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The role of vitamin D in weight-related metabolic derangements, inflammation and insulin resistance

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The role of vitamin D in maintaining calcium homeostasis and bone mineralization is well-established. We investigated the effect of vitamin D on weight-related inflammation, metabolic derangements and associated histological changes of the live. Vitamin D treatment induced a significant gradual decrease in animals' weight under high fat diet fed conditions compared to a steady increase in the weights of controls. Furthermore, vitamin D reduced the levels of inflammatory markers including TNF-α, CRP and IL-6. There were also lower concentrations of insulin and C-peptide, which indicates improvement in insulin resistance. Liver histology showed a marked accumulation of fat droplets in approximately 60-70% of hepatocytes of animals while vitamin D rendered the whole structure more normal. Transcriptional regulation of genes in tissues primarily involved in glucose homeostasis in response to vitamin D treatment revealed a general trend of up-regulation in the expression levels of insulin receptor (IR), glucose transporter-4 (Glut4), insulin receptor substrate (IRS) and vitamin D receptor (VDR) in particular under conditions resembling obesity (high fat fed animal model). These data signify an important effect of vitamin D on weight-related insulin resistance and a protective consequence on the liver structure.

Biography

Khalid M Alkharfy is Associate Professor and the team leader of drug discovery and development at the Biomarkers Research Program (BRP) of King Saud University (KSU), Riyadh, Saudi Arabia. He received his PhD in Pharmaceutical Sciences from the University of Pittsburgh, Pittsburgh, PA, USA. After completing his BSc Pharm from KSU, he earned a PharmD degree from the University of Tennessee, Memphis, TN, USA. Khalid M Alkharfy is the lead or contributing author on many peer-reviewed articles, book chapters, and an editorial member of reputed journals; he has multiple international conference presentations.

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