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The Role of Meal Consumption on Postprandial Oxidative Stress and Inflammation

Tuğçe Bulmuş Tüccar[1], Gamze Akbulut[2]

[1] Yüksek İhtisas University, Department of Nutrition and Dietetics, Faculty of Health Sciences, 06520, Ankara/Turkey

[2]Gazi University, Department of Nutrition and Dietetics, Faculty of Health Sciences, 06560, Ankara/Turkey

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Abstract

Noncommunicable diseases (cardiovascular diseases, cancer, chronic respiratory diseases and diabetes etc.), also known as chronic diseases, are the leading causes of global deaths. Oxidative stress and inflammation are important underlying factors in the development of these diseases and are directly related to nutrition. The oxidative and inflammatory response due to nutrition may occur in the long term, as well as immediately following meal consumption. It is known that each meal produces a short-term and a certain level of oxidative and inflammatory responses in the postprandial state depending on the energy content and pattern. Nutritional factors that cause postprandial oxidative stress and inflammatory response are energy, carbohydrate and fat contents of the meal. It is known that frequent recurrence of acute responses that start in the postprandial state and its chronic exposure may play an important role in the development of chronic diseases such as cancer, diabetes and cardiovascular diseases in the long term. Considering that nowadays most of the days are spent in the postprandial state, the importance of the energy content and pattern of the meals has increased. In this review, the effects of meal consumption and nutrition on postprandial oxidative stress and inflammation metabolism were investigated.

1. Introduction

Noncommunicable diseases (NCDs) are the major global disease burden and are the leading cause of death in the world. (WHO, 2018). Oxidative stress and inflammation are important underlying factors in the development of cancer, diabetes and cardiovascular diseases, which are the main NCDs (Chatterjee, 2016). Nutrition is known to be a factor affecting oxidative stress and inflammation (Ursini et al., 1998). Nutritional oxidative stress is defined as the deterioration of the oxidative balance between the increase in reactive oxygen species (free radicals, ROS) such as hydroxyl radical, superoxide radical and hydrogen peroxide, which are formed during cellular metabolism, and the antioxidant defense system (Betteridge, 2000; Liguori et al., 2018; Moylan & Reid, 2007). Overproduction of ROS can oxidize biomolecules or trigger signal cascades, causing or increasing an inflammatory response (Chatterjee, 2016). Therefore, inflammation and oxidative stress are two processes that can be responsible for each other's formation in a vicious circle (Reuter, Gupta, Chaturvedi, & Aggarwal, 2010).

Oxidative and inflammatory response in the organism due to nutrition may occur in the long term or just after meal consumption (Kanner et al., 2017). Proinflammatory cytokines and oxidative stress have been shown to increase together postprandially (Evans, Goldfine, Maddux, & Grodsky, 2002; Van Oostrom, Sijmonsma, Rabelink, Van Asbeck, & Cabezas, 2003; van Oostrom, Sijmonsma, Verseyden, et al., 2003; Wellen & Hotamisligil, 2005). Studies with healthy individuals show that a single meal with a high fat and / or carbohydrate content can cause sudden increases in oxidative stress and inflammation, which can have adverse effects on postprandial endothelial dysfunction, vasoconstriction, and systolic blood pressure (Jakulj et al., 2007; O'Keefe, Gheewala, & O'Keefe, 2008). It is known that the frequent repetition of these acute responses that start in the postprandial phase will lead to chronic exposure and may play an important role in the development of NCDs such as cancer, diabetes and cardiovascular diseases in the long term (Khansari, Shakiba, & Mahmoudi, 2009).

Nowadays, especially in modern societies, a significant part of the day is spent in the postprandial phase (Devaraj, Wang-Polagruto, Polagruto, Keen, & Jialal, 2008; O'Keefe & Bell, 2007). For this reason, the effects of different dietary patterns on postprandial oxidative stress and inflammation can potentially be associated with the risk of chronic disease, so research on this subject is increasing and gaining importance.

