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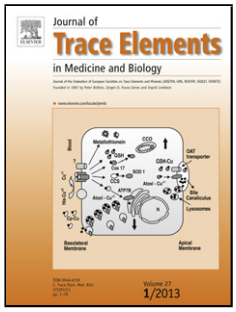

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Authors: Soo-Hwaun Kim, Young-Wook Lim, Kyung-su Park, Ji-Yeon Yang

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Relation of rice intake and biomarkers of cadmium for general population in Korea

Soo-HwaunKim[a,c], Young-Wook Lim[a], Kyung-suPark[b], Ji-YeonYang[a*]
[a.] The Institute for Environmental Research, College of Medicine, Yonsei University
[b.] Korea Institute of Science and Technology
[c.] Department of Public health, Graduate School, Yonsei University, College of Medicine, 134 Shinchon-dong, Seodeamun-gu, Seoul 120-752, Republic of Korea

Abstract

Environmental exposure to cadmium can cause renal damage. Foods containing cadmium are generally regarded as the main environmental sources of human exposure to cadmium. In this study, foods that are ingested in large amounts, including rice and other types of food with a high concentration of cadmium, were investigated to determine the correlation between the foods' cadmium content and biomarkers. The datasets required for this study, including blood cadmium concentration, biomarker concentration, and data on the amount of consumption by food item, were obtained from KNHNES. Furthermore, data on food groups with high daily exposure to hazardous amounts of cadmium were obtained by monitoring raw food sources from 2010 to 2012.

The investigation was then followed by correlation analysis, which was performed to assess the relationship between the amount of rice consumption and cadmium concentration. The Pearson coefficient analysis on the relationship between the amount of food consumption and the biomarker showed that the correlation between foods' cadmium content and blood cadmium and that of between foods' cadmium content and other biomarkers were confirmed as statistically significant in the case of the cadmium content of white rice, while, in the case of brown rice, it was confirmed by a few biomarkers.

Keywords: Cadmium; rice; food intake; biomarker

1. Introduction

Cadmium is found in most natural foods, particularly in grains and seaweed among vegetables, and in fishes and shellfishes among animals. Generally, in Asian countries like South Korea and Japan, where rice is the staple food, exposure to cadmium through rice is known as the major cause of non-occupational exposure to cadmium. Human exposure to cadmium is mostly through foods (90%). Cadmium exposure levels of 30–50 μg/day have been estimated among adults, and these levels have been linked to increased risk of bone fracture, cancer, kidney dysfunction, and hypertension [1, 2]. Diet is considered the main source of Cd intake among non-smokers [3], especially since food cultured in Cd-rich soil constitutes a major source of Cd [4]. The process by which cadmium is absorbed in the gastrointestinal tract depends on one's intake and nutritional status [5]. The exposure rate through smoking is also somewhat high, while exposure through water or air is not significant [6]. The cadmium absorbed by the body travels through the blood, and approximately 50% will be found in the liver and in the kidneys. Its biological half-life is reported to be between 10 and 30 years in the kidneys [7]. Cadmium does not only have a long half-life, but is also considered a metal accumulated in the body that is hardly excreted or metabolized [6]. Thus, it is a matter of serious concern as the elderly may be more sensitive to Cd damage compared to younger people. Previous studies also revealed that bone damage caused by Cd was more common among elderly women [8, 9, 10]. The cadmium in the body stabilizes as metallothionein, a low molecular derived protein complex, in the liver, kidneys, intestines, and muscles; then, it moves to the kidneys and is excreted with urine, thus leaving no toxic symptoms. A large amount of cadmium, however, cannot form metallothionein and causes toxic symptoms [11].

Changes in the kidney marker beta-2 microglobulin was regarded as a critical endpoint in the risk assessment of cadmium [12, 13], but there are ongoing discussions in the scientific literature concerning other potential critical effects, including bone damage and cancer [14, 15]. Investigations indicate that a critical concentration in urine similar to that established by the EFSA for beta-2 microglobulin also apply to cadmium-induced osteoporosis [16]. Cadmium hinders generative functions, raises the risk of preeclampsia, and causes blood vessel disorders in the testicles in the long term [11]. The absorbed cadmium spreads around the entire body through the blood, but it is mostly accumulated in the liver and in the kidneys [17]. Right after cadmium is absorbed, cadmium concentration in the liver rises dramatically, but it is then slowly redistributed to the kidneys. As absorption continues, the concentrations of cadmium in the liver and in the kidneys also increase [18]. The lethal dose of cadmium is 20-30