Editorial

Guest Editor:
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Maillard Reaction and Health Aspects

From the roasting of coffee beans to the preparation of cooked meats, an understanding of the underlying mechanisms of thermally generated aromas in foods is necessary for the development of palatable, high-quality processed foods. The complex set of reactions known collectively as the Maillard reactions have been considered to be responsible for the generation of roasted, toasted and caramel-like aromas, as well as for the development of browned surfaces in foods. The Maillard reaction also has both nutritional and toxicological effects on processed food. Many of the antinutritional aspects of the Maillard reaction, such as effects on the availability of essential amino acids, effects on enzyme activity, as well as the effects on the absorption/utilization of metals, have been extensively studied. The Maillard reaction is responsible for the formation of potent mutagenic/carcinogenic heterocyclic aromatic amines (HAAs) in heated meat and fish. Asparagine-mediated Maillard reaction is known to lead to the formation of neurotoxic acrylamide. In this issue, Cheng *et al.* [1] review the chemistry and toxicity of HAAs. The potential mutagenicity of caramelized sucrose is discussed by Kitts *et al.* [2]. On the other hand, Hayase *et al.* [3] discuss that some melanoidin intermediates can effectively suppress peroxidation, scavenging free radicals and prevent oxidative cell injury.

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In vivo Maillard reaction and its negative effect on human health and diseases has been an active research area in the past 20 years. Diabetes is a heterogeneous disorder that involves resistance of glucose and lipid metabolism in peripheral tissues to the biological activity of insulin and the inadequate insulin secretion by pancreatic β cells, and it is generally accompanied with multiple complications. The Diabetes Control and Complications Trial has identified hyperglycemia as a risk factor for the development of diabetic complications. Increasing evidence identifies the formation of advanced glycation end Products (AGEs) as the major pathogenic link between hyperglycemia and diabetes-related complications. N^{ε}-(carboxymethyl)lysine (CML) was the first advanced AGE identified in tissue proteins. CML was first described as a product of the oxidative cleavage of the Amadori adduct of sugar to protein. Nonenzymatic glycation is a complex series of reactions between reducing sugars and amino groups of proteins, lipids and DNA. As the first step of AGEs formation, proteins in the tissues are modified by reducing sugars through the reaction between a free amino group of proteins and a carbonyl group of the sugars, leading to the formation of fructosamines via a Schiff base by Amadori rearrangement. Then, both Schiff base and Amadori product further undergo a series of reactions through dicarbonyl intermediates (*e.g.*, glyoxal and methylglyoxal), to form AGEs. Glyoxal and methylglyoxal, the two major α-dicarbonyl compounds found in human, are extremely reactive and readily modify lysine, arginine, and cysteine residues on proteins. More and more evidences indicate the increase in reactive carbonyl intermediates is the consequence of hyperglycemia in diabetes. "Carbonyl stress" leads to increased modification of proteins and lipids, followed by oxidant stress and tissue damage. Several studies have shown that higher levels of glyoxal and methylglyoxal were observed in diabetic patients' plasma than those in healthy people's plasma. Several dicarbonyl-derived products in proteins from diabetic individuals have been identified. Decreasing the levels of glyoxal and methylglyoxal will be a useful approach to prevent the formation of AGEs.

In this issue, Nemet *et al.* [4] review the chemistry and analytical methods for the methylglyoxal formed in food and biological system. Nguyen [5] discusses the relationship of dietary AGEs and endogenous AGEs. Lo *et al.* [6] report that green tea and black tea polyphenols are able to trap methylglyoxal in simulated physiological condition. The structure of adducts from the interaction of epigallocatechin have been identified. Finally, Huang *et al.* [7] showed that flavonoid antioxidants such as quercetin have protective effects against the oxidative damage and inflammation mediated by AGEs in human monocytes.

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