Chapter 7

Mitigation of Acrylamide in Foods: An African Perspective

Oluwafemi Ayodeji Adebo, Eugenie Kayitesi, Janet Adeyinka Adebiyi, Sefater Gbashi, Makumba Chewe Temba, Adeseye Lasekan, Judith Zanele Phoku and Patrick Berka Njobeh

Additional information is available at the end of the chapter

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Abstract

Acrylamide (ACR) is a possible human carcinogen, with neurotoxic properties. It is a heat-generated food toxicant particularly found in carbohydrate-rich foods. Its occurrence is of global concern and constitutes a major challenge to food safety, due to its presence in several thermally processed foods worldwide. Since its discovery, ACR has been recognized as one of the most widely investigated heat-induced food contaminant, and several reports on its formation and occurrence since its discovery have been reported. However, information on the extent of ACR occurrence in foods consumed in different parts of Africa is rather too limited. This is particularly a concern considering that most carbohydrate-based foods, subjected to varying degrees of thermal processing, are consumed as staple diets almost on daily basis in the continent. As such, African populations may be exposed to high levels of ACR daily. Thus, this chapter covers the formation, occurrence and health impact of ACR in foods. It further summarizes previous studies looking at ACR reduction and mitigation strategies, especially those that may be applicable in the continent. Adequate sensitization of the populace about the prevention of ACR as a food contaminant is essential to ensure the safety of heat-processed carbohydrate-rich foods in the continent.

Keywords: acrylamide, prevention, toxicity, heat-processed foods, Africa

1. Introduction

The prevalence of acrylamide (ACR) in ready-to-eat diets and its toxicological effects currently on humans is a public concern. The formation of this heat-generated toxic substance in foods,

principally in carbohydrate-rich foods, was first reported by Tareke et al. [1] and has since been identified as a global challenge in the food industry. It has been classified as a potential occupational (Group 2A) carcinogen by the International Agency for Research on Cancer (IARC) and some US government agencies [2]. This is due to the fact that ACR is known to potentially exhibit carcinogenic effects in experimental animals, albeit its dietary link to human cancer. Its neurotoxicity in humans is well known from accidental and occupational exposures and experimental studies in animals which have shown genotoxic, reproductive and carcinogenic effects [3].

For over a decade since its discovery, several studies have been published in the literature on its formation, presence in various food products and toxicity in different parts of the world [1, 4–6]. In contrast, there is a dearth of information on its incidence and prevalence in Africa. This is probably why no information regarding limits regulating ACR in foods has not been established or enforced. Although other regions of the world can be affected by ACR contamination, it could be easily identified that Africa can be the most affected. It can, however, be difficult to affirm this assertion considering the lack of well-established or insufficient data on ACR levels in processed foods, degree of human exposure and risk assessment in the continent. This chapter appraises studies presenting information on the formation of ACR in foods and toxicity associated with it in other parts of the world. The main strategies for controlling or preventing its occurrence in the literature are also reviewed herein with a view of their possible adoption in Africa.

2. Discovery and properties of acrylamide

Acrylamide (IUPAC name—prop-2-enamide) (**Figure 1**) was accidentally discovered in foods in April 2002 by a group of researchers in Sweden working on heat processing technology of carbohydrate-rich foods [1, 7]. It is a white, odourless and crystalline compound with the chemical formula C_3H_5NO, molar mass of 71.08 g/mol, melting point of 84.5°C, vapour pressure of 0.007 mmHg at 25°C and boiling point at 136°C [8]. ACR is soluble in chloroform, ether, ethanol and water and decomposes in the presence of acids, oxidizing agents, bases, iron and iron salts [9] to form ammonia, carbon dioxide, carbon monoxide and oxides of nitrogen [10]. ACR is a heat-induced contaminant naturally formed during industrial processing and home cooking of many foods daily consumed around the world [8, 11].

ACR is used as a chemical intermediate in the production of polyacrylamides, which are used as a flocculating agent for sewage/wastewater treatment and other industrial applications

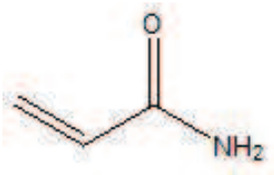

Figure 1. Chemical structure of acrylamide.

