

ELL

Reactivity:Human Mouse

Tested applications:WB

Recommended Dilution:WB 1:200 - 1:500

Calculated MW:68kDa

Observed MW:Refer to Figures

Immunogen:

A synthetic peptide of human ELL

Storage Buffer:

Store at 4. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Synonym:

ELL;C19orf17;DKFZp434I1916;ELL1;MEN ;

Catalog #:A0668

Antibody Type:

Polyclonal Antibody

Species:Rabbit

Gene ID:8178

Isotype:IgG

Swiss Prot:P55199

Purity:Affinity purification

For research use only.

Background:

Actin is a key regulator of RNA polymerase (Pol) II-dependent transcription. Positive transcription elongation factor b (P-TEFb), a Cdk9/cyclin T1 heterodimer, has been reported to play a critical role in transcription elongation. However, the relationship between actin and P-TEFb is still not clear. In this study, actin was found to interact with Cdk9, a catalytic subunit of P-TEFb, in elongation complexes. Using immunofluorescence and immunoprecipitation assays, Cdk9 was found to bind to G-actin through the conserved Thr-186 in the T-loop. Overexpression and in vitro kinase assays showed that G-actin promotes P-TEFb-dependent phosphorylation of the Pol II C-terminal domain. An in vitro transcription experiment revealed that the interaction between G-actin and Cdk9 stimulated Pol II transcription elongation. ChIP and immobilized template assays indicated that actin recruited Cdk9 to a transcriptional template in vivo and in vitro. Using cytokine IL-6-inducible p21 gene expression system, we revealed that actin recruited Cdk9 to endogenous gene. Moreover, overexpression of actin and Cdk9 increased histone H3 acetylation and acetylated histone H3 binding to a transcriptional template through the interaction with histone acetyltransferase, p300. Taken together, our results suggested that actin participates in transcription elongation by recruiting Cdk9 for phosphorylation of the Pol II C-terminal domain, and the actin-Cdk9 interaction promotes chromatin remodeling.

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