Food Control 56 (2015) 135–146

Contents lists available at ScienceDirect

Food Control

journal homepage: www.elsevier.com/locate/foodcont

Review

Rapid methods for detecting acrylamide in thermally processed foods: A review

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ARTICLE INFO

Article history:
Received 22 October 2014
Received in revised form
15 March 2015
Accepted 16 March 2015
Available online 1 April 2015

Keywords:
Acrylamide
Detection
Rapid methods
Computer vision
ELISA
Electrochemical biosensing

ABSTRACT

Acrylamide (AA), a neurotoxin and potential carcinogen, has been found in various thermally processed foods such as potato chips, biscuits, and coffee. LC–MS/MS and GC–MS as standard detection methods show high sensitivity, selectivity, stability, and repeatability. However, these methods require expensive instruments, skilled technicians in laboratories, and high testing costs, and cannot meet the needs for real-time and on-line detection of AA in foods. Therefore, rapid detection methods with merits of simplicity and portability such as computer vision, ELISA, electrochemical biosensing, and fluorescent biosensing have obtained an increasing amount of attention. Reported research on rapid methods has shown similar sensitivity and selectivity, but requires less time and cost in comparison with standard methods through the use of nanomaterials and biomolecules with high affinity to AA. These improvements show great promise for high-throughput, real-time, and on-line detection of AA. This paper provides a comprehensive overview of rapid detection methods for AA in foods with comparison between rapid and standard methods. Meanwhile, suggestions for further research on rapid methods for detecting AA are also discussed based on technical challenges and industry needs.

Contents

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http://dx.doi.org/10.1016/j.foodcont.2015.03.021
0956-7135/

1. Introduction

Acrylamide (AA, 2-propenamide, C_3H_5NO (71.09 g mol^{-1}, CAS No. 79-06-1)) is a colorless and odorless crystalline produced by the hydration of acrylonitrile. It dissolves in water, alcohol, and other polar solvents, but tends to be hydrolyzed into acrylic acid in an acidic and alkali environment. Polyacrylamide can be easily produced at melting point or under ultraviolet light, which is widely used in industrial and academic fields (Friedman, 2003). Increasing research on AA in foods has been investigated in the past thirty years, involving toxicity, formation, mitigation, and detection, as shown in Fig. 1.

1.1. Toxicity of AA

In 1994, International Agency for Research on Cancer (IARC) classified AA as "Group 2A" (probably carcinogenic to humans) (Lyon, 1994), due to its neurotoxicity, carcinogenicity, and genotoxicity (Erkekoglu & Baydar, 2010; Hogervorst et al., 2010). Subsequently, AA was classified as a Category 2 carcinogen and Category 2 mutagen by European Commission (EC, 2002) as well as a substance of "very high concern" by European Chemical Agency in 2010 (ECHA, 2010).

The 72nd meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA) have systematically summarized and updated the studies of AA throughout the world since their 64th meeting (JECFA, 2005, 2011). The resulted document indicated that multi-organ tumors were discovered in experimental animals after the exposure to AA, although no significant association between AA dietary intake and an increase in multiple cancers has been established from epidemiological research (Friedman, Dulak, & Stedham, 1995; Johnson et al., 1986). The genotoxicity of AA can be presented in two ways. First, AA can be converted to its metabolite glycidamide, which is 3 times higher in mutability compared to AA and can induce point mutations in various systems (Fazendeiro, 2013). Secondly, AA can act as a Michael acceptor to form adducts with thiol, hydroxyl, and amino groups in DNA, which leads to DNA damage (Doroshyenko et al., 2009; Erkekoglu & Baydar, 2010; Hogervorst et al., 2010; Watzek et al., 2012; Zeiger et al., 2009). Workers who experience occupational exposure to AA suffer from the damage of both peripheral and central nervous systems, since the neurotoxic effects of AA are cumulative and chronic (Huang et al., 2011; Pennisi et al., 2013). The no-observed-adverse-effect level for morphological changes in the nerve systems of rats was 200 μg kg^{-1} b.w. per day. Tardiff, Gargas, Kirman, Leigh Carson, and Sweeney (2010) reported that the tolerable daily intake (TDI) of AA was 40 μg kg^{-1} per day for neurotoxicity, and 2.6 μg kg^{-1} per day for cancer.