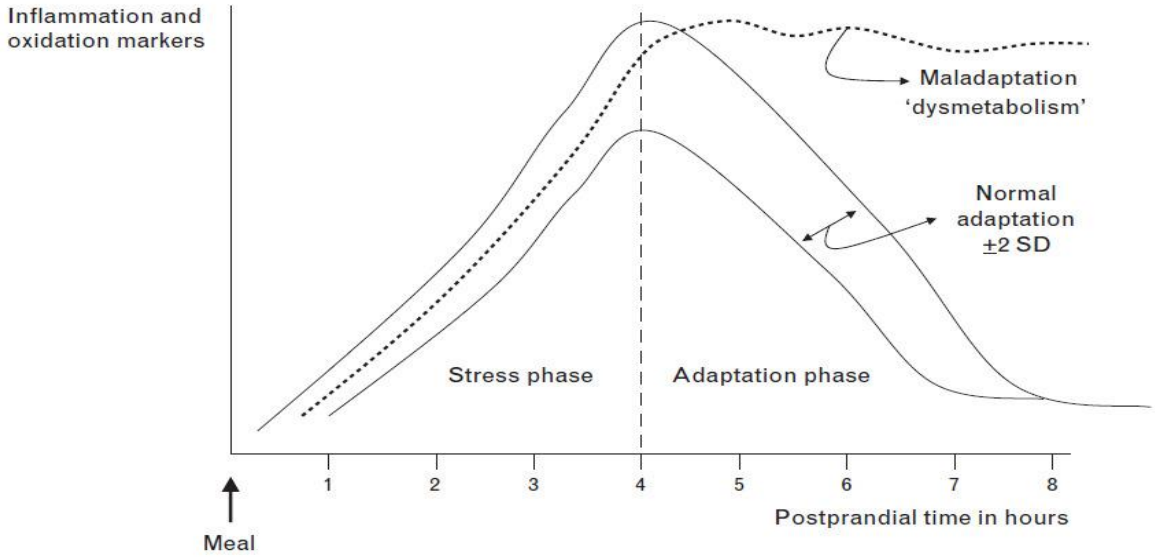
2. Postprandial Metabolism

Nutrition can be anti-inflammatory or pro-inflammatory depending on the metabolic response that it causes (Panickar & Jewell, 2015; Sears, 2015). It is known that each meal produces a short-term and certain level of oxidative and inflammatory response postprandially. This response consists of two phases. The first stress phase, begins within minutes with food consumption and typically reaches a peak level between 2 and 4 hours. The second phase is the adaptation phase in which the immune / metabolic homeostatic balance is restored under normal conditions. As long as these phases are balanced,

***Corresponding author:** Tuğçe Bulmuş Tüccar,
e-mail address: gtugcebulmus@gmail.com

there is a state of homeostasis. However, as a result of the stress phase being too strong or the adaptation phase being too weak, this response may last for hours and 'dysmetabolism' may occur due to failure to balance (Figure 1)(Bloomer, Kabir, Marshall, Canale, & Farney, 2010; Herieka & Erridge, 2014; Margioris, 2009; O'Keefe & Bell, 2007; Sies, Stahl, & Sevanian, 2005).

Figure 1. Adaptation to meal consumption-induced stress and dysmetabolism (Margioris, 2009)



3. Metabolic Consequences of Oxidative Stress and Inflammation

When postprandial changes and dysmetabolism are repeated many times each day, cells cannot cope with this chronic condition. This stress causes changes in cells that predispose to chronic and degenerative diseases (Bhatti, Bhatti, & Reddy, 2017)

Oxidative stress and inflammation are particularly important in the pathogenesis of endothelial dysfunction contributing to atherosclerosis (de Vries et al., 2014). Endothelial dysfunction is accepted as an early marker of atherosclerosis (Davignon & Ganz, 2004). The idea that atherogenesis is a postprandial phenomenon was first suggested by Zilversmit. According to this view, the accumulation of lipoproteins with high triglyceride content in the postprandial state and this prolonged hyperlipidemic state contributes to vascular damage and the development of atherosclerosis (Zilversmit, 1979).

Postprandial hyperglycemia due to the high carbohydrate content of the meal causes irreversible glucosylation of proteins (AGEs). These AGEs bind to receptors on endothelial smooth muscle cells and fibroblasts, resulting in increased permeability of the endothelium, proliferation of vascular smooth muscle cells, extracellular matrix formation and hypercoagulation (Brownlee, 2001).

In the face of excessive increase in free radical levels in the organism, inadequate antioxidant defense mechanisms (antioxidants; tocopherols, ascorbic acid, glutathione or enzymes involved in oxygen radical scavenging; catalase, peroxidase and superoxide dismutase) cause cellular organelles and enzymes to be damaged. The proliferation of

***Corresponding author:** Tuğçe Bulmuş Tüccar,
e-mail address: gtugcebulmus@gmail.com

damaged (mutated) cells can lead to cytotoxicity, genotoxicity and carcinogenesis (Gagné, 2014). This can also lead to increased lipid peroxidation and the development of insulin resistance. These consequences of oxidative stress can promote the development of diabetes complications (Hadziabdic, Bozikov, Pavic, & Romic, 2012). Oxidative stress is responsible for the pathogenesis of many diseases such as cancer, diabetes, cardiovascular and neurological diseases, atherosclerosis and inflammatory disorders (Liguori et al., 2018).