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such as those in the formulation of several cosmetics [12]. Its application as a grouting agent is also extended to the construction of dam foundations, sewers and tunnels and cosmetics and in electrophoresis gels [13]. It has also been used in pesticide production, cement formulations, ore processing, sugar manufacturing, food packaging, plastic and paper production and for the prevention of soil erosion [2]. Sequel to its detection in foods and potential toxic effects, several studies have been initiated and reported worldwide, some of which will be highlighted in the succeeding sections of this chapter.

2.1. Formation of acrylamide

ACR is principally formed via Maillard reaction involving asparagine and carbonyl sources such as reducing sugars [14]. Although asparagine may be converted to ACR by thermally induced deamination and decarboxylation, carbohydrates are necessary to effect its conversion to ACR (**Figure 2**) [15]. While several other carbonyl compounds can enhance this reaction, α-hydroxyl carbonyl compounds such as glucose or fructose are more efficient [16]. Claus et al. [14] indicated that the first step in this reaction is the formation of a Schiff base intermediate as a low-energy alternative in decarboxylating this product intact. The formed Schiff base intermediate can either hydrolyze to form 3-aminopropionamide, a precursor of ACR, or further undergo elimination reaction leading to direct formation of ACR [17]. Nevertheless, the formation of ACR from reducing sugars and asparagine in the Maillard reaction represents the main formation route [14].

The formation of acrolein and acrylic acid through the dehydration of fats when heated at high temperature has been proposed as another mechanism of ACR formation [6, 14]. The studies of Becalski et al. [19] also indicated that ACR can be formed along with ammonia from

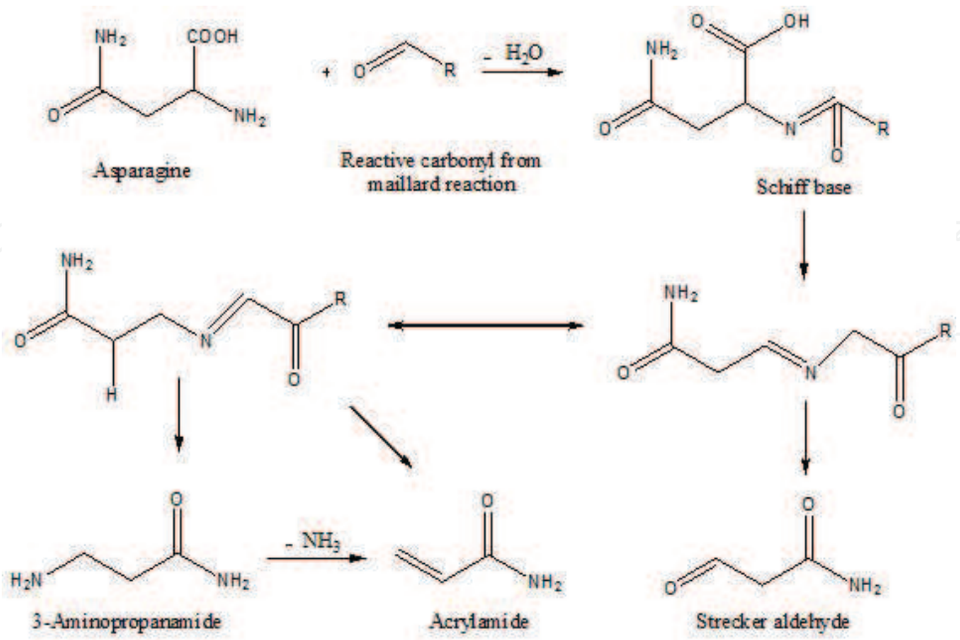

Figure 2. Proposed mechanism for the formation of the acrylamide in heat-treated foods. Adapted from Vleeschouwer et al. [18].

the degradation of amino acids. Although based on experiments with ammonium salts, acrolein and oils, this mechanism suggested might be inappropriate for ACR formation in foods [20]. Nevertheless, there are a number of factors that impact on the development of ACR in foods. Such factors are subsequently reviewed.

