PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

Transcriptional switch

ArticleInfo		
ArticleID	:	4177
ArticleDOI	:	10.1186/gb-spotlight-20010815-01
ArticleCitationID	:	spotlight-20010815-01
ArticleSequenceNumber	:	248
ArticleCategory	:	Research news
ArticleFirstPage	:	1
ArticleLastPage	:	2
ArticleHistory	:	RegistrationDate : 2001–08–15 OnlineDate : 2001–08–15
ArticleCopyright	:	BioMed Central Ltd2001
ArticleGrants	:	
ArticleContext	:	130592211

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Transcription is regulated by the coordinated assembly of protein complexes that synergize to switch on gene expression. The virally induced enhanceosome of the interferon-β (IFN-β) gene is one of the best characterized transcriptional switches. In the August 10 Science, Nikhil Munshi and colleagues from Columbia University describe how acetylation of the architectural high-mobility-group protein HMGI(Y) regulates stability of the IFN-β enhanceosome (*Science* 2001, **293:**1133-1136). The HMGI(Y) protein is a target for acetylation by two different enzymes (CBP and PCAF/GCN5) on distinct lysine residues (Lys65 and Lys71, respectively). Munshi *et al.* mutated Lys71 and observed decreased virus-induced transcription and reduced interaction with enhanceosome proteins. They propose that HMGI(Y) acetylation by PCAF/GCN5 facilitates enhanceosome assembly. They performed chromatin immunoprecipitation experiments to demonstrate that K71 acetylation coincides with enhanceosome assembly and activation (3 hours-post infection), whereas Lys65 acetylation occurred at the time of enhanceosome disruption (after 6 hours). Also, Lys71 acetylation decreased the efficiency of CBP acetylation on Lys65. Munshi *et al.* propose a model in which the ordered recruitment of, and acetylation by, PCAF/GCN5 followed by CBP regulates enhanceosome stability (assembly and disassembly, respectively) and the transcriptional switch.

References

1. Virus induction of human IFN beta gene expression requires the assembly of an enhanceosome

2. *Science* , [http://www.sciencemag.org]

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