Chapter 16
Antioxidants as Functional Foods in Metabolic Syndrome

Abishek B. Santhakumar
Charles Sturt University, Australia

Indu Singh
Griffith University, Australia

ABSTRACT

In the recent years, there has been a great deal of attention in investigating the disease preventive properties of functional foods. Particularly, impact of the antioxidant property of functional foods in reducing the risk or progression of chronic diseases has gained considerable interest amongst researchers and practitioners. Free radicals such as reactive oxygen species are generated in the body by exposure to a number of physiochemical or pathological mechanisms. It is imperative to preserve a balance between the levels of free radicals and antioxidants for routine physiological function, a disparity of which would accelerate oxidative stress. Increased oxidative stress and associated consequences in metabolic disorders such as obesity, cardiovascular diseases and diabetes has warranted the need for exogenous antioxidant concentrates derived from natural foods to alleviate the adverse effects. This chapter provides an overview on the efficacy of functional foods in reducing free radical-mediated damage in metabolic syndrome.

INTRODUCTION

Food not only provides the essential nutrients needed for life but also other bioactive components necessary for disease prevention and maintenance of good health. Elaborate research on the role of physiologically active food components derived from plants (phytochemicals) and animal (zoochemicals) have led to the development of a new class of foods known as functional foods. The term functional foods was first introduced in Japan during the mid-1980s and were known as Foods for Specified Health Use (FOSHU), which comprised foods that would enhance human health, in addition to being nutritious. For this reason, functional foods are defined as a group of foods or food ingredients that have a beneficial effect on human health and/or help in reducing the risk of chronic disorders beyond the widely accepted nutritional functions.

DOI: 10.4018/978-1-5225-7039-4.ch016

Metabolic syndrome is a collection of at least three of five precursors such as high fasting glucose, elevated triacylglycerol, high blood pressure, low high density lipoprotein (HDL) or abdominal obesity (Ford, Giles, & Dietz, 2002). Metabolic syndrome is associated with the risk of developing chronic conditions such as diabetes, cardiovascular diseases and obesity which are major health concerns in developed as well as developing nations (Alberti, Zimmet, Shaw, & Group, 2005). Oxidative stress plays one of the central roles in the pathogenesis of such conditions. Extensive research during the past decade has demonstrated that free radicals, particularly, reactive oxygen species (ROS) and reactive nitrogen species (RNS) are an important contributor to the damage caused by oxidative stress (Apel & Hirt, 2004). There is increased oxidative stress due to conditions such as diabetes, which impairs muscle glucose uptake, by damaging the blood vessel walls by oxidising lipids and in turn activating platelets resulting in thrombosis and other cardiac complications (Maritim, Sanders & Watkins, 2003). We will discuss this mechanism in detail later in the chapter. The results of many epidemiological studies have demonstrated a protective role of antioxidants through a diet rich in fruits and vegetables against the development and progression of metabolic syndromes. We will now deliberate the role of antioxidants as functional foods in reducing oxidative stress thereby reducing the risk or progression of metabolic dysfunction.

FREE RADICALS AND OXIDATIVE STRESS

A chemical reaction in which the electrons get transferred from a substance to an oxidizing agent is known as oxidation, which in turn produces free radicals (any independently existing chemical species having one or more unpaired electrons), which produced in excess, ultimately results in oxidative stress. Oxidation reactions are an essential part of normal metabolism as oxygen is the ultimate electron acceptor in the electron flow system that produces adenosine triphosphate – ATP, source of energy in body. Problems may arise when electron flow and energy production become uncoupled followed by the increased production of oxygen free radicals like ROS. It is a known fact that molecular oxygen can be reduced to water. The intermediate steps involved in this reduction of molecular oxygen are the formation of free radicals such as superoxide anions, hydrogen peroxide and hydroxyl radicals. Once these radicals are formed, they can react with each other or with another molecule depending on its concentration. The increase in such pro-oxidants is directly involved in pathogenesis or as a result of an aerobic metabolic process. The body continually battles to maintain levels of pro-oxidants and antioxidants to reduce the occurrence of chronic diseases. Nevertheless, reduced concentration of endogenous antioxidants to neutralise the released free radicals results in severe oxidative stress consequently leading to cell damage and death.

All forms of life maintain a reducing environment within their cells. Enzymes that maintain the reduced state through a constant input of metabolic energy preserve the cellular redox environment. Disturbances in this normal redox state can cause toxic effects through the production of peroxidase and free radicals that damage components of the cell such as lipids and DNA. ROS are continuously produced within the cell as a result of mitochondrial electron transfer processes or as byproducts of the activity of enzymes including xanthine oxidase (catalyze the oxidation of xanthine to uric acid), lipoxygenases (catalyze polyunsaturated fatty acids to lipoxidase) and cyclooxygenases (involved in platelet activation and inflammation) (Szocs, 2004). They can also be generated as a consequence of the intracellular metabolism of foreign compounds, toxins, drugs or because of exposure to environmental factors such as excessive iron salts or ultraviolet radiation (Ichihashi et al., 2003). External sources of oxidative stress include ageing, smoking, alcohol consumption, and environmental pollution, all of which increase