2.2. Factors affecting formation of acrylamide

2.2.1. Processing conditions

Food processing conditions such as time and temperature are vital factors affecting the formation and levels of ACR in food [21, 22]. However, the manner of heat transfer to foods (for instance, baking, frying, microwave heating and roasting) does not, however, necessarily impact the rate of ACR formation [23]. An exponential increase in ACR levels from 265 to 2130 μg/kg in French fries was observed when temperature increased from 150 to 190°C [21]. In potato slices with low and high surface-to-volume ratios (SVRs), ACR levels increased with increasing frying temperature and time, reaching maximum levels of 2500 and 18,000 μg/kg, respectively [24]. Studies on the effect of time and temperature on ACR formation in bread revealed that more ACR was formed in the crust layer and the levels increased with baking temperature and time [20, 25]. Roasting temperature and time had an impact on ACR formation in coffee beans [23, 26].

In Africa, traditional food processing operations and techniques are commonly applied but vary among different ethnic groups, communities and settlements. Heat processing operations such as frying, roasting and baking are common processes used in preserving and processing foods for further use. Thus, this presents a significant risk of ACR exposure. Elsewhere, it has been observed that concentrations of ACR are highly correlated with the degree of crust surface browning of processed foods [20]. These authors asserted that because ACR and brown colour of foods are formed due to Maillard reaction, it is most likely that ACR is formed in parallel with browning. Thus, the degree of surface browning (though may not necessarily indicate amount of ACR) could be used as a visual indicator of ACR formation in foods during cooking [25].

2.2.2. Substrate composition

The formation of ACR intermediates is usually determined by the amount and form of amino acids and sugars present [27]. It has thus been postulated that ACR formation and consequent concentration are relative to amino acid and sugar composition in the substrate [21, 27]. While these precursors affect ACR formation, the presence of other compounds that compete with amino acids and reducing sugars in the Maillard reaction are also vital compositional factors [21]. In potato and cereal products, ACR levels are highly correlated with glucose/fructose and asparagine concentrations [28]. Varieties in crop cultivar could also affect the reducing sugar content in the produce [29]. Short-term storage of potatoes at 4°C significantly increased the potential for ACR formation [30, 31]. Cooling potatoes to temperatures less than 10°C causes reducing sugars to increase, thereby increasing the potential for ACR formation [21, 30, 31]. Temperature and moisture levels in the food substrate are also other factors that affect ACR

formation. While temperature has been discussed in the preceding section, Matthaus et al. [32] reported that a quick reduction of the water content in the outer layers of the product (as a consequence of high temperatures) favours ACR formation.

2.2.3. Soil properties and fertilization

Both nitrogen and sulphur are important compositions of the soil, and subsequent concentrations and amount have significant effect on the formation of ACR precursors [33–36]. According to Halford et al. [34], increasing soil sulphur levels and reducing nitrogen levels can effectively decrease the levels of ACR precursors, such as asparagine [34]. In a study conducted by De Wilde et al. [33] on the influence of soil enrichment on ACR formation in potatoes, differences occurred in ACR formation from crops grown on different soils. The effect of nitrogen fertilizer usage on crops has been reported in the literature to have an impact on asparagine and reducing sugar concentrations [37]. A decrease in nitrogen fertilizer resulted in a 30–65% increase in ACR formation [33, 38]. Moderate nitrogen fertilizer with a good provision of potassium may result in low levels of free asparagine and reducing sugars in tubers [39]. This clearly indicates that mineral composition due to either fertilizer employed or soil composition may impact on the presence and concentrations of ACR precursors. Deficiencies in phosphorus, potassium and magnesium can cause asparagine levels to rise in potato and wheat [34]. An appropriate balance between levels of fertilizer application and minimum requirements of the crop while taking into account possible environmental impacts and legal fertilizer limits should be reached to obtain food products less prone to ACR formation [33].

2.3. Occurrence and levels of acrylamide in foods

There have been considerable efforts made since the discovery of ACR in foods by regulatory agencies such as the US Food and Drug Administration (FDA), the World Health Organization (WHO) and the European Commission (EC) to gather data on food levels of ACR [40, 41]. However, none of these were reported from the continent of Africa. Prevalent sources of ACR differ among countries due to differences in the diet, method of preparing foods and the nature of soil/agricultural practices [42]. Cereal-, tuber- and coffee-related products contribute mostly to the sources of ACR intake [43]. Since ACR is present in a variety of food products which are consumed on a daily basis, the risks of exposure apply to almost all consumers. Children may be more vulnerable due to their smaller body mass as compared to adults [44].

