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Effects of Selenomethionine and Sodium Selenite Supplementation on the Risks of Type-2 Diabetes in Kuo Kondo Alel-y (KKAy) Mice under Different Status of Selenium Level

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ABSTRACT

Introduction: Oxidative stress in obesity raises the risks of type-2 diabetes. Selenium, an essential micronutrient, has antioxidant properties known as GPx and Se-P that counteract reactive oxygen species (ROS) in oxidative stress and are involved in insulin signaling process. Selenium status of subjects before supplementation (baseline status) and types of selenocompound supplemented are presumed to contribute to different effects of selenium to the risks of type-2 diabetes. This study aimed to clarify the effects of different baseline status and selenocompound types in selenium supplementation to the risks of type-2 diabetes.

Methods: Six-weeks-old KKAy mice were fed high fat diet (HFD) and grouped into deficient (no selenium) and sufficient (selenomethionine 0.1 ppm) groups for two weeks. For the next 4 weeks, deficient mice were subgruped into: 1) deficient control (n=10), 2) deficient + selenomethionine (n=10), and the sufficient mice into: 3) sufficient control (n=10), 4) sufficient + selenomethionine (n=10), and 5) sufficient + selenite (n=10). Selenium levels, GPx activity, blood glucose, oral glucose tolerance test (OGTT), HOMA-IR, QUICKI, insulin, and adiponectin levels were measured and analyzed by using t test and ANOVA.

Results: After HFD feeding, KKAy mice gained significant weight but no difference was found among the groups. Selenium levels of sufficient group at baseline (8-weeks-old) were higher compared to deficient group [t=1.432 p=0.202 (plasma), t=1.98, p=0.18 (liver), t=6.616 p= 0.003 (kidney)]. At the end of study (12-weeks-old), selenium levels and GPx activities of all supplemented groups increased and all mice became diabetic except one, with sufficient groups had lower blood glucose than deficient groups. Blood glucose of sufficient+selenomethionine group of OGTT at minute 120 (161.60 ± 76.80 mg/dL) was lower than sufficient control and sufficient+selenite groups, with mean 336.75 ± 57.90 mg/dL (p=0.017) and 306.20 ± 92.90 mg/dL (p=0.035), respectively. Sufficient+selenomethionine group also had non-significantly better insulin sensitivity parameters (HOMA-IR and QUICKI) compared to other supplemented groups. Whereas, adiponectin levels in all groups dropped at the end of study period.

Conclusion: Representing obese and type-2 diabetic models, KKAy mice in selenium sufficient groups gained benefit from selenomethionine supplementation as they were more glucose tolerant than non supplemented and selenite supplemented mice.