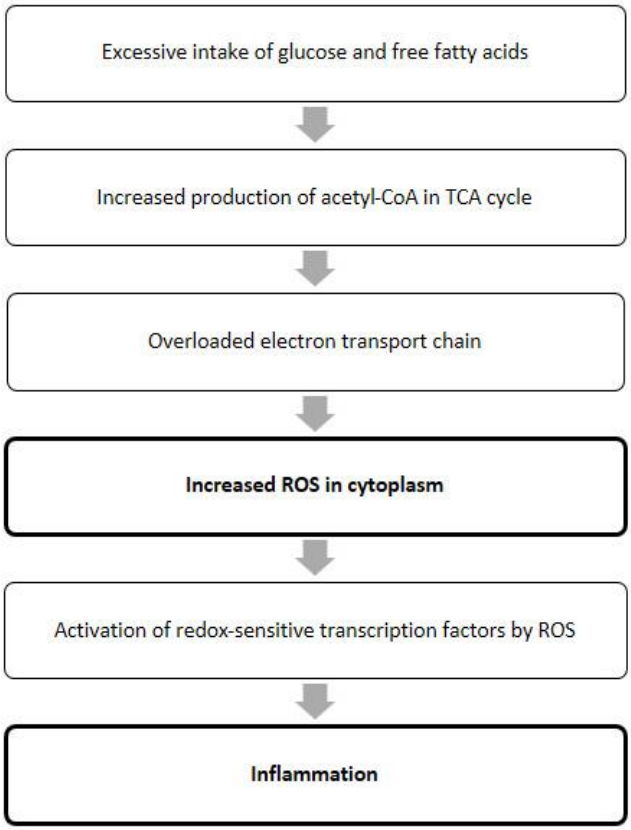
4. Effect of Nutrition

Main factors related to nutrition affecting the development of dysmetabolism in the postprandial phase; carbohydrate and fat content, fatty acid composition and energy content of the meal.

4.1. Carbohydrate and fat content

Excessive intake of glucose and fatty acids leads to an increase in the production of acetyl coenzyme A (acetyl CoA), an important enzyme for cellular metabolism. Increasing acetyl CoA level results in an increase in the formation of reduced nicotinamide adenine dinucleotide (NADH) in the tricarboxylic acid (TCA) cycle. As a result, the mitochondria are stimulated to produce excess superoxide in the electron transport chain, and the superoxide level increases in the mitochondria (Munoz & Costa, 2013). Superoxide is a relatively unstable intermediate and is substantially converted into hydrogen peroxide. The newly formed hydrogen peroxide yields a highly reactive hydroxyl radical that can oxidize mitochondrial proteins, deoxyribonucleic acid DNA, and lipids and enhance the effects of oxidative stress initiated by superoxide. As a result, excessive amounts of ROS is produced. This can activate redox-sensitive transcription factors and inflammatory cascades are triggered. This process is primarily mediated by nuclear factor-κB (NF-κB), a proinflammatory transcription factor (Figure 2) (O'Keefe & Bell, 2007). Postprandial inflammation in the short term is characterized by an increase in the levels of circulating NF-κB-mediated inflammatory cytokines, tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) (Munoz & Costa, 2013). The first marker of cellular inflammation is high sensitivity (sensitive) C reactive protein (hs-CRP). This protein is synthesized in the liver in response to high levels of IL-6 in the blood (Ndumele, Pradhan, & Ridker, 2006).

Figure 2. Postprandial metabolism (Munoz & Costa, 2013)



***Corresponding author:** Tuğçe Bulmuş Tüccar,
e-mail address: gtugcebulmus@gmail.com

4.2. Fatty acid composition

Fatty acids are thought to be the most effective triggers of the postprandial inflammatory response. The most important modulators of postprandial response are considered to be n-6, n-3 polyunsaturated fatty acids and saturated fatty acids. In general, n-3 fatty acids reduce the postprandial inflammatory response, while n-6 fatty acids promote. The dietary source of n-6 fatty acids is linoleic acid (LA). Arachidonic acid (AA) synthesized from LA is the precursor of proinflammatory eicosanoids that stimulate inflammation (Margioris, 2009).

It is known that as the LA content and thus the AA amount of the diet increases, the inflammatory load also increases. Delta-6 and delta-5 desaturase enzymes that control AA synthesis from LA are rate limiting enzymes. Insulin hormone plays a role in the activation of these enzymes (Brenner, 1981). Postprandial hyperglycemia, which may be occured by the content of the meal, increases insulin secretion. Potential activation of these enzymes increases as insulin secretion increases. Therefore, when high insulin level is combined with diet and high LA intake, the conversion of LA to AA increases significantly. Proinflammatory eicosanoid formation increases significantly with increasing AA levels in cells (Sears, 2015).

