Effects of Nutrition Style on Metabolism

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Abstract
Meal frequency regulation and caloric restriction are gaining importance in modern dietary recommendations. The effects of caloric restriction on metabolism are approximately known. However, the combined effects of meal frequency and caloric restriction have not been adequately investigated. New research is needed to determine the ideal nutritional model that can be effective in preventing diabetes, cardiovascular disease, obesity, and many cancers. The current study emphasizes that there are not enough studies in the literature on the effects of meal frequency and calorie restriction on metabolism.

Keywords
Nutrition; Meal Frequency; Caloric Restriction; Metabolism; Insulin Resistance

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Introduction

Nutrition is defined as organisms taking nutritional elements from outside and using them effectively in order to facilitate all functions of living, especially growth and development [1]. The effects of nutrition on metabolism have been examined by various researchers for years. The increasing incidence of obesity and prominence of the relationship between nutrition and various chronic diseases necessitate detailed investigation of the relationship between nutrition and metabolism. This study aims to draw attention to the effects of meal frequency and caloric restriction on metabolism. In our literature review, we did not encounter any studies that examined the effects on the antioxidant system of types of feeding in conjunction with caloric restriction. Additionally, most of the experimental and clinical studies focus on the effects on the antioxidant system of the meal contents, rather than the meal frequency or the amount of calories [2-6]. Studies investigating the effects of meal frequency on metabolism are rather outdated ones that examine the effects on metabolic enzymes and hormones without investigating the antioxidant system [7-11]. The general relationship between meal frequency and metabolism has by now been established, but the results of the current study will help to reveal the effect of meal frequency on the antioxidant system. It is known that excessive energy intake in humans increases the risk of diabetes, cancer, and cardiovascular diseases. On the other hand, the effects of increased meal frequency on human health or lifespan are not clear [12]. Increased meal frequency in humans has been linked to hepatic steatosis, increase in triglycerides, and obesity [13]. Caloric restriction in animals is the intake of calories reduced by 20-40% as opposed to feeding ad libitum [14]. Caloric restriction in humans is recommended in similar ratios. When caloric intake is reduced by 20-40%, vitamin and mineral supplementation should increase as intake decreases in order to prevent malnutrition [15]. Caloric restriction, due to its effects on the blood lipid profile, reduces blood pressure and heart beats per minute [16, 17]. Caloric restriction also prevents development of insulin resistance by reducing fasting insulin and glucose levels [18].

From an evolutionary perspective, in the periods where sources of nutrition were not continuously available, bodies needed to store excess amounts of energy in order to sustain life. Fat cells located in fat tissue reservoirs in the human body have adapted to store excess energy as triglycerides and to release these as free fatty acids to be used as energy in other parts of the body when needed. The fat storage and usage system governed by neural and endocrinal systems helps humans to survive periods of prolonged hunger. However, the combination of excessive feeding and sedentary life style, along with genetic factors, increases fat reservoirs excessively and creates negative health conditions. Obesity, the condition where fat tissue mass is in excess, is the primary cause of other diseases [19].

Caloric restriction may be defined as the reduction of caloric intake while sustaining essential nutritional needs, and it is different from constant hunger. In other words, caloric restriction is the reduction of total intake of food without causing malnutrition or sacrificing specific essential nutrients [20, 21]. Another study has shown that caloric restriction has anti-aging and lifespan-increasing effects and has also provided information on possible neuroprotective effects [22]. Oxidative stress, mitochondrial damage, inflammation, and changes in the structure of proteins' DNA are important factors inducing neurodegeneration. It is thought that reduction of oxidative stress and stimulation of neutrophil production lie behind the mechanism related to reduction of neurodegenerative changes by caloric restriction [23, 26]. Another study also found that caloric restriction increased insulin sensitivity and protection against insulin resistance; decreased triglyceride, total cholesterol, and LDL cholesterol levels; increased HDL cholesterol levels; and decreased the incidence of cerebrovascular diseases [27].

