Sci Biotechnol 20(3):835–839
126. Tas CA et al (2010) Determination of acrylamide in foods by standard gas chromatography-mass selective detection. Agro Food Indus Hi Tech 21:5
127. Kepekci Tekkeli SE et al (2012) A review of current methods for the determination of acrylamide in food products. Food Anal Methods 5(1):29–39
128. Chen Q et al (2011) Determination of acrylamide in potato crisps by capillary electrophoresis with quantum dot-mediated LIF detection. Electrophoresis 32(10):1252–1257
129. Gezer PG et al (2016) Detection of acrylamide using a biodegradable zein-based sensor with surface enhanced Raman spectroscopy. Food Control 68:7–13
130. Zhu Y et al (2016) An indirect competitive enzyme-linked immunosorbent assay for acrylamide detection based on a monoclonal antibody. Food Agric Immunol 27(6):796–805
131. Zhu Y et al (2016) The kinetics of the inhibition of acrylamide by glycine in potato model systems. J Sci Food Agric 96(2):548–554
132. Quan Y et al (2011) Development of an enhanced chemiluminescence ELISA for the rapid detection of acrylamide in food products. J Agric Food Chem 59(13):6895–6899
133. Wu J et al (2014) Hapten synthesis and development of a competitive indirect enzyme-linked immunosorbent assay for acrylamide in food samples. J Agric Food Chem 62(29):7078–7084
134. Singh G et al (2014) Development of a highly sensitive competitive indirect enzyme-linked immunosorbent assay for detection of acrylamide in foods and water. Food Anal Methods 7(6):1298–1304
135. Liu X et al (2016) Electrochemical sensor based on imprinted sol-gel polymer on au NPs-MWCNTs-CS modified electrode for the determination of acrylamide. Food Anal Method 9:114–121
136. Zhu Y et al (2014) Highly sensitive electrochemical sensor using a MWCNTs/GNPs-modified electrode for lead (II) detection based on Pb2+-induced G-rich DNA conformation. Analyst 139(19):5014–5020
137. Wang X et al (2014) Direct, reagentless electrochemical detection of the BIR3 domain of X-linked inhibitor of apoptosis protein using a peptide-based conducting polymer sensor. Biosens Bioelectron 61:57–62
138. Dutta MK et al (2015) A computer vision based technique for identification of acrylamide in potato chips. Comput Electron Agric 119:40–50
139. Dutta MK et al (2016) An imaging technique for acrylamide identification in potato chips in wavelet domain. LWT – Food Sci Technol 65:987–998
140. Adedipe OE et al (2016) Development and validation of a near-infrared spectroscopy method for the prediction of acrylamide content in French-fried potato. J Agric Food Chem 64(8):1850–1860

141. Marchettini N et al (2013) Determination of acrylamide in local and commercial cultivar of potatoes from biological farm. Food Chem 136(3):1426–1428
142. Postles J et al (2013) Effects of variety and nutrient availability on the acrylamide-forming potential of rye grain. J Cereal Sci 57(3):463–470
143. Sanny M et al (2012) Is lowering reducing sugars concentration in French fries an effective measure to reduce acrylamide concentration in food service establishments? Food Chem 135(3):2012–2020
144. Zhu X et al (2016) Silencing of vacuolar invertase and asparagine synthetase genes and its impact on acrylamide formation of fried potato products. Plant Biotechnol J 14(2):709–718
145. Elmore JS et al (2007) Changes in free amino acids and sugars in potatoes due to sulfate fertilization and the effect on acrylamide formation. J Agric Food Chem 55(13):5363–5366
146. Elmore JS et al (2010) Effects of sulphur nutrition during potato cultivation on the formation of acrylamide and aroma compounds during cooking. Food Chem 122(3):753–760