Though a summary of reports on the incidence of ACR have been reported in other parts of the world [40, 41, 43, 44], a search of literature revealed few reports from Africa. The different studies reported are summarized in **Table 1** on ACR incidence levels reaching 12626, 9499, 7310 and 6968 μg/kg in South America, Africa, the Middle East and Europe, respectively. Of the very few incidences reported so far, only a few reported on African commodities were in Ghana [45], Kenya [46] and South Sudan (**Table 1**) [47, 48]. Though majority of these commodities were mainly baked and fried, this does not suggest that other heat-intense processed foods do not lead to ACR. The death of dogs after consuming the burnt part of maize meal is worth noting [49]. The death of these dogs was ascribed to ACR poisoning by the authors and should awaken intensive research on this. Further considering the fact that maize meal is a

Product/produce	Region of the world	Acrylamide content	Reference
Arabica	Europe	48–3210	[50]
Baby biscuit	Europe	588	[51]
Baby bread—rusks	Europe	660	[51]
Baby food	Europe	0–130	[41]
Baked *gorasa*	Africa	20	[48]
Baked *minnan*	Africa	17	[48]
Baked *hilmur*	Africa	59	[48]
Baked potato chips	Middle East	329–7310	[52]
Biscuit	Asia	119	[53]
		487	[54]
		232	[55]
	Europe	4200	[56]
		1177	[51]
		214	[57]
		1514	[58]
	South America	3180	[59]
Bread	Asia	103	[55]
	Middle East	90–802[a]	[60]
	Europe	2430	[56]
		695	[51]
Bread and rolls	Europe	400	[58]
Breakfast cereal	Asia	117.3	[53]
	Europe	1600	[56]
		762	[51]
		674	[58]
	South America	2288	[59]
Cakes	Europe	13–50	[61]
Candy bars	Europe	39–61	[61]
Cantonese moon cake	Asia	207	[62]
Cereal-based baby foods	Europe	353	[56]
		11–16	[61]
Cereals	Europe	52–1057	[41]
Cereal bar	Europe	820	[58]
Chocolate	Europe	750	[58]

Product/produce	Region of the world	Acrylamide content	Reference
Chocolate powder	South America	1017	[59]
Chocolate products	Asia	96.7	[53]
Coffee	Asia	7–19	[63]
		100–668	[64]
	Europe	1158	[56]
		16–503	[65]
		3800	[58]
	South America	3797	[59]
Coffee beans	Europe	172	[66]
		48–6968	[50]
Coffee substitute	Europe	5400	[58]
Cookies	Asia	50–700	[64]
Corn-based chips	Middle East	329–6360	[52]
	South America	78–441	[59]
Crackers	Europe	2666	[51]
	South America	194–1271	[59]
Crisp *mahua*	Asia	218	[62]
Fast food	Europe	210–2680	[50]
Follow-on formula	Europe	32–312	[67]
Follow-on formula (ready to eat)	Europe	4–46	[67]
French fries	Europe	20–1325	[41]
		320	[68]
		724[c]	[69]
	Asia	135	[53]
		78–496	[63]
		940	[55]
		441	[70]
French fries (home made)	Europe	2668	[56]
French fries (fast food rest.)		210–2922	[50]
	South America	12626–12661	[59]
Fried creole	South America	83–209	[59]
Fried chicken rolls	Asia	752	[55]
Fried eggplant	Africa	338	[47]
		325	[48]

Product/produce	Region of the world	Acrylamide content	Reference
Fried instant noodle	Asia	54	[54]
Fried potato	Africa	750	[47]
		227	[48]
Fried potato chips	Middle East	375–7024	[52]
Fried puffs	Asia	524	[55]
Fried sweet potato	Africa	1043	[45]
Fried *taamia*	Africa	68	[47]
Fries	Europe	3300	[58]
Gingerbread	Europe	2100	[58]
Home-cooked potato products	Europe	2175	[56]
Hot beverages	Europe	93–5399	[41]
Infant biscuits	Europe	3–516	[67]
Infant cereals in powder	Europe	65–296	[67]
Infant cereals (ready to eat)		11–52[a]	
Infant cereal with follow-on formula in powder		17–260	
Infant cereal with follow-on formula (ready to eat)		3–46	
Infant powdered formula	South America	1821	[59]
Instant cereal-based baby food	Europe	19.2–34.7	[61]
Jarred baby foods	Europe	162	[56]
		2–162	[67]
Juice	Europe	267	[41]
Mahua	Asia	234	[62]
Moon cake	Asia	201	[62]
Non-fried instant noodles	Asia	5	[54]
Nuts products	Asia	105	[53]
Paicha	Asia	214	[62]
Plantain chips	Africa	568	[45]
Popcorn	Europe	1100	[58]
	South America	781	[59]
Potato	Europe	131–5360	[78]
Potato-based chips	Middle East	375–7310	[52]
Potato chips	Asia	1021	[54]

