RGS2 and Human Adipogenesis

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Abstract

Obesity is characterized by excess adipose tissue and is one of the leading public-health issues of the industrialized world. Gain of adipose tissue can result from accumulation of fat in existing adipocytes (fat cells), as well as increased commitment of stem cells into new adipocytes through a process known as adipogenesis. Although a number of key adipogenic transcription factors such as PPARgamma2 and CEBPalpha are well characterized, the temporal and spatial molecular and cellular events that occur during adipogenesis are still largely unknown. Better understanding of these events could have important implications in finding treatments for obesity.

Human mesenchymal stem cells (hMSCs) are a type of adult stem cells that can commit to a number of mature cell types including adipocytes upon receiving appropriate stimuli. Through a microarray study, a number of genes whose expression was significantly up- or down-regulated during adipogenesis were uncovered, including RGS2, a member of the Regulators of G-protein signaling factors (RGS) family. RGS2 is a GTPase regulator and is significantly up regulated upon adipogenic stimulation. In this study, we focus on understanding the role of RGS2 in the adipogenic lineage commitment of adipose-derived hMSCs (ad-hMSCs).

Our results so far demonstrated that down regulation of RGS2 at 40-50% level as induced by *siRGS2* during adipogenic initiation significantly inhibited adipogenesis, suggesting that RGS2 plays a positive role in the adipogenic commitment process of ad-hMSCs. Future studies will focus on examining the effect of RGS2 overexpression during adipogenesis.

Keywords

RGS2; Human Mesenchymal Stem Cells (hMSCs); Adipogenesis

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