東北大学グローバルCOE

Network Medicine 創生拠点

NM高等教育セミナー

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「Signaling to chromatin for transcription regulation」

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医学部5号館 201号室

In response to extracellular cues, signal transduction activates downstream transcription factors to induce expression of target genes. We demonstrate that the ATAC (Ada Two A Containing) histone acetyltransferase (HAT) complex serves as a transcriptional co-factor for c-Jun at the Jun N-terminal kinase (JNK) target genes jra and chickadee. ATAC is required for c-Jun occupancy of these genes. The Atac2 subunit is required for H4K16 acetylation at the Jra enhancer, promoter and transcribed sequences. Under conditions of osmotic stress, ATAC co-localizes with c-Jun, recruits the upstream kinases Misshapen, MKK4 and JNK and suppresses further activation of JNK. Relocalization of these MAPKs and suppression of JNK activation by ATAC is dependent on the CG10238 subunit of ATAC. Thus, ATAC governs the transcriptional response to MAP kinase signaling by serving as both a co-activator of transcription and as a suppressor of upstream signaling.

参考文献

1. Signals and combinatorial functions of histone modifications. Suganuma T, Workman JL. Annu Rev Biochem. 2011; 80: 473-99. (Review)
2. The ATAC acetyltransferase complex coordinates MAP kinases to regulate JNK target genes. Suganuma T, Mushegian A, Swanson SK, Abmayr SM, Florens L, Washburn MP, Workman JL. Cell. 2010; 142 (5): 726-36.

本セミナーは医学履修課程特別セミナーを兼ねています。受講学生は履修振替簿を持参し、セミナー修了後にサインを受けること。聴講は自由大歓迎です。学部生の皆さんもぜひどうぞ。

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