The high saturated fatty acid content of the diet also increases the activation of NF-κB, a transcription factor that plays an important role in the formation of proinflammatory biomarkers (TNF-α, IL-1, IL-6, etc.) (Fritsche, 2015; Huang et al., 2012).

4.3. Energy content of the meal

Consumption of a high-energy meal may cause a strong postprandial immune / inflammatory response and the emergence of cellular oxidative stress. This response may only be seen due to the high energy content, even if the meal pattern is balanced (Alderton, 2018; Zhang et al., 2008). Regardless of the macronutrient composition of the meal, the amount of food consumed also affects the blood glucose level. Even a well-balanced meal, if the portion is large, can cause a significant increase in blood glucose (O'Keefe et al., 2008) and an acute postprandial hyperglycemic response (Franz, 2001). It is known that postprandial hyperglycemia causes oxidative response and can induce inflammatory cascades through oxidative metabolism. Superoxide production and ROS level increase due to intracellular hyperglycemia. Increased oxidative stress causes the production of reactive intracellular dicarbonyls and the emergence of inflammatory cytokines, which react with amino acids to form advanced glycation end products (AGEs) in macrophages and procoagulator (Munoz & Costa, 2013). Acute hyperglycemia results in increased levels of circulating inflammatory cytokines, including TNF-α, IL-6 and (interleukin-18) IL-18 (Galland, 2010).

5. Evaluation of Diet Patterns - Mediterranean Diet and Western-Style Diet

Considering the effects of energy and nutrient components of the meals consumed during the day on postprandial metabolism and the long-term results, the importance of the pattern of the diet increases. In this context, high content of monounsaturated and n-3 polyunsaturated fatty acids,

***Corresponding author:** Tuğçe Bulmuş Tüccar,
e-mail address: gtugcebulmus@gmail.com

high content of saturated fat and n-6 polyunsaturated fatty acids; The Mediterranean diet, which is rich in antioxidants and has a low glycemic index, is considered a diet pattern with anti-inflammatory and antioxidative effects. (Casas, Sacanella, & Estruch, 2014; Davis, Bryan, Hodgson, & Murphy, 2015). Mediterranean diet is a rich in plant-based foods (cereals, fruits, vegetables, legumes, nuts, oilseeds); the main oil source is olive oil; fish and seafood consumption is high; eggs, poultry and dairy products consumption is moderate; red meat and processed meat consumption is low (Bach-Faig et al., 2011; Pekcan, 2019). Adherence to the Mediterranean diet has been shown to reduce the level of inflammatory factors (C-reactive protein, IL-6) in serum even in a very short period of time (Hadziabdic et al., 2012; Roberts et al., 2006).

The protective effect of the Mediterranean diet against chronic diseases such as cardiovascular diseases, cancer, and diabetes is evaluated through its effect on oxidative stress and inflammation, which are the common mechanisms in the development of these diseases. Therefore, interest in the antioxidative and anti-inflammatory effects of the Mediterranean diet has been increasing recently (Ahluwalia, Andreeva, Kesse-Guyot, & Hercberg, 2013; Hadziabdic et al., 2012).

The Mediterranean diet is included in the nutrition policies of many countries, including our country, as a health-protective diet recommendation (TÜBER, 2016). However, globalization also affects nutritional habits and an increasing "westernization" is observed in the nutritional habits of societies (Harrell, Ussery, Greene-Cramer, Ranjit, & Sharma, 2015; Pingali, 2007; Uusitalo et al., 2005). The term "Western diet" refers to the diet pattern with high fat, carbohydrate and energy content, which emerged as a result of the impact of industrial and agricultural revolutions on nutritional habits. With the industrial period, the glycemic load, fatty acid composition, macronutrient composition and micronutrient content of diets have changed significantly (Cordain et al., 2005). Accordingly, the dietary pattern in western societies has an oxidative and inflammatory effect (Kopp, 2019).

6. Conclusion

Inflammation and oxidative stress play an important role in the development and progression of chronic and degenerative diseases and are directly related to nutrition. Considering the oxidative and inflammatory response to be created in metabolism by spending a significant part of the day in the postprandial state, the importance of the energy content and pattern of meals increases even more. In this context, it should be kept in mind that compliance with the Mediterranean diet containing high levels of antioxidant components, unsaturated fatty acids, fiber and low energy levels is important in preventing chronic diseases.

Conflicts of interest

The authors declare they have no conflict of interest.

***Corresponding author:** Tuğçe Bulmuş Tüccar,
e-mail address: gtugcebulmus@gmail.com

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***Corresponding author:** Tuğçe Bulmuş Tüccar, e-mail address: gtugcebulmus@gmail.com

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***Corresponding author:** Tuğçe Bulmuş Tüccar,
e-mail address: gtugcebulmus@gmail.com