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1-deoxynojirimycin inhibits glucose absorption and accelerates glucose metabolism in streptozotocin-induced diabetic mice

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We investigated the role of 1-deoxynojirimycin (DNJ) on glucose absorption and metabolism in normal and diabetic mice. Oral and intravenous glucose tolerance tests and labeled 13C6-glucose uptake assays suggested that DNJ inhibited intestinal glucose absorption in intestine. We also showed that DNJ down-regulated intestinal SGLT1, Na+/K+-ATP and GLUT2 mRNA and protein expression. Pretreatment with DNJ (50 mg/kg) increased the activity, mRNA and protein levels of hepatic glycolysis enzymes (GK, PFK, PK, PDE1) and decreased the expression of gluconeogenesis enzymes (PEPCK, G-6-Pase). Assays of protein expression in hepatic cells and in vitro tests with purified enzymes indicated that the increased activity of glucose glycolysis enzymes was resulted from the relative increase in protein expression, rather than from direct enzyme activation. These results suggest that DNJ inhibits intestinal glucose absorption and accelerates hepatic glucose metabolism by directly regulating the expression of proteins involved in glucose transport systems, glycolysis and gluconeogenesis enzymes.

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