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Resistin, a 12.5-kDa cystein-rich protein, is recently postulated to be an important link between obesity and insulin resistance. Although it has been reported that resistin is mainly expressed and secreted by adipocytes, it has not been understood which transcription factors are involved in this gene regulation. To investigate the molecular mechanism of resistin gene expression, we cloned human resistin promoter by use of PCR. Sequence analysis of 5'-UTR of resistin promoter reveals that there are putative binding sites for several transcriptional factors including SREBP, C/EBP, P300 and Sp1 (GC box). Gel mobility shift assays showed that ADD1/SREBP1c binds to the SRE region in the resistin promoter. In order to study the transcriptional regulation of the human resistin gene, we constructed luciferase reporter containing resistin promoter (-816~+89). With this construct, we found that ADD1/SREBP1c transactivates the promoter of human resistin via SRE dependent manner. These results clearly demonstrated that ADD1/SREBP1c is required for the expression of resistin gene during adipogenesis.

D121 Expressin Pattern of Runt Domain Transcription Factor, run, in Caenorhabditis elegans

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The *C. elegans* run gene encodes a Runt domain factor. Runx1, Runx2, and Runx3*Runx1, Runx2, Runx3* are three known mammalian homologs of *run*. *Runx1* has been identified at the breakpoint of chromosome translocations responsible for human leukemia and plays an essential role in hematopoiesis. *Runx2* plays an essential role in osteogenesis and cleidocranial dysplasia. To understand possible role of Run in *C. elegans*, we used GFP reporter constructs and ds RNAi. The expression of run was detected as early as bean stage exclusively in the nuclei of seam cells and lasted until L3 stage. At larval stage, expression of run was additionally detected in intestinal cells. The stage and and cell type specific expression was regulated by 7.2 kb long intron located between exon 3 and 4. ds RNAi analysis to target run gene showed early larval lethal phenotype with apparent malformation or degeneration of hypodermis and intestine. These results suggests that run is involved in hypodermis and gut development.

D122 HSP16-1, a small heat shock protein in C. elegans, is strongly induced by hypoxia stress

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Many heat shock proteins (HSPs) are induced by various stresses. We found that the expressions of the HSP16 family proteins were up-regulated by ethanol in a microarray analysis of ethanol-treated *C. elegans* animals. In this report, we characterized HSP16-1, one of the small HSP proteins. We found that HSP16-1 is a chaperone that responds to general stresses. Furthermore, we found that HSP16-1 protein is the only HSP16 protein to be significantly induced by hypoxia. We found that induction of HSP16-1 by hypoxia required the distal heat shock responsive elements (HSE) and its neighboring region, while heat shock induction of HSP16-1 needs only one of the two HSEs existing in the promoter. The induction of HSP16-1 by hypoxia was independent of the conventional HIF-1/HRE, indicating that the hypoxia response of HSP16-1 protein may be mediated by a new mechanism.