Letter

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Regulating Hypothalamus Gene Expression in Food Intake: Dietary Composition or Calorie Density? (*Diabetes Metab J* 2017;41:121-7)

Bo Kyung Koo

Department of Internal Medicine, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul National University College of Medicine, Seoul, Korea

Jang et al. [1] reported that high saturated fatty acid (SFA) or n-3 polyunsaturated fat acid (PUFA) increased proopiomelanocortin (POMC) mRNA expression significantly compared with baseline. The expression of POMC was more prominently increased in SFA compared with PUFA. However, compared with carbohydrate (CHO)-rich diet, even SFA-rich diet lead to a significantly lower level of POMC mRNA expression. In contrast, in the case of neuropeptide Y (NPY), the mRNA expression was significantly lower in CHO-rich diet compared with SFA or PUFA-rich diet.

Although Jang et al. [1] mentioned that the evidence regarding direct effect of different fatty acid diets on satiety in hypothalamus is sparse, previous studies have consistently reported that PUFA increases POMC expression by G-protein-coupled receptor 40 [2,3]. In the case of SFA, even though SFA decreased NPY mRNA expression [4], SFA could not alter POMC expression level [4,5], which is opposite to study of Jang et al. [1] that claimed SFA increased POMC mRNA level more prominently compared with PUFA. They should address the controversy between the previous studies and theirs.

In addition, there have been studies on the association between glucagon-like peptide-1 (GLP-1) and dietary macronutrient composition. As is already well known, GLP-1 affects food intake [6-8]. Fat-rich meal increased GLP-1 response more prominently compared with isocaloric CHO-rich meals in human [9]. In addition, dietary fat composition also affects GLP-1 response: unsaturated fatty acids increase GLP-1 more than SFA [10]. Considering that GLP-1 reduces food intake by directly stimulating POMC/cocaine- and amphetamine-regulated transcript (CART) neurons [6,7], there is a discrepancy between the previous studies [6,7] and findings of Jang et al. [1] that showed CHO-rich meal and SFA increased POMC expression level more compared with PUFA. Along with POMC stimulation, GLP-1 can also affect NPY; it indirectly inhibits neurons expressing NPY and agouti-related peptide (AgRP) via γ-aminobutyric acid (GABA)-dependent signaling [6,7]. Hypothalamic NPY and AgRP mRNAs are significantly increased by fasting, which can be significantly attenuated by GLP-1 treatment [8]. Considering the different effect on GLP-1 response according to dietary fatty acid composition, the effect of dietary fatty acid on POMC or NPY mRNA expression should be interpreted in terms of changes in GLP-1 level.

Lastly, plasma glucose, free fatty acid, lipid, leptin, and ghrelin levels are very important factors in satiety regulation. I wonder if there were any differences in these levels in response to the interventions.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

Corresponding author: Bo Kyung Koo https://orcid.org/0000-0002-6489-2656
Department of Internal Medicine, Seoul Metropolitan Government Seoul National University Boramae Medical Center, Seoul National University College of Medicine, 20 Boramae-ro 5-gil, Dongjak-gu, Seoul 07061, Korea
E-mail: bokyungkoomd@gmail.com

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