All these studies show that how much you eat and how often you eat is as important as what you eat. In other words, nutrition style is important for regulation of metabolism. These studies have yielded promising findings about the effect of meal frequency on the antioxidant system. The study results have shown that, like caloric restriction, reducing meal frequency probably strengthens the antioxidant system and reduces oxidative stress. Therefore, eating less and eating less frequently represent an alternative protective treatment in the prevention of the hundreds of diseases known to be related to oxidative stress [28-30]. In our previous studies, we have found beneficial effects of reduced meal frequency and caloric restriction on insulin resistance, weight gain, the antioxidant system, diabetes, obesity, and metabolism [31-33].

Conclusion

The amount and frequency of food intake are as important as the content. Future studies of longer duration; life-long observational studies regarding the effects on lifespan; incorporating the factor of sex; and establishing study groups combining nutrition and exercise interventions may lead to new findings that are likely to confirm past results. Reduced meal frequency and caloric restriction have positive effects on metabolic efficiency and function–specifically, on key enzymes and basic regulatory hormones in carbohydrate, lipid, and protein synthesis and degradation pathways. Thus, it may be possible to avert insulin resistance, diabetes, obesity, and many other related diseases by eating less frequently and in smaller amounts.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

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Conflict of interest

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References

1. Gürdöl F. Bilimin Mum Işığında Yemek; Beslenmenin Biyokimyası. İstanbul: Nobel Tıp Kitabevleri, 2014, p: 1-5.
2. Steffens AB. Blood glucose and FFA levels in relation to the meal pattern in the normal rat and the ventromedial hypothalamic lesioned rat. Physiology & Behavior 1969;4:2:215-6.
3. Lima FB, Hell NS, Timo-Iaria C. Carbohydrate metabolism and food intake in food-restricted rats. Relationship between the metabolic events during the meal and the degree of food intake. Physiology & Behavior 1985;35:5:695-700.
4. Glendinning JI, Smith JC. Consistency of meal patterns in laboratory rats. Physiology & Behavior 1994;56:1:7-16.
5. Surina-Baumgartner DM, Arnold M, Moses A, Langhans W. Metabolic effects of a fat- and carbohydrate-rich meal in rats. Physiology & Behavior 1996;59:4–5:973-81.
6. Melhorn SJ, Krause EG, Scott KA, Mooney MR, Johnson JD, Woods SC, et al. Acute exposure to a high-fat diet alters meal patterns and body composition. Physiology & Behavior 2010;99:1:33-9.
7. Romsos DR, Leveille GA. Effect of meal frequency and diet composition on glucose tolerance in the rat. J Nutr 1974;104:11:1503-12.
8. Wolever TM. Metabolic effects of continuous feding. Metabolism 1990;39:9:947-51.
9. Stote KS, Baer DJ, Spears K, Paul DR, Harris GK, Rumpler WV, et al. A controlled trial of reduced meal frequency without caloric restriction in healthy, normal-weight, middle-aged adults. Am J ClinNutr 2007;85:4:981-8.
10. Muiruri KL, Leveille GA. Metabolic adaptations in meal fed rats: effects of increased meal frequency or ad libitum feedingin rats previously adapted to a single daily meal. J Nutr 1970;100:4:450-60.
11. Ohkawara K, Cornier MA, Kohrt WM, Melanson EL. Effects of increased meal frequency on fat oxidation and perceived hunger. Obesity (Silver Spring) 2013;212:336-43. doi: 10.1002/oby.20032.
12. Mattson MP. Energy intake, meal frequency, and health: a neurobiological perspective.

