

Thermal Processing Contaminants in Foods

Iplikcioglu Cil G^{1*} and Sireli UT²

¹Ankara University, Faculty of Veterinary Medicine, Food Hygiene and Technology Department, Diskapi/Ankara, Turkey

²Department of Pharmacology and Toxicology, Ankara University, Faculty of Veterinary Medicine, Ankara, Turkey

***Corresponding Author:** Iplikcioglu Cil G, Faculty of Veterinary Medicine, Food Hygiene and Technology Department, Diskapi/Ankara, Turkey.

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Introduction

Thermal processing of food products has been used in the food industry for many years. This process is based on heating of foods for a certain length of time at a certain temperature. Heat treatments are traditionally applied various foods not in industry but also in homes. The main purposes of heat treatments are to improve the safety, sensory and nutritional qualities of foods. Besides these, heat is used to advance food preservation by eliminating some enzymatic activities. Various non-thermal methods have been developed in the last few decades, but thermal processing still regarded as the most valid and frequently used method in processed food product market.

The reactions occur during the heat treatment are of great importance for the production of aroma, taste and color. This kind of changes are desirable, because they produce the specific sensory and texture features of foods, such as bread, cereals, chocolate, coffee, nuts, malt and cooked meat. However, studies showed that heat treatment may be accompanied by a reduction in the nutritive value of foods and by the formation of toxic compounds. Losses of nutritive value may be the result of overheating, provoking protein degradation and other deteriorative reactions. One of the most important reaction for the formation of undesirable compounds is Maillard Reaction, generally known as non-enzymatic browning. The negative side of this reactions is the possible formation of potentially carcinogenic compounds such as heterocyclic amines. Heterocyclic amines have attracted a growing interest during the past two decades and there are many studies about them. But beside heterocyclic amines, scientific knowledge about some other processing contaminants in food has grown considerably in recent years. As a consequence of the recent discovery of the widespread occurrence of furan, advanced glycation end products, acrylamide and chloropropanols in various foods, several studies have been launched to better understand the formation, potential control, and health risks of this contaminants. Reduction of this kind of compounds in foods is challenging due to the large number of precursors in many foods and the requirement of heat process in food industry.

In this review, information about the presence, formation and potential risks to public health of some important processing contaminants will be provided.

Acrylamide

Acrylamide (2-propenamid, $\text{CH}_2=\text{CH}-\text{CONH}_2$) is a synthetic vinyl compound produced as an intermediate in the production and synthesis of polyacrylamides. Acrylamide has been widely used since the 1950s in industrial applications, such as in the treatment of wastewater, in textile, in paper and dye processing and in mining and mineral production. Also, it is known as a component of tobacco smoke.

The Swedish National Food Authority and the University of Stockholm was discovered in 2002, considerable levels of acrylamide forms when certain foods are prepared at temperatures usually above 120°C and low moisture. Acrylamide in foods is predominantly formed via the Maillard reaction. This reaction is also known as 'non-enzymatic browning' and responsible for the brown color and the formation of the characteristic flavor compounds formed during the processing of foods like, bread, meat, coffee and nuts. Also, acrylamide found in various baked or fried carbohydrate rich foods, including French fries, potato crisps and biscuits. Reducing sugars and asparagine is

the most important components for the formation of acrylamide. Although, acrylamide in foods is predominantly formed by the Maillard reaction, several other formation mechanisms have been reported. It was shown that acrylamide can be formed from acrolein and acrylic acid, especially in lipid rich foods when fats are heated at an improperly high temperature. The pyrolytic acrylamide formation was demonstrated from purified wheat gluten (Claus, *et al.* 2006, Stadler and Scholz 2004).