Product/produce	Region of the world	Acrylamide content	Reference
		151	[53]
		0.4–14	[63]
		330–2300	[64]
		723	[70]
		233	[71]
	Europe	18–1782	[50]
	Middle East	90–800[a]	[60]
	South America	82–1852	[59]
Potato crisps	Africa	4565	[72]
		ND[b]–9499	[46]
	Asia	244–1688	[73]
	Europe	30–2300	[1]
		59–2336	[74]
		4180	[56]
		2311[c]	[75]
		954[c]	[69]
		3200	[58]
	South America	40–1770	[76]
Powdered baby food	Europe	174	[51]
Robusta	Europe	160–6968	[50]
Seasoned laver	Asia	103	[53]
Soft bread	South America	102–594	[59]
Sweet *binggan*	Asia	226	[62]
Taco, tostada and tortilla products	Europe	29–794	[41]
Tajadas	Europe	240	[77]
Tea products	Asia	108	[53]
Toast	Asia	530	[71]
	Europe	460	[58]
Twisted cruller	Asia	209	[71]
Wafer	South America	687–2497	[59]
Yougao	Asia	212	[62]
Youtiao	Asia	248	[62]

[a]μg/L.
[b]ND, not detected.
[c]Average value.

Table 1. Reported occurrence of acrylamide in foods by regions.

staple food in Southern African and gets burnt during its preparation, there are indications of a huge risk of ACR exposure to millions of individuals consuming this product daily.

2.4. Toxicity of acrylamide

The neurotoxicity of ACR in humans is well known from occupational and accidental exposures [79]. Owing to its low molecular weight and polarity, ACR is readily distributed and incorporated in mammals [80]. After ingestion, ACR is rapidly circulated throughout the whole body via the bloodstream [81] and can be found in the liver, kidney, thymus, brain, heart and human breast milk [82]. The conjugation of ACR to glutathione, and its epoxidation to glycidamide in the liver via cytochrome P450, is one of the major metabolic routes [83]. The formation of glycidamide is considered to be the critical step for the toxic effects of ACR and its metabolites. ACR and glycidamide, the latter at a much higher rate, can react with macromolecules such as haemoglobin and enzymes [80]. According to the European Food Safety Authority, ACR and its metabolite glycidamide have shown evidences of genotoxicity (DNA damage) and carcinogenicity [44]. Although evidence from studies on human exposure and possible causes of cancer is currently limited, epidemiological studies designed to target different populations and different organs in relation to cancer risks have been presented, with absolutely none reported from the African continent [84].

Calleman [85] reported peripheral neuropathy symptoms of highly exposed workers in China. Characterized by numbness of hands and feet, ataxia and skeletal muscle weakness, ACR has been shown to be toxic to both the central and peripheral nervous system [86]. ACR induces nerve terminal degeneration [79] and has deleterious effects on the thalamus, hippocampus and cerebral cortex [79, 86]. A recent study demonstrated evidence of ACR neurotoxic effects of fried potato chips on rat postnatal development, causing cerebellar cortical defects and myodegeneration of the gastrocnemius muscle during the postnatal development of pups [87]. It has been postulated that neurotoxicity of ACR might be cumulative as the same neurotoxic effects can be seen at low and high doses of ACR with the low doses requiring longer exposure [86, 88].

In 1994, ACR was classified by the International Agency for Research on Cancer (IARC) as Group 2A, indicating that it is probably carcinogen to humans (Group 2A) [89, 90]. This was based on positive bioassay results in rodents, buttressed by evidence that ACR is transformed in mammalian tissues to a more reactive genotoxic metabolite (glycidamide) [8]. Evidence on experimental rodents indicates that ACR causes tumours in the skin, uterus, lungs, brain, thyroid and mammary gland [91]. The genotoxicity of ACR and glycidamide is also manifested as both clastogenicity and mutagenicity. ACR has proven to be genotoxic in vivo to the somatic and germ cells as well as to cell cultures [8] and mammalian cells [81]. As indicated by Rice [91], the oxidation of this contaminant to glycidamide is the prerequisite for genotoxicity of ACR. This is attributed to the higher reactivity of this metabolite (glycidamide) to form adducts with DNA [83].