Annu Rev Nutr 2005;25:237-60.
13. Koopman KE, Caan MWA, Nederveen AJ, Pels A, Ackermans MT, Fliers E, et al. Hypercaloric Diets With Increased Meal Frequency, but Not Meal Size, Increase Intrahepatic Triglycerides: A Randomized Controlled Trial. Hepatology 2014;60:2:545-53.
14. Hursting SD, Lavigne JA, Berrigan D, Perkins SN, Barret JC. Calorie restriction, aging and cancer prevention; mechanisms of action and applicability to humans. Annu Rev Med 2003;54:131-52.
15. Canto C, Auwerx J. Caloric restriction, SIRT1 and longevity. Trends Endocrinol Metab 2009;20:7:325-31. doi: 10.1016/j.tem.2009.03.008.
16. Trepanowski JF, Canale RE, Marshall KE, Mohammad MK, Bloomer RJ. Impact of caloric and dietary restriction regimens on markers of health and longevity in humans and animals: a summary of avaliable findings. Nutr J 2011;10:107. doi: 10.1186/1475-2891-10-107.
17. Lee CK, Allison DB, Brand J, Weindruch R, Prolla TA. Transcriptional profiles associated with aging and middle age-onset caloric restriction in Mouse hearts. Proc Natl Acad Sci U S A 2002;99:23:14988-93.
18. Wang Z, Masternak MM, Al-Regaiey KA, Bartke A. Adipocytokines and the regulation of lipid metabolism in growth hormone transgenic and calorie-restricted mice. Endocrinology 2007;148:6: 2845-53.
19. Jameson JL. Harrison Endokrinoloji, 16. Baskı, İstanbul: Nobel Tıp Kitabevi, 2009, p:268-271.
20. Mattson MP, Duan W, Wan R, Guo Z. Prophylactic activation of neuroprotective stress response pathways by dietary and behavioral manipulations. NeuroRx 2004;1:111-6.
21. Akman C, Zhao Q, Liu X, Holmes GL. Effect of food deprivation during early development on cognition and neurogenesis in the rat. Epilepsy Behav 2004;5:4:446-54.
22. Mattson MP, Duan W, Guo Z. Meal size and frequency affect neuronal ğlasticity and vulnerability to disease: cellular and molecular mechanisms. J Neurochem 2003;84:417-31.
23. Prolla TA, Mattson MP. Molecular mechanisms of brain aging and neurodegenerative disorders: lessons from dietary restriction, Trends Neurosci 2001;11(Suppl.): 21–31.
24. Mattson MP, Chan SL, Duan W. Modification of brain aging and neurodegenerative disorders by genes, diet and behavior. Physiol. Rev 2002;82:637–672.
25. Bruce-Keller AJ, Umberger G, McFall R, Mattson MP. Food restriction reduces brain damage and improve behavioral outcome following excitotoxic and metabolic insults. Ann. Neurol 1999;45:8–15.
26. Contestabile A, Ciani E, Sparapani M, Guarnieri T, Dell'Erba G, Bologna F, et al. Activation of the ornithine decarboxylase – polyamine system and induction of c-fos and p53 expression in rela¬tion to excitotoxic neuronal apoptosis in normal and microencephalic rats. Exp. Brain Res 1998;120:519–26.
27. Karason K, Wikstrand J, Sjostrom L, Wendelhag I. Weight loss and progression of early atherosclerosis in the carotid artery: a four- year controlled study of obese subjects, Int J Obes Relat Metab Disord 1999;9:948 –956.
28. Halliwell B, Gutteridge JMC. Free radicals in biology and medicine. Third ed. Oxford: Oxford Science Publications, 2000.
29. Erel O. A novel automated direct measurement method for total antioxidant capacity using a new generation, more stable ABTS radical cation. Clin Biochem 2004; 37: 4: 277-85.
30. Erel O. A new automated colorimetric method for measuring total oxidant status. Clin Biochem 2005; 38: 12: 1103-11.
31. Savas HB, Gultekin F. The effects of meal frequency and calorie restriction on oxidant-antioxidant systems in rats. Journal of Cellular Neuroscience and Oxidative Stress. Special Issue: 2016; 8:1:495.
32. Savas HB, Gultekin F. Effects of Meal Frequency and Calorie Restriction on Metabolism in Rats. Turk J Biochem Special Issue 2016; 41:4.
33. Savaş HB, Gültekin F. Farklı Öğün Sıklığının, Sıçanlarda Metabolizma ve Antioksidan Sistem ile Nörodavranış Üzerine Etkileri. SDÜ Sağlık Bilimleri Dergisi 2016, 7.3: 68.