147. Muttucumaru N et al (2008) Reducing acrylamide precursors in raw materials derived from wheat and potato. J Agric Food Chem 56(15):6167–6172
148. Yoshida M et al (2005) Acrylamide in japanese processed foods and factors affecting acrylamide level in potato chips and tea. Adv Exp Med Biol 561:405–413
149. Anese M et al (2014) Effect of vacuum roasting on acrylamide formation and reduction in coffee beans. Food Chem 145(7):168–172
150. Mestdagh F et al (2007) Influence of oil degradation on the amounts of acrylamide generated in a model system and in French fries. Food Chem 100(3):1153–1159
151. Shojaee-Aliabadi S (2013) Acrylamide reduction in potato chips by selection of potato variety grown in Iran and processing conditions. J Sci Food Agric 93:2556–2561
152. Banchero M et al (2013) Supercritical fluid extraction as a potential mitigation strategy for the reduction of acrylamide level in coffee. J Food Eng 115(3):292–297
153. Rydberg P et al (2003) Investigations of factors that influence the acrylamide content of heated foodstuffs. J Agric Food Chem 51(24):7012–7018
154. Truong V-D et al (2014) Processing treatments for mitigating acrylamide formation in sweetpotato french fries. J Agric Food Chem 62(1):310–316
155. Tuta S et al (2010) Effect of microwave pre-thawing of frozen potato strips on acrylamide level and quality of French fries. J Food Eng 97(2):261–266
156. Mestdagh F et al (2008) Impact of additives to lower the formation of acrylamide in a potato model system through pH reduction and other mechanisms. Food Chem 107(1):26–31
157. Pedreschi F et al (2006) Acrylamide content and color development in fried potato strips. Food Res Int 39(1):40–46
158. Jung MY et al (2003) A novel technique for limitation of acrylamide formation in fried and baked corn chips and in french fries. J Food Sci 68(4):1287–1290
159. Sadd PA et al (2008) Effectiveness of methods for reducing acrylamide in bakery products. J Agric Food Chem 56(15):6154–6161
160. Granda C et al (2010) Reduction of acrylamide formation in potato chips by low-temperature vacuum frying. J Food Sci 69(8):E405–EE11
161. Wei JC (2015) Study on microwave-infrared combined baking equipment and optimization of cookie baking process parameters. Master degree thesis of China Agricultural University. 2015; [In Chinese]
162. Anese M et al (2011) Modelling the effect of asparaginase in reducing acrylamide formation in biscuits. Food Chem 126(2):435–440
163. Capuano E et al (2009) Effect of flour type on Maillard reaction and acrylamide formation during toasting of bread crisp model systems and mitigation strategies. Food Res Int 42(9):1295–1302
164. Cha M (2013) Enzymatic control of the acrylamide level in coffee. Eur Food Res Technol 236(3):567–571
165. Onishi Y et al (2015) Effective treatment for suppression of acrylamide formation in fried potato chips using L-asparaginase from Bacillus subtilis. Biotechnology 5:783–789

166. Dias FFG et al (2017) Acrylamide mitigation in French fries using native l-asparaginase from Aspergillus oryzae CCT 3940. LWT – Food Sci Technol 76:222–229
167. Sun Z et al (2016) A novel bacterial type II l-asparaginase and evaluation of its enzymatic acrylamide reduction in French fries. Int J Biol Macromol 92:232–239
168. Bhagat J et al (2016) Single step purification of asparaginase from endophytic bacteria Pseudomonas oryzihabitans exhibiting high potential to reduce acrylamide in processed potato chips. Food Bioprod Process 99:222–230
169. Shi R et al (2017) Biochemical characterization of a novel L-asparaginase from Paenibacillus barengoltzii being suitable for acrylamide reduction in potato chips and mooncakes. Int J Biol Macromol 96:93–99