For cancer-related effects, the margin of exposures (MOEs) of ACR have been estimated to range from 50 for high-consuming toddlers to 425 for average adult consumers. These numbers indicate concerns for public health [44]. Essentially, since any level of exposure to a genotoxic substance could possibly cause DNA damage and lead to cancer, no tolerable daily

intake (TDI) of ACR is set by European scientists [44], not to mention Africa. Nonetheless, Shipp et al. [13] reported that ACR administered to drinking water of rodents at doses of ≥5 mg/kg bw/day resulted in significant decreases in the number of live pups. At higher doses, signs of copulatory behaviour as well as effects on sperm motility and morphology were observed by these authors. ACR toxicity in male animals includes decrease in sperm number/abnormal sperm, decrease in fertility rates, degeneration of the epithelial cells of the seminiferous tubules and retarded development of pups [92]. These reproductive toxic effects may be attributed to the interfering effect of ACR on the kinesin motor proteins, resulting in a reduced sperm motility and subsequent fertilization [92].

3. Prevention and mitigation of acrylamide

Agencies such as the FAO and WHO in collaboration with the academia and food industry have put forth strategies for reducing levels of ACR in food. In Europe, food manufacturers have collaborated with researchers and the academia through the Confederation of the Food and Drink Industries of the EU (CIAA) to produce series of strategies called the 'CIAA Acrylamide Toolbox' for decreasing ACR levels in different foods [93]. However, it should be noted that designing mitigation strategies is quite challenging, considering the fact that precautions must be taken to avoid compromising the nutritional, chemical, physical and microbiological quality and safety of the food. Accordingly, such measures must not result into the formation of other process contaminants nor detrimentally affect the organoleptic properties and acceptability of the final product [20, 94].

3.1. Methods that interrupt reactions leading to acrylamide formation

Several approaches have been successful at preventing ACR formation by preventing the key reactions responsible for generating it. Lowering the pH of foods blocks the nucleophilic addition of asparagine with a carbonyl compound, preventing the formation of the Schiff base, a critical intermediate in the formation of ACR [95, 96]. While this approach could be successful in lowering ACR levels in fried potato products, it may bring about undesirable taste to foods [21]. The use of organic acids and the addition of mono- and divalent cations (Na^+ or Ca^{2+}) to foods are other approaches of mitigating ACR by preventing the Schiff base formation [95–97]. The addition of proteins or free amino acids other than asparagine has also been investigated as a strategy for reducing ACR formation by causing competitive reactions and/or covalently binding ACR via Michael addition reactions [95, 96]. These additions however had low-to-moderate success at decreasing ACR levels in both cereal-based and potato foods [98].

3.2. Treatments that reduce acrylamide precursor's levels

As asparagine and reducing sugars are the major ACR precursors in foods, eliminating either of these substrates is a viable way to reduce ACR formation [94]. Procedures for achieving this include rinsing and blanching treatments, using asparaginase, fermentation and controlling storage conditions [21].

Rinsing, blanching and soaking treatments have been effective at reducing ACR formation in potato products [4, 21]. Soaking potato slices in water before frying resulted in over 50% reduction in ACR [21]. Further experiments by blanching slices in warm or hot water removed more glucose and asparagine than ordinary water immersion [4]. Changing the design of frying units to reverse the flow direction of the heated oil may alter the thermal load, which will reduce ACR levels in finished products [99]. Blanching and soaking treatments reduce ACR formation by leaching out asparagine and sugars from the surface of the slices [4]. Using asparaginase, an enzyme which hydrolyzes asparagine into aspartic acid and ammonia, has successfully reduced ACR levels in potato and bakery products [4, 20]. Asparaginase treatment of gingerbread dough resulted in a 75% decrease in free asparagine and a 55% reduction in ACR levels in the baked products [20]. To this effect, two commercial asparaginase preparations have been developed and are available in the market: Acrylaway® (Novozymes, Denmark) and PreventAse™ (DSM Food Specialties, Denmark), respectively, synthesized from *Aspergillus oryzae* and *Aspergillus niger*. They are generally recognized as safe (GRAS) ingredients [100].

