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Gomisin N inhibits adipogenesis and prevents high-fat diet-induced obesity

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Gomisin N (GN) is a physiological lignan derived from *Schisandra chinensis*. In the present study, we investigated the inhibitory effects of GN on differentiation of 3T3-L1 preadipocytes and the anti-obesity effects of GN in high-fat diet (HFD)-induced obese mice. Incubation with GN significantly inhibited the differentiation of 3T3-L1 preadipocytes in a dose-dependent manner. This inhibitory effect primarily occurred at an early adipogenic stage through impairment of mitotic clonal expansion (MCE) caused by cell cycle arrest at the G1/S phase transition. GN inhibited the extracellular signal-regulated kinase and phosphoinositide 3-kinase/protein kinase B signaling in the MCE process and activated AMP-activated protein kinase. Furthermore, GN down-regulated CCAT/enhancer-binding protein β (C/EBPβ) and histone H3K9 demethylase JMJD2B during early stages of adipogenesis and therefore repressed the expression of C/EBPβ-targeted cell cycle genes. In addition, GN also repressed the expression of histone H3K4 methyltransferase MLL4 and reduced PPARγ expression. Moreover, GN effectively lowered the final body weight, adipose tissue mass and reduced the serum levels of glucose, total triglyceride and cholesterol in the HFD-induced obese mice. GN also markedly reduced hepatic triglyceride level induced by HFD. Collectively, these findings suggest that GN has potential as a novel agent for the prevention and treatment of obesity.

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