Influence of vitamin D treatment on transcriptional regulation of insulin-sensitive genes.

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Abstract

BACKGROUND: Obesity is a risk factor for diabetes and metabolic syndrome, which are characterized by insulin resistance. Inflammation is a co-morbid condition associated with obesity. Vitamin D, besides being a transcriptional regulator, is an inflammation suppressor. However, the role of vitamin D in alleviating obesity-induced insulin resistance is still not well understood.

METHODS: The influence of vitamin D treatment on the transcriptional level of insulin receptor (IR), insulin receptor substrate (IRS-1), glucose transporter type 4 (GLUT-4), and vitamin D receptor (VDR) in insulin target tissues of liver, adipose, and muscle of mice fed on a high-fat diet (HFD) or low-fat diet (LFD) was studied by quantitative RT-PCR.

RESULTS: A gradual weight reduction was observed in HFD-fed mice treated with vitamin D compared to a steady weight increase in control animals (P<0.01). In HFD mice, vitamin D decreased VDR expression to 0.5-fold in muscle (P=0.002), and increased it to 3.6-fold in the liver (P<0.001); however, VDR transcription was unaltered in adipose tissue. Similarly, vitamin D did not influence tissue expression of IR in either LFD- or HFD-fed mice. Muscle IRS-1 transcription level was upregulated to 2.4-fold (P=0.005) in HFD mice, whereas it was reduced to 0.15-fold in liver tissue (P<0.001). Vitamin D treatment had no effect on GLUT-4 transcript levels in any of the tissues under HFD conditions.

CONCLUSION: Vitamin D treatment influenced the expression of insulin-sensitive genes in a tissue-specific fashion. On the basis of the present findings, vitamin D does not aid glucose transport across cells of liver and adipose tissues, the major insulin-sensitive tissues, in HFD-fed mice; however, it appears to enhance the intracellular mechanisms of insulin action mediated by IRS-1 and VDR in muscle tissue.

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