Likewise, fermentation with yeast has been identified as a way to reduce ACR through the elimination of free asparagine [28]. A 2 h fermentation of rye and whole wheat dough caused a 77 and 87% reduction in ACR levels in rye and grain breads, respectively [101]. Yeast fermentation was observed to be more effective than sourdough fermentation in reducing the asparagine content of the dough [101]. Ingredients and additives may also increase ACR formation during baking of cereal-based products. In a study by Amrein et al. [20], baking agent and ammonium bicarbonate reportedly improved ACR formation in bakery products, possibly by creating more reactive carbonyl compounds. Using an alternative baking agent (sodium hydrogen carbonate), sucrose rather than honey or inverted sugar syrup can also reduce ACR content by more than 60% [20].

3.3. Modifying processing/cooking conditions

A reduction in cooking temperatures and times can decrease ACR levels in foods. However, loss of desirable colour, flavour and texture may occur, since the Maillard reaction which is responsible for ACR formation also guarantees desirable flavour and colour compounds in heated food [28]. Conditions that minimize ACR in French fries involve optimizing frying or baking processes to obtain a surface golden in colour and crispy texture [21]. Blanching, soaking, parboiling and washing treatments may be adopted, as these can leach the reducing sugar/asparagine reactants before the subsequent cooking step [102]. Overall, prolonged baking/frying and excessive browning should be avoided to minimize ACR formation in baked and fried products. Since a linear relationship exists between ACR formation and baking process, there is a need to ensure proper and optimum cooking endpoint to minimize ACR formation. This suggests that the degree of surface browning could be used as a visual indicator of ACR formation during cooking.

3.4. Agronomic factors

Selective crop propagation is a potential strategy for controlling ACR levels by decreasing levels of ACR precursors [94]. Since the first occurrence of ACR in foods, several researchers

have demonstrated the significance of variety and cultivar selection on the formation of ACR [103]. Amrein et al. [29] found a 50-fold variation in total reducing sugars in the different potato cultivars the authors studied. Cultivars with low reducing sugars were more suitable for potato products, cooked or processed at high temperatures [29]. Konings et al. [28] established the significant impact of fertilizer application rate on ACR levels mainly due to differences in crude protein and asparagine and contents. A study by Claus et al. [38] demonstrated the effect of nitrogen-based fertilizers in causing high amounts of protein and amino acid. This resulted in increased ACR levels in breads, ranging from 10.6 to 55.6 μg/kg [38]. Producers should best adopt effective fertilizer application regimes that will subsequently yield suitable produce for processing, as this influence the levels of reducing sugars [29]. Tubers should be harvested at full maturity as selection of immature tubers for further processing increases the chances of ACR occurrence, because they have relatively higher reducing sugars and produce products with potentially higher ACR levels [104, 105]. Unfortunately, most African subsistence farmers harvest immature tubers to immediately obtain income for their needs. While this should be discouraged, effective handling, packaging and storage of produce must also be emphasized. Selection and use of crop varieties that are low in ACR precursors will most definitely help reduce ACR occurrence. Storage is also another component and practicable way of mitigating ACR in foods. While storage of potatoes at low temperatures is generally meant to minimize shrinkage and spoilage, studies have shown that low temperatures tend to increase sugar levels (an ACR precursor) [106]. Though for a short-term storage, hot temperature is desirable (this can however lead to sprouting, which can be controlled using suppressants); for long term, a minimum storage temperature of 6°C is desirable [97].

3.5. Antioxidants and other phytochemicals

According to Kahkeshani et al. [107], the correlation between antioxidants and ACR can be considered from two different points of view, namely, antioxidants as exogenous additives and as endogenous secondary metabolites. According to these authors, lack of sufficient studies and discord in the results available from literature hinders a logical judgement about the effectiveness of phytochemicals against ACR. While some reports have reported their beneficial effect, some have been shown to facilitate ACR production [107]. While these compounds can react with asparagine to produce ACR, they could also possibly react with the amide group of the intermediates in Maillard reaction and block ACR formation [107–110]. The oxidation of polyphenols to corresponding quinones, which can react with 3-animopropionamide (3-APA), thus preventing the deamination of 3-APA to ACR, has also been proposed as a mechanism for ACR reduction by these compounds [111]. Nonetheless, studies demonstrating the effectiveness of these compounds towards the reduction of ACR have been presented in the literature. Fernandez et al. [112] recorded a 50% ACR reduction after the addition of a flavonoid spice mix to potato chips. Zhang and Zhang [108] reported a 76% reduction in ACR after French fries were dipped into extracts of bamboo leaves with antioxidant properties, while a 59% decrease in ACR was recorded when fried chicken wings were dipped into same extracts [111]. Further reports in the literature on this have been adequately reviewed by Kahkeshani et al. [107] and can be consulted for further reading. Interestingly, Africa is the home to a vast and diverse number of plants and other botanicals with rich phytochemicals. Extracts of these plants and

