CIS REGULATORY MODULE DISCOVERY IN TH1 CELL DEVELOPMENT

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Abstract:

Immune response enables the body to resist foreign invasions. The Inflammatory response is an important aspect in the immune response which is articulated by elements such as cytokines, APC, T-cell and B-cell, effector cell or natural killer. Of these elements, T-cells especially T-helper cells; a sub class of T-cells plays a pivotal role in stimulating the immune response by participating in various biological reactions such as, the transcription regulatory network. Transcriptional regulatory mechanisms are mediated by a set of transcription factors (TFs), that bind to a specific region (motifs or transcription factor binding sites, TFBS), on the target gene(s) controlling the expression of genes that are involved in T-helper cell mediated immune response. Eukaryotic regulatory motifs, referred to as cis regulatory modules (CRMs) or cistrome, co-occur with

the regulated gene's transcription start site (TSS) thus, providing all the essential components for building the transcriptional regulatory networks that depends on the relevant TF-TFBS interactions. Here, we study IL-12 stimulated transcriptional regulators in STAT4 mediated T helper 1 (Th1) cell development by focusing on the identification of TFBS and CRMs using a set of Stat4 ChIP-on-chip target genes. A region containing 2000 bases of Mus musculus sequences with the Stat4 binding site, derived from the ChIP-on-chip data, has been characterized for enrichment of other motifs and, thus CRMs. Our experiments identify some potential motifs, (such as NF-κB and PPARγ/RXR) being enriched in the Stat4 binding sequences compared to neighboring background sequences. Furthermore, these predicted CRMs were observed to be associated with biologically relevant target genes in the ChIP-on-chip data set by meaningful gene ontology annotations. These analyses will enable us to comprehend the complicated transcription regulatory network and at the same time categorically analyze the IL-12 stimulated Stat4 mediated Th1 cell differentiation.

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