



P53 Rather Than B-Catenin Mediated the Combined Hypoglycemic Effect of Innamomum Cassia (L.) and Zingiber of Ficinale Roscoe in The Streptozotocin-Induced Diabetic Model

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Abstract

Background

The antioxidant, hypoglycemic, and insulin-enhancing effects of ginger and cinnamon were previously confirmed in experimental and human studies, while the combined effect of ginger and cinnamon was not thoroughly investigated until now.

Objectives

This study was designed to assess the antidiabetic effect of administration of ginger (*Zingiber officinale* Roscoe) and cinnamon (*Cinnamomum cassia* L.) in streptozotocin (STZ)-induced diabetic rats compared to metformin and to explain the mechanism behind this effect.

Materials and methods: STZ was utilized to induce diabetes mellitus in male Sprague-Dawley rats.

Assessments of fasting blood glucose level (BGL), the total antioxidant capacity (TAC), serum insulin, HOMA-IR, and HOMA- β cells were performed. Pancreatic gene expression of β -catenin and p53 was assessed using RTPCR.

Assessment of histopathological alterations of pancreatic islet cells was performed using routine and immunohistochemical techniques.

Results

BGL significantly decreased ($p < 0.01$), while serum insulin and TAC significantly increased ($p < 0.001$) in both metformin- and ginger plus cinnamon-treated groups compared to the untreated diabetic group. HOMA- β cell index significantly increased ($p < 0.001$) in ginger plus cinnamon, indicating their enhancing effect on insulin secretion in diabetic conditions. p53 gene expression was significantly upregulated ($p < 0.001$), while β -catenin was insignificantly downregulated ($p < 0.32$) in ginger plus cinnamon-treated groups. Insulin immunoexpression in β cells significantly increased ($p < 0.001$, $p < 0.004$) in metformin- and ginger plus cinnamon-treated groups, respectively.

Conclusions

The combined administration of ginger and cinnamon has a significant hypoglycemic and antioxidant effect in STZ-induced diabetes mostly through enhancing repair of islet cells mediated via upregulation of pancreatic p53 expression. Therefore, testing this effect in diabetic patients is recommended.