Journal of Ethnic Foods

REVIEW ARTICLE

Open Access

A journey to explore the health properties of traditional Korean diet: a commentary

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Abstract

Modern food consumption in Korea has been modified to the Western style in parallel with the rapid globalization and economic growth, which might be a cause of skyrocketing increase of cardiometabolic disorders such as obesity, type 2 diabetes mellitus, insulin resistance, dyslipidemia, hypertension, coronary artery disease, metabolic syndrome, and non-alcoholic fatty liver disease. This is not a Korea-specific phenomenon but also a worldwide common public health problem. A plausible mechanism underlying the unhealthy changes could be genetics, especially metabolic genes that cannot adequately respond to the fast dietary changes and are unable to deal with the high intake of animal fat and calorie. If this is true, then the following questions are raised: (1) do we need to go back to the traditional K-diet (K-diet) to reduce the development of these unhealthy cardiometabolic disorders?, (2) if so, what could be the underlying mechanism by which K-diet may convey those health effects?, and (3) if the K-diet is not sufficient enough to solve the current problem, how can we modify the K-diet to fit our purposes?

Keywords Korean diet, Carbohydrate metabolism, Lipid metabolism, Metabolomics, Urine organic acid, Inflammation, MicroRNA

Introduction

Modern food consumption in Korea has been modified to the Western style in parallel with the rapid globalization and economic growth, which might be a cause of skyrocketing increase of cardiometabolic disorders such as obesity, type 2 diabetes mellitus, insulin resistance, dyslipidemia, hypertension, coronary artery disease, metabolic syndrome and non-alcoholic fatty liver disease. This is not a Korea-specific phenomenon but also a worldwide common public health problem. A plausible mechanism underlying the unhealthy changes could be genetics, especially metabolic genes that cannot adequately respond to the fast dietary changes and are unable to deal with the high intake of animal fat and calorie. If this is true, then the following questions are raised: (1) do we need to go back to the traditional K-diet (K-diet) to reduce the development of these unhealthy cardiometabolic disorders?, (2) if so, what could be the underlying mechanism by which K-diet may convey those health effects?, and (3) if the K-diet is not sufficient enough to solve the current problem, how can we modify the K-diet to fit our purposes?

It is very hard to define the K-diet, but in general, it is rich in vegetables, fiber and grains, moderate to high in legumes and fish, and low in calories, red meat, and animal fat [1]. According to the recent definition by the Seoul declaration of Korean diet [2], the characteristics of K-diet were various recipes based on rice and grains, more fermented foods, vegetables, and legumes, and less red meat and deep-fat fried cooking. This characteristic of the K-diet suggests that it could be good for lipid metabolism but bad for the carbohydrate

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metabolism. To investigate the healthy properties of the K-diet, we conducted a pilot study followed by a crossover study with increased subject numbers and study duration.

In the pilot study [3], which enrolled 10 obese Korean women aged [3] 50–60 years and randomly divided into two groups, we provided each group with either a K-diet or modern Westernized Korean diet as a control diet for two weeks (n = 5). In the crossover study, which enrolled 52 obese Korean women aged 30–50 years, the components of the diets were similar to the pilot study. All subjects consumed both diets for one month each with one month washout period between the two dietary interventions so that every subject had their own control.

K-diet and lipid metabolism

In the pilot study [3], energy from total fat in the K-diet was lower than that in the control diet (19.2% vs. 27.4, $p < 0.0001$). Animal-based fat content in the K-diet was also lower than that in the control diet (2.3% vs. 10.4%, $p < 0.0001$), while plant-based fat content was comparable in both diets. The pilot study was conducted for only two weeks, but the K-diet significantly decreased the total cholesterol to the normal level (from 239.40 ± 15.14 mg/dL to 198.20 ± 13.25, $p = 0.0163$) but not in the control diet. On the other hand, low density lipoprotein cholesterol, high density lipoprotein cholesterol and triglyceride showed no change.

In the crossover study [4], calories from total fat in the K-diet were also lower than that in the control diet (19.5% vs. 24.1% $p < 0.0001$). In the K-diet group, animal-based fat content (3.88% vs. 9.54%, $p < 0.0001$) was lower, but plant-based fat content (15.6% vs. 14.5%, $p < 0.0001$) was higher than that of the control diet. The total cholesterol lowering effect of K-diet was significantly higher than that of the control diet (-30.02 ± 19.1 mg/dL vs. 12.8 ± 20.0, $p < 0.0067$), and the non-HDL cholesterol lowering effect was also significant (-21.10 ± 17.6 mg/dL vs. -6.79 ± 17.5, $p < 0.0435$). Interestingly, blood triglyceride was also significantly reduced in the K-diet group compared to the control diet (-26.35 ± 43.6 mg/dL vs. -6.58 ± 3.46, $p = 0.0177$).

