Abstract


Vitamin K2 alleviates type 2 diabetes in rats by induction of osteocalcin gene expression.

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OBJECTIVES: The biological mechanisms behind the association between vitamin K (Vit K) and glucose metabolism are uncertain. We aimed to analyze the expression of insulin 1 (Ins 1), insulin 2 (Ins 2) and cyclin D2, the expression of adiponectin and UCP-1. In addition, we aimed to estimate the doses of Vit K2 able to affect various aspects of glucose and energy metabolism in type 2 diabetes.

METHODS: Thirty adult male rats were allocated equally into five groups: control group, diabetes mellitus group, and groups 3, 4, and 5, which received Vit K2 at three daily dose levels (10, 15, and 30 mg/kg, respectively) for 8 wk. At the end of the study, blood samples were collected to quantify total osteocalcin, fasting plasma glucose, fasting insulin, and relevant variables. The expression of OC, Ins 1, Ins 2, cyclin D2, adiponectin, UCP-1 genes was analyzed by real-time polymerase chain reaction.

RESULTS: After administration of Vit K2, a dose-dependent decrease in fasting plasma glucose, hemoglobin A1c and homeostatic model assessment method insulin resistance, and a dose-dependent increase in fasting insulin and homeostatic model assessment method β cell function levels, when compared with diabetes mellitus rats, were detected. There was significant upregulation of OC, Ins 1, Ins 2, or cyclin D2 gene expression in the three treated groups in a dose-dependent manner when compared with the diabetic rats. However, expression of adiponectin and UCP-1 were significantly increased at the highest dose (30 mg/kg daily) only.

CONCLUSIONS: Vit K2 administration could improve glycemic status in type 2 diabetic rats by induction of OC gene expression. Osteocalcin could increase β-cell proliferation, energy expenditure, and adiponectin expression. Different concentrations of Vit K2 were required to affect glucose metabolism and insulin sensitivity.

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