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Administration of 1-deoxynojirimycin attenuates hypothalamic endoplasmic reticulum stress and regulates food intake and body weight in mice with high-fat diet-induced obesity

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The α -glucosidase inhibitor, 1-deoxynojirimycin (DNJ), is widely used for its antiobesity and antidiabetic effects. Researchers have demonstrated that DNJ regulates body weight by increasing adiponectin levels, which affects energy intake and prevents diet-induced obesity. However, the mechanism by which centrally administered DNJ exerts anorexigenic effects has not been studied until now. We investigated the effect of DNJ in the hypothalamus of mice with high-fat diet-induced obesity. Results showed that intracerebroventricular (ICV) administration of DNJ reduced hypothalamic ER stress, which activated the leptin-induced Janus-activated kinase 2 (JAK2)/signal transducers and activators of transcription 3 (STAT3) signaling pathway to cause appetite suppression. We conclude that DNJ may reduce obesity by moderating feeding behavior and ER stress in the hypothalamic portion of the central nervous system (CNS).

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