In both studies, we found that K-diet can effectively reduce the total cholesterol level. Triglyceride reducing effect was not statistically significant in the 2-week intervention of K-diet (from 237.80 ± 116.24 mg/dL to 125.40 ± 16.36, p = NS) but was significantly different in the 4-week intervention of K-diet. It appears that the K-diet, which is low in fat, especially animal-based fat, is good enough to reduce total blood cholesterol and triglyceride.

K-diet and carbohydrate metabolism

In the pilot study, the K-diet provided 63.7% of calories from carbohydrates, while the control diet provided 57% of calories from carbohydrates. However, carbohydrate components were significantly different between the two diets. The K-diet had a higher proportion of total grains (277.7 ± 3.9 g/day vs. 217.4 ± 5.1, $p < 0.001$) and whole grains (267.9 ± 4.9 g/day vs. 0.4 ± 0.1, $p < 0.001$) compared to the control diet. Thus, the dietary glycemic index of the K-diet was lower than that of the control diet (49.81 ± 0.24 vs. 54.35 ± 0.53, $p < 0.0001$), while dietary glycemic load of K-diet was still higher than that of the control diet (149.98 ± 1.60 vs. 139.17 ± 2.82, $p = 0.0012$). After the 2 weeks of dietary intervention, both diet groups did not show any significant differences in the levels of fasting blood glucose, fasting insulin, and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) compared with their levels before intervention.

It appears that the high proportion of whole grains in the K-diet mitigates the high proportion of carbohydrates. However, considering the high proportion of carbohydrates in the K-diet (63.7% for the pilot study and 66.5% for the crossover study), future studies are warranted to determine the necessity of reducing the carbohydrate proportion from the K-diet.

K-diet and the other metabolisms

Clinical parameters used in our K-diet studies might be good enough to determine the clinical effects of K-diet on lipid and carbohydrate metabolisms. However, to understand the detailed mechanisms we needed more specific metabolic parameters. In the pilot study, we measured the urine organic acid profiles using LC/MS/MS [5]. Among 46 tested urine organic acids, five urine organic acids showed a difference in the K-diet group. Succinate and hydroxymethylglutarate, both of which are intermediates of energy metabolism, as well as three tryptophan metabolites, 5-hydroxyindolacetate, indican, and kynurenate, were decreased in the K-diet. It suggests that we need to investigate the effects of K-diet on the other metabolisms, and urine organic acids may provide a better understanding of metabolic pathways, through which the K-diet conveys the health effects.

In the crossover study, we measured plasma metabolites using UPLC-QTOF-MS and the results were more interesting [4]. As we found from the urine organic acid study that demonstrated the changes in three urine organic acids associated with tryptophan metabolism, the plasma tryptophan level change in the K-diet group was significantly higher than that in the control diet ($p < 0.0003$). Homocysteine lowering effects were also significantly higher in the K-diet group compared with the

control group ($p < 0.0168$). K-diet that contains higher levels of fruits (75.4 ± 1.44 g/day vs. 69.7 ± 1.73, $p < 0.001$) and vegetables (395 ± 20.6 g/day vs. 278 ± 11.4, $p < 0.001$) compared to the control diet might have provided more B vitamins that are known to reduce the blood homocysteine levels through one-carbon metabolism. In fact, folate levels were higher in the K-diet compared with the control diet (670 ± 48.4 μg vs. 503 ± 21.9, $p < 0.001$). It is well known that high homocysteine levels are associated with increased risk of cardiovascular diseases, even though it is not yet clear if reducing homocysteine through B-vitamin supplementation can actually reduce the risk of cardiovascular diseases [6]. Changes in branched chain amino acids and nucleosides such as uric acid and uridine were also different in the two diets. In sum, the data indicate that other metabolisms including amino acid metabolism and one-carbon metabolism [7] need to be investigated for the better understanding of the metabolic effects of K-diet.