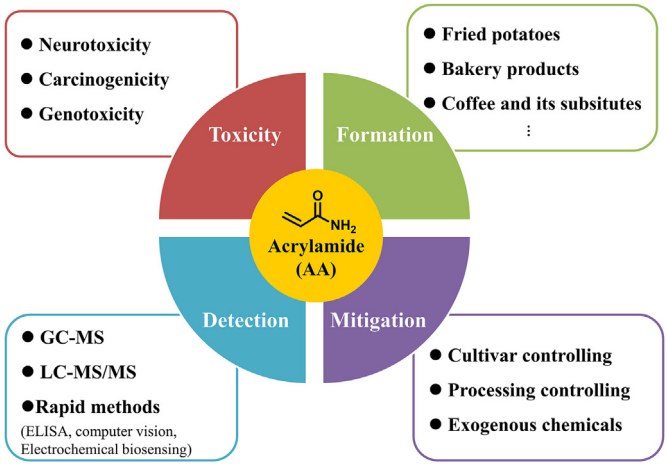

Fig. 1. Recent researches on AA in thermally processed foods.

1.2. Existence and formation of AA in foods

In April 2002, Swedish National Food Agency and researchers from Stockholm University reported a large amount of AA was found in some thermally processed foods. Since then, AA has received broad concerns from FAO/WHO, JECFA, FDA, other national food agencies, and other research institutions.

AA exists in different food products at different levels. A report on monitoring AA levels in food from 2007 to 2010 by the European Food Safety Authority (EFSA) showed that mean AA levels range from 31 μg kg^{-1} (processed cereal based foods for infants and young children) to 1350 μg kg^{-1} (coffee substitutes) (EFSA, 2011). The trend analysis did not show obvious changes in AA levels during the past four years. Fried potatoes (~272–570 μg kg^{-1}), bakery products (~75–1044 μg kg^{-1}), and coffee and its substitutes (~229–890 μg kg^{-1}) were still the top three categories of foods containing AA, which were also the major contributors to adult AA exposure. The levels for mean and high dietary exposure to AA were 1 and 4 μg kg^{-1} b.w. per day (JECFA, 2011), respectively, as represented at the 64th JECFA meeting (JECFA, 2005). Based on long-term monitoring, the indicative values of AA in 10 food categories were proposed in 2011 and updated in 2013, which may be a step to set regulatory limits in the next several years (EU, 2011, 2013).

AA has been found in numerous carbohydrate-rich foods after being fried, baked, or roasted at a temperature higher than 120 °C. The well-accepted mechanism of AA formation in foods is the asparagine-reducing sugars (usually fructose and glucose) pathway accompanied by the Millard reaction, wherein an intermediate Schiff base is first formed, followed by AA formation via the Strecker pathway or N-glycoside pathway (Claeys, De Vleeschouwer, & Hendrickx, 2005; Mottram, Wedzicha, & Dodson, 2002; Stadler et al., 2002). Another pathway that has been reported is the acrolein pathway after organic acid decarboxylation (Medeiros Vinci, Mestdagh, & De Meulenaer, 2012; Yaylayan & Stadler, 2005). The research indicated raw materials (cultivars, the contents of free amino acid and reducing sugars) and processing parameters (heating duration, heating temperature, and water activity) influence the level of AA in processed foods (De Vleeschouwer, Plancken, Van Loey, & Hendrickx, 2007, 2010; Halford et al., 2012; Muttucumaru et al., 2008; Wicklund et al., 2006).

1.3. Standard and conventional methods for the detection of AA in foods

Liquid Chromatography (LC) and high performance liquid chromatography (HPLC) coupled with mass spectrometry (MS) are