170. Pedreschi F et al (2004) Reduction of acrylamide formation in potato slices during frying. LWT – Food Sci Technol 37(6):679–685
171. Zuo S et al (2015) Reduction of acrylamide level through blanching with treatment by an extremely thermostable l-asparaginase during French fries processing. Extremophiles 19(4):841–851
172. Bartkiene E et al (2013) Study on the reduction of acrylamide in mixed rye bread by fermentation; with bacteriocin-like inhibitory substances producing lactic acid; bacteria in combination with Aspergillus niger glucoamylase. Food Control 30(1):35–40
173. Zhou W et al (2015) The effect of biological (yeast) treatment conditions on acrylamide formation in deep-fried potatoes. Food Sci Biotechnol 24(2):561–566
174. Dastmalchi F et al (2016) The impact of lactobacillus plantarum, paracasei, casei–casei, and sanfranciscensis on reducing acrylamide in wheat bread. J Agric Sci Technol 18:1793–1805
175. Bartkiene E et al (2016) Reducing of acrylamide formation in wheat biscuits supplemented with flaxseed and lupine. LWT – Food Sci Technol 65:275–282
176. Kolek E et al (2007) Nonisothermal kinetics of acrylamide elimination and its acceleration by table salt – a model study. J Food Sci 72(6):E341–E3E4
177. Pedreschi F et al (2007) Color kinetics and acrylamide formation in NaCl soaked potato chips. J Food Eng 79(3):989–997
178. Pedreschi F et al (2010) Acrylamide mitigation in potato chips by using NaCl. Food Bioprocess Technol 3(6):917–921
179. Kolek E et al (2006) Effect of NaCl on the decrease of acrylamide content in a heat-treated model food matrix. J Food Nutr Res 45(1):17
180. Kolek E et al (2006) Inhibition of acrylamide formation in asparagine/d -glucose model system by NaCl addition. Eur Food Res Technol 224(2):283–284
181. Kim MK et al (2012) Consumer awareness of salt and sodium reduction and sodium labeling. J Food Sci 77(9):S307–SS13
182. Friedman M, Levin CE (2008) Review of methods for the reduction of dietary content and toxicity of acrylamide. J Agric Food Chem 56(15):6113–6140
183. Graf M et al (2006) Reducing the acrylamide content of a semi-finished biscuit on industrial scale. LWT – Food Sci Technol 39(7):724–728
184. Açar ÖÇ et al (2012) Effect of calcium on acrylamide level and sensory properties of cookies. Food Bioprocess Technol 5(2):519–526
185. Gökmen V, Şenyuva HZ (2007) Acrylamide formation is prevented by divalent cations during the Maillard reaction. Food Chem 103(1):196–203
186. Gökmen V, Şenyuva HZ (2007) Effects of some cations on the formation of acrylamide and furfurals in glucose-asparagine model system. Eur Food Res Technol 225(5-6):815–820
187. Kalita D, Jayanty SS (2013) Reduction of acrylamide formation by vanadium salt in potato French fries and chips. Food Chem 138(1):644–649
188. Moreau L et al (2009) Influence of sodium chloride on colour, residual volatiles and acrylamide formation in model systems and breakfast cereals. Int J Food Sci Technol 12:2407–2416
189. Hughes JC et al (1975) Texture of cooked potatoes: The effect of ions and ph on the compressive strength of cooked potatoes. J Sci Food Agric 26(6):739–748

190. Mestdagh F et al (2008) Impact of chemical pre-treatments on the acrylamide formation and sensorial quality of potato crisps. Food Chem 106(3):914–922
191. Zeng X et al (2009) Inhibition of acrylamide formation by vitamins in model reactions and fried potato strips. Food Chem 116(1):34–39
192. Arribaslorenzo G, Morales FJ (2009) Effect of pyridoxamine on acrylamide formation in a glucose/asparagine model system. J Agric Food Chem 57(3):901–909
193. Kamkar A et al (2015) The inhibitory role of autolysed yeast of Saccharomyces cerevisiae, vitamins B3 and B6 on acrylamide formation in potato chips. Toxin Rev 34(1):1–5