K-diet and inflammation

Meta-inflammation and inflamm-aging have been highlighted to explain the mechanisms behind metabolic disorders and age-associated diseases. This type of inflammation is chronic, sustained, low grade, and subclinical without specific medicines to deal with it. It appears that a regular meal plan can be the perfect strategy to cope with such sustained inflammation given the frequency of consumption and lower intensity in meta-inflammation. It is well known that some nutrients, foods or diets have anti-inflammatory effects. Obviously, other nutrients, foods or diets can also have pro-inflammatory properties. For example, zinc has been regarded to have anti-inflammatory effect, while iron has pro-inflammatory effects.

In both the pilot study and crossover study, C-reactive protein (CRP), an inflammatory marker produced in the liver, showed no changes. In fact, CRP is the most commonly used inflammatory marker to determine the effects of diet on inflammation. On the other hand, in the pilot study nuclear factor kappa B (NF-kB), a pro-inflammatory mediator, was decreased and interleukin (IL)-10, an anti-inflammatory mediator, was elevated in the K-diet group but not in the control diet group, suggesting that K-diet may have anti-inflammatory effects [3].

When we calculated dietary inflammatory index (DII), the DII score of the K-diet group was lower than that of the control group. For the DII calculation, consumption of nutrients and foods per person per day was calculated using the CAN-Pro 5.0 program developed by the Korean Nutrition Society. All of the food parameter-specific DII scores were added to calculate the individual DII scores [3]. The lower the DII score, the more the effects were anti-inflammatory, while higher scores were more pro-inflammatory (+1 was assigned if the effects were pro-inflammatory, while −1 was assigned if the effects were anti-inflammatory) [8]. The DII score for K-diet was −0.94 and 1.04 for the control diet ($p < 0.0001$). It appears that K-diet has anti-inflammatory effects that could increase anti-inflammatory mediators and decrease pro-inflammatory mediators. Further studies are needed to delineate whether the anti-inflammatory effect of K-diet is practically effective on meta-inflammation or inflamm-aging. The K-diet study on inflammation can also be extended to the effect on immune functions that regulate inflammation.

K-diet and microRNA

MicroRNA (miRNA) has been regarded as a means by which a nutrient can affect gene expression. In the pilot study, a miRNA screening array, which measures 84 miRNAs detected in plasma, was conducted after extracting miRNAs from plasma and saliva samples [1]. Validation was done by the individual RT-qPCR. Eight plasma miRNAs showed changes in the K-diet and two plasma miRNAs showed changes in the control diet. Five saliva miRNAs showed changes in the K-diet and three saliva miRNAs showed changes in the control diet. There was no overlap between plasma miRNAs and saliva miRNAs. However, their functions are mainly associated with type 1 and type 2 diabetes mellitus. Furthermore, miRNAs associated with prediabetes, cholesterol metabolism and obesity were also found in the plasma of K-diet group and miRNAs associated with obesity, adipogenesis, non-alcoholic fatty liver disease and acute coronary syndrome were also found from the saliva of K-diet group. Collectively, circulating miRNAs associated with diabetes mellitus and cardiometabolic disorders showed changes by the K-diet. These miRNAs might be useful to determine the metabolic effects of K-diet in the future studies.

Future perspectives

Even though we cannot describe in this commentary, several unpublished data are available regarding the effects of K-diet on global and gene-specific DNA methylation, one-carbon metabolism, single nucleotide polymorphisms that can predict the response to the K-diet, leptin and adiponectin response, gut microbiota, and telomere length. In this regard, further analysis can more precisely delineate the metabolic effects of K-diet.

Diet study is more complicated than studies of individual foods, nutrients or medicines, because diet includes many different components including macronutrients, micronutrients, and bioactive food components as well as different calories and types of food stuffs, making the analysis more complex. Meticulous study designs, precise

conduction of the study, and optimal analysis are prerequisites. However, we need to carry on studies that can reveal health effects of the K-diet in the interest of public health.

Acknowledgements
Non-applicable

Author contributions
Conception of idea, reference search, writing original draft, review and editing were performed by S-W Choi.

Funding
This research was supported by the Nutrigenomics study (E0150302-06, E0220602-02) of the Korea Food Research Institute, funded by the Ministry of Science and ICT.

Availability of data and materials
There are no available data or materials.

Declarations

Competing interests
The author declares no potential competing interests.

Received: 30 December 2022 Accepted: 20 March 2023
Published online: 01 May 2023

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