EFFECTS OF MINERAL-RICH SOLAR SALT ON BLOOD PRESSURE AND INSULIN RSISTANCE IN RATS

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Introduction

Salt plays various important roles in human physiology such as maintaining membrane potential of cells, nutrient absorption and transport, and maintaining blood volume and blood pressure. There is no substitute for salt and a regular intake is required to maintain body functions.

However, it is known that excessive intake of salt causes increment of blood pressure and is a major factor of circulating system-related diseases. However, this knowledge comes from researches that used reagent-grade salt, purified salt or rock salt, all of which are lacking of minerals. There are various types of salt for food consumption. Korean solar salt, which is produced in tide flat (marsh land), has plenty of minerals such as potassium, calcium, magnesium, etc. than other salts. Those minerals have been reported to reduce blood pressure. Thus, it is quite possible that mineral-rich salt (MRS) does not increase blood pressure as much as mineral-deficient salt (MDS) does. We have investigated this possibility using salt-sensitive rats and Sprague Dawley rats fed mineral-rich Korean solar salt and mineral-deficient salt.

On the other hand, insulin resistance is also known to be induced by high-salt consumption. Insulin resistance contributes significantly to the pathophysiology of type 2 diabetes mellitus. Type 2 diabetes mellitus is affecting more than 150 million people worldwide. It is characterized by insulin resistance, impaired glucose tolerance (IGT) and reduced glucose uptake from blood into adipose tissue and skeletal muscle. One mechanism that mediates insulin resistance involves phosphorylation of serine residue in insulin receptor substrate-1 (IRS-1) as known to be promoted by elevated circulating levels of several metabolites, including free fatty acid and glucose. Moreover, adipose-derived cytokines like TNF-α also activate Ser^{307} phosphorylation of IRS-1 by inhibiting PTB domain, which uncouples IRS-1 from insulin receptor, leading to diminish the recruitment of PI 3-kinase and to stimulate the IRS-1 degradation pathway. Insulin-resistant states and Ser^{307} phosphorylation of IRS-1 are associated with the activation of NF-κB. However, the precise molecular mechanisms by which NF-κB contributes to the development of insulin resistance remain unclear. In this study, we have investigated whether effects of mineral-rich solar salt on insulin resistance and signaling are different from those of mineral-deficient-salt.

Summary

Experiments were designed using salt-sensitive or normal rats fed diets containing various types of salts to investigate the differences in the effects between mineral-rich salt (MRS) and mineral-deficient salt (MDS) on blood pressure and insulin resistance. The blood pressures in rats fed high salt diets gradually increased compared to that of rat fed normal diet containing no salt during four weeks. However, MRS group fed mineral-rich salt showed significantly lower systolic and diastolic blood pressures than MDS group in any cases using salt-sensitive

rat and normal rat, indicating that effect of mineral-rich dietary salt on blood pressure is different from that of mineral-deficient salt. On the other hand, male Sprague-Dawley rats were fed diets containing the same amount of NaCl (8%) of MRS (94% NaCl) and MDS (more than 99% NaCl) for four weeks to see whether the effects of mineral-rich salt (MRS) on insulin resistance and insulin signaling are different from those of MDS. Our research suggests that MRS intake is preferable to MDS intake in reducing the risk of insulin resistance by improving glucose tolerance and insulin-stimulated glucose uptake. Moreover, MRS group enhanced insulin signaling by diminishing Ser^{307} phosphorylation of IRS-1 and the expression of NF-κB and IκB that are involved in inflammation compared to MDS group.