194. Cheng KW et al (2010) Effects of fruit extracts on the formation of acrylamide in model reactions and fried potato crisps. J Agric Food Chem 58(1):309–312
195. Zhu F et al (2009) Evaluation of the effect of plant extracts and phenolic compounds on reduction of acrylamide in an asparagine/glucose model system by RP-HPLC-DAD. J Sci Food Agric 89:1674–1681
196. Zhang Y et al (2007) Addition of antioxidant from bamboo leaves as an effective way to reduce the formation of acrylamide in fried chicken wings. Food Addit Contam 24(3):242–251
197. Khanam UKS et al (2012) Phenolic acids, flavonoids and total antioxidant capacity of selected leafy vegetables. J Funct Foods 4(4):979–987
198. Wang Y, Ho C-T (2009) Polyphenolic chemistry of tea and coffee: a century of progress. J Agric Food Chem 57(18):8109–8114
199. Ou S et al (2010) Effect of antioxidants on elimination and formation of acrylamide in model reaction systems. J Hazard Mater 182(1):863–868
200. Jin C et al (2013) Relationship between antioxidants and acrylamide formation: a review. Food Res Int 51(2):611–620
201. Tareke E (2003) Identification and origin of potential background carcinogens: endogenous isoprene and oxiranes, dietary acrylamide [Doctoral thesis, comprehensive summary]. Department of Environmental Chemistry, Stockholm
202. Zhu F et al (2011) Dietary plant materials reduce acrylamide formation in cookie and starch-based model systems. J Sci Food Agric 91(13):2477–2483
203. Li J et al (2012) Effect of water migration between arabinoxylans and gluten on baking quality of whole wheat bread detected by magnetic resonance imaging (MRI). J Agric Food Chem 60(26):6507–6514
204. Kotsiou K et al (2011) Effect of standard phenolic compounds and olive oil phenolic extracts on acrylamide formation in an emulsion system. Food Chem 124(1):242–247
205. Zhang Y, Zhang Y (2008) Effect of natural antioxidants on kinetic behavior of acrylamide formation and elimination in low-moisture asparagine–glucose model system. J Food Eng 85(1):105–115
206. Hedegaard RV et al (2008) Acrylamide in bread. Effect of prooxidants and antioxidants. Eur Food Res Technol 227(2):519–525
207. Ou S et al (2008) Reduction of acrylamide formation by selected agents in fried potato crisps on industrial scale. Innov Food Sci Emerg Technol 9(1):116–121
208. Bråthen E et al (2005) Addition of glycine reduces the content of acrylamide in cereal and potato products. J Agric Food Chem 53(8):3259–3264
209. Claeys WL et al (2005) Effect of amino acids on acrylamide formation and elimination kinetics. Biotechnol Prog 21(5):1525–1530
210. Liu J et al (2011) The pathways for the removal of acrylamide in model systems using glycine based on the identification of reaction products. Food Chem 128(2):442–449
211. Low MY et al (2006) Effect of citric acid and glycine addition on acrylamide and flavor in a potato model system. J Agric Food Chem 54(16):5976–5983
212. Casado FJ et al (2010) Reduction of acrylamide content of ripe olives by selected additives. Food Chem 119(1):161–166
213. Hao R et al (2011) Acrylamide-taurine adducts formation as a key mechanism for taurine's inhibitory effect on acrylamide formation. Int J Food Sci Technol 46(6):1282–1288

214. Shin D-C et al (2010) Reduction of acrylamide by taurine in aqueous and potato chip model systems. Food Res Int 43(5):1356–1360
215. Cook DJ, Taylor AJ (2005) On-line MS/MS monitoring of acrylamide generation in potato- and cereal-based systems. J Agric Food Chem 53(23):8926–8933
216. Sansano M et al (2016) Protective effect of chitosan on acrylamide formation in model and batter systems. Food Hydrocolloids 60:1–6