Epidemiological researches revealed that infants, toddlers and other children were the most exposed group to the acrylamide. Acrylamide is mostly metabolized to glycidamide in the liver via cytochrome P450, which is a reactive compound formed by the epoxidation of the double bond. The formation of glycidamide is considered to be the critical step for the adverse effects of acrylamide to the human health. The toxicological effects of acrylamide have been studied in animal models. At high levels of exposure, acrylamide is acting as a neurotoxin and may cause a range of symptoms such as numbness in the hands and feet. This high levels cannot be reached by food consumption so it can be disregarded. In contrast, in various *in vivo* and *in vitro* studies, carcinogenic and genotoxic effects of acrylamide was revealed. Exposure to acrylamide leads to DNA damage and carcinogenic action in both male and female mice and rats has been described in multiple tissues. The major tumors produced by this compound are adenomas, fibro adenomas and fibromas of the mammary gland, thyroid gland follicular cell adenomas or carcinomas.

The International Agency for Research on Cancer (IARC) has classified acrylamide as “probably carcinogenic to humans” (group 2A). In 2011, the National Toxicology Program (NTP) determined that Acrylamide is reasonably anticipated to be a human carcinogen (NTP, 2011) and the US-EPA characterized this compound ‘likely to be carcinogenic to humans’ (US-EPA, 2010).

Because acrylamide is present in a wide range of foods, it is hard to minimize the risk to exposure. The main factors that can be modified to lower its formation are cooking time and temperature. Frying, baking and roasting at lower temperatures and for shorter times reduce the amount of browning of the product and also reduce the amount of acrylamide produced.

Joint FAO/WHO Expert Committee on Food Additives determined the maximum daily intake of acrylamide as 0.004 mg/kg bw per day. In EU legislations, still there is no limits for the acrylamide.

Advanced Glycation End Products (AGEs)

Advanced glycation end-products (AGEs), are a heterogeneous group of highly reactive chemical compounds. They also referred as glycotoxins. AGEs can occur naturally in animal tissues but they become pathogenic when they reached high levels in the body. Although a number of different AGEs have been identified, N ϵ -karboksimetilizin’ in (CML), pentosidine and derivatives of methylglyoxal (MG) have been studied in detail and have been used as markers for AGEs.

In addition to AGEs that form as part of a normal metabolism, they also produced during the cooking and processing of certain foods. The main mechanism of formation is the non-enzymatic glycation of proteins, lipids, and nucleic acids. During high temperature cooking processes, like frying, roasting and grilling AGEs formation markedly accelerates. High levels of both CML and MG have been found in cooked red meats, poultry, fish and eggs. It was reported that high fatty products, such as butter, margarine, and mayonnaise may also contain significant amount of AGEs. Also, the presence of high levels of AGEs in mature and high-fat cheeses, suggests that they may be produced more slowly by other mechanisms. Tobacco smoke is another important exogenous source of AGEs.

Their potential affects to human health was ignored, because it was assumed that dietary AGEs (dAGEs) are poorly absorbed. However, experimental animal and human studies showed that dAGEs are absorbed and contribute significantly to the body’s AGE pool (Uribarri 2010). The deleterious effects of AGEs to human health are attributed to their highly oxidative, and inflammatory actions. AGEs effects within the body by two main mechanisms: structural deformation or cross-linking of body proteins, and interaction with AGE receptors. By this mechanisms AGEs, can involved in the pathology of a number of diseases, including diabetes and insulin resistance, cardiovascular and kidney diseases, hypertension, and Alzheimer’s disease.

Formation of AGEs in foods and the effects to human health is a complex subject involving several reactions and many end products. More studies needed for better understanding. The current recommendations for measures to reduce the level of dAGEs are to cook meat and fish at temperatures not more than 200°C and preferring indirect cooking methods, such as stewing, poaching and steaming, rather than grill, fry or barbecue.

Chloropropanols

Chloropropanols are chlorinated derivatives of glycerol, having one or two chlorine atoms on the glycerol molecule. The primary members of this group are 3-monochloropropane-1,2-diol (3-MCPD), 2-monochloro-propane-1,3-diol (2-MCPD), 3-chloropropane-1,2-diol (3-CPD), 1,3-dichloro-2-propanol (1,3-DCP) and 2,3-dichloro-1-propanol (2,3-DCP). The formation of these contaminants was first recognized in 1970's during the production of acid hydrolyzed vegetable protein (HVP). HVP is a common ingredient widely used in savoury foods including soups, sauces, bouillon cubes, snacks and soy sauce.