herbs are used for various purposes prominently in traditional medicines. Application of such extracts would go a long way in the mitigation of ACR occurrence in foods.

3.6. Genetic modification

As defined by Key et al. [113], genetically modified crops/plants are those that have been genetically altered through the use of recombinant DNA technology. This may be to express a gene not native to the plants or to modify endogenous ones [113]. This issue has in the recent years attracted worldwide attention especially regarding its risk to the environment and human health. While it is widely accepted in many parts of the world, including parts of America and Asia, it still remains a controversial issue in Europe [35]. Most African continents follow the latter, as concerns regarding the immediate and long-term effects of genetically modified crops are major hindrances to adopting this technology. Although different authors, government agencies and international organizations have backed and supported genetic modification, there is still a stiff opposition against its acceptance. Inconsistencies in free sugar, amino acid and asparagine contents in crops of different cultivars (varieties) and genotypes however suggest that the varying concentration of these parameters is due to genetic variations [19, 29, 38, 114]. Consequently, fast tracking the natural breeding process through the use of genetic engineering to develop cultivars (varieties) with lower concentration of asparagine and reducing sugars should be possible and encouraged [35]. It has been shown that simultaneous silencing of the genes (StAst1 and StAst2) that encode for asparagine synthetase which is the enzyme that catalyse the formation of asparagine in potato, significantly reduced the levels of asparagine in the transgenic crop [115]. Another study demonstrated reduction in the concentration of reducing sugars, which also participate in reaction leading to ACR formation. For instance, since reducing sugar is accumulated during the cold storage of potato (cold-induced sweetening), silencing of the enzyme acid invertase resulted in potato with reduced concentration of fructose and glucose as well as low ACR concentration when processed into French fries [116]. While genetic modification continues to be controversial, the farmers, the food industry and other vital stakeholders should be proactive in the development of crop varieties that would yield lower ACR levels in food.

4. Conclusion and future prospects

This chapter gives an overview of ACR in foods, significant progress in its formation and mitigation strategies with a dearth of information in Africa. Its occurrence and exposure in other parts of the world have been extensively reviewed by other authors with little focus on the African continent. Starch-based foods and food products constitute a major and basic daily diet for millions in the developing world, particularly in Africa. Coupled with the myriad of associated traditional heat processing operations, it is justified to conclude that inhabitants in this region are exposed to high risk of ACR contamination. This is expected to stimulate interest among scientists working in the field of food safety and quality, for making better efforts towards investigating the occurrence and exposure of ACR in Africa. With such data lacking, there is also insufficient information on the impact of lower levels of exposure to ACR content,

which needs to be established. Concerted efforts must also be directed towards this, using validated models of predicting dose exposure and mechanism of toxicity relationship to assist in measuring the public health risk of ACR in foods. Furthermore, adequate enlightenment and sensitization of the populace by government agencies and the industry about the dangers and possible ways of reducing this food contaminant must be provided and emphasized.

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Author details

Oluwafemi Ayodeji Adebo[1]*, Eugenie Kayitesi[1], Janet Adeyinka Adebiyi[1], Sefater Gbashi[1], Makumba Chewe Temba[1], Adeseye Lasekan[2], Judith Zanele Phoku[3] and Patrick Berka Njobeh[1]*

*Address all correspondence to: oluwafemiadebo@gmail.com and pnjobeh@uj.ac.za

1 Department of Biotechnology and Food Technology, Faculty of Science, University of Johannesburg, Doornfontein, Gauteng, South Africa

2 Department of Food Science and Human Nutrition, University of Maine, Orono, ME, USA

3 Water and Health Research Unit, Faculty of Health Science, University of Johannesburg, Doornfontein, Gauteng, South Africa

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