The formation of chloropropanols in food is not fully understood but the main mechanism is the reaction of hydrochloric acid with residual lipids. Chloropropanols and related esters can be found at relatively high concentrations in refined vegetable oils and seeds such as, palm oil, soy, rapeseed and peanut. The temperature used during the deodorization step of vegetable oils and fats has been shown to affect the extent of chloropropanol formation. Therefore, vegetable fat-containing products, including infant formula, are the main sources for these compounds. Recent studies revealed that elevated levels of chloropropanols, especially 3-MCPD, can occur in a wide range of foods such as cakes, bakery products, malt, meat and dairy products.

Chloropropanols were recognized as food contaminants after the discovery of chloropropanols and many chlorinated compounds are toxic. Among these compounds, 1,3-DCP has the most toxicological relevance. It was known that 1,3-DCP shows clear carcinogenic effects in rats. According to the studies, kidney and testis appeared to be the main target organs for 3-MCPD. Glycidol ester is another compound from this group, determined as genotoxic carcinogen and classified by IARC as group 2A, probably carcinogenic to human.

Main concern from food safety aspects is the effect of low doses over a long time. In 2002 European Union adopted a regulatory limit of 0.02 mg/kg for 3-MCPD, based on a 40% dry matter content, in soy sauce and HVP. Also, provisional maximal tolerable daily intake of 2 mg/kg bw has been allocated for 3-MCPD. To obtain a significant reduction of chloropropanols and their esters in foods is much more complex and needing some more researches.

Furan

Furan (C₄H₄O) is a highly volatile and lipophilic organic compound. It is used in industry as a solvent or intermediate in polymer synthesis and can be produced synthetically. Furan and its derivatives also occur in foods and they are connected with the taste and smell of some certain foods. It has long been known a component of cigarette smoke. Furan, should not be confused with the monocyclic compound like polychlorinated dibenzofurans which are environmental contaminants.

Presence of furan in heat treated-foods was reported during the 1960s. It was not expected to be a widespread food contaminant because of their volatile structure and difficult detection with analytical methods. However, scientific reports outlined the occurrence of furan in a broad variety of foods, such as coffee, canned or jarred foods, baked products and most notably baby foods. After classifying furan as "possibly carcinogenic to humans" (Group 2B) by the IARC, a great concern is given to the analysis of this compound. Still, there is limited information about the mechanism of furan formation in foods. Different studies indicate multiple reactions for occurring such as; thermal degradation of reducing sugars or certain amino acids, and thermal oxidation of ascorbic acid, poly-unsaturated fatty acids and carotenoids.

According to the animal experiments, furan is absorbed from the intestine and the lung, rapidly and extensively. It can pass through biological membranes and enter various organs by its low polarity. Furan is clearly induces tumors in rats and mice and the liver is the main target organ. Experiments showed a furan dose-dependent increase in hepatocellular adenomas and carcinomas. Also, high inci-

dence of cholangiocarcinomas even at the lowest furan dose tested (2 mg/kg b.w.). Although there have been no human studies, concern still exists, whether furan may also cause cancer in humans through long-term exposure to low levels of furan in foods.

Reduction of furan formation in food seems to be more challenging compared to other process contaminants, due to its connection with the large number of precursors in many foods. There is a need to more detailed exposure assessment data, and better toxicological information on which to base a comprehensive risk assessment.

Conclusion

Epidemiological studies showed that different disease conditions, such as cancer, endocrine disruption and diabetes can be coupled to the presence of chemical contaminants in food. However, the detailed formation mechanisms of heat-induced contaminants in foods are poorly understood. Advances in technology allowed optimization of thermal processing for minimum deterioration of food quality and harmful effects. But, the fact that thermally generated compounds occurring simultaneously and give rise to many reactive intermediates, making their interpretation and control difficult. More studies needed at international level to better understand the formation, potential control, and health risks of this contaminants.

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