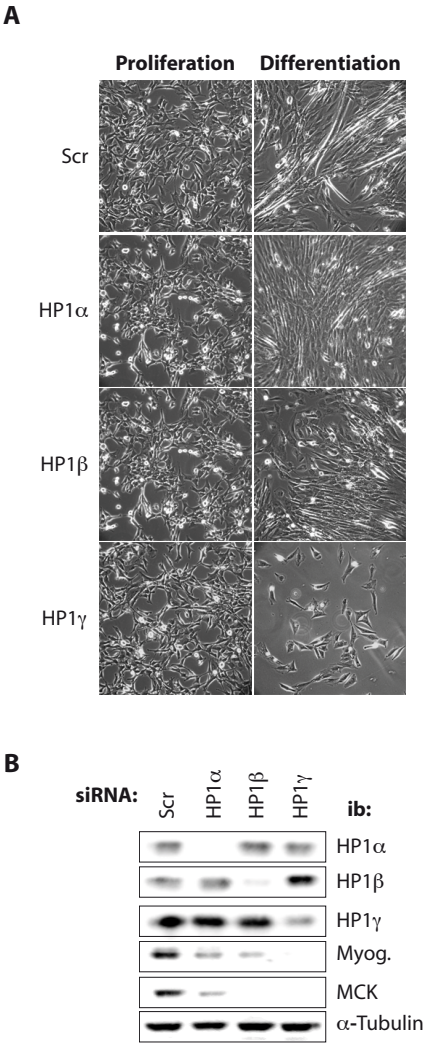
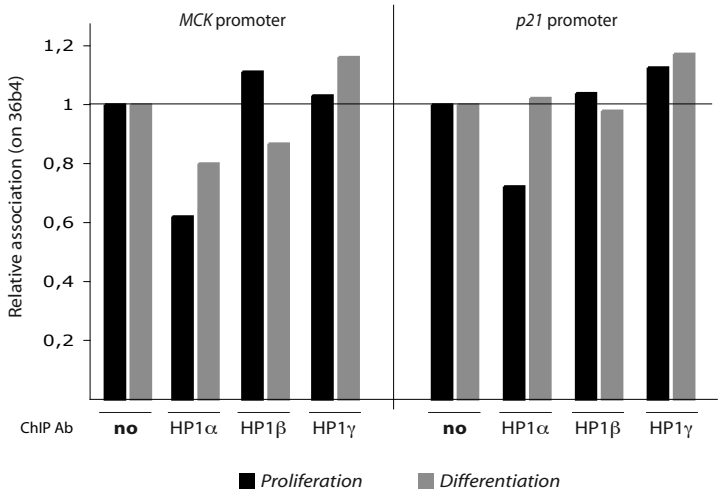
Supplemental data 1, Yahi et al.



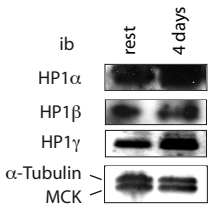
Supplemental data 1: HP1 isoforms knock downs affect muscle terminal differentiation. **A, B.** C2C12 myoblasts were transfected with control siRNA (Scrambled, Scr) or with HP1 isoform-specific siRNAs as indicated. 48 hours post-transfection (Proliferation) cells were placed in differentiation medium (Differentiation) for 96 hours. Cells were then analyzed by microscopy (**A**) and by western blotting with isoform-specific anti-HP1, anti-myogenin (Myog.), anti-MCK, and anti-a-tubulin as a loading control (**B**). Note that the kinetic studies were carried out in the same 10 cm diameter cell culture dish for each sample.

Data not shown, Yahi et al.



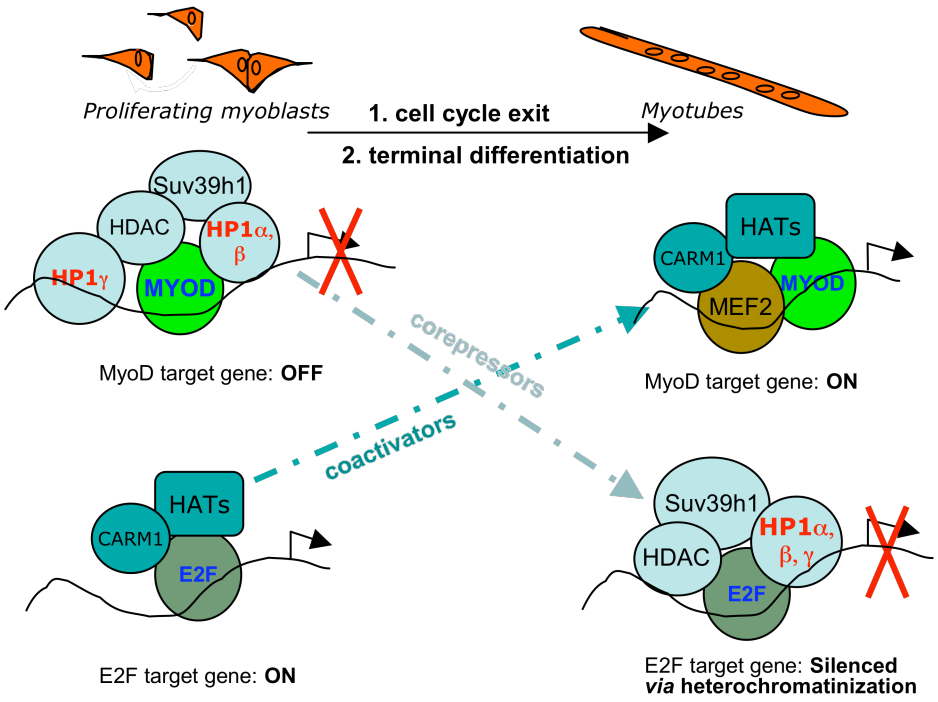
Data not shown: Study of the possible association of HP1 isoforms and transcribed regions of MyoD traget genes *p21* and *MCK*.
Chromatin fractions from either proliferating C2C12 myoblasts (black bars), or cultured 48 hours in differentiating medium (grey bars), were immunoprecipitated using anti-HP1 antibodies (HP1) or no antiobody (no), and analyzed by quantitative PCR (as in Figure 5). We quantified copy numbers of *p21* and *MCK* coding regions (in exons). *36B4* gene were used as negative controls to normalize the ChIP results. Results are the mean of 2 independent experiments.
The exonic primers used were :
-*p21*: forward: gtccaatcctggtgatgtc; reverse: tagaccttgggcagcccta
-*MCK* : forward: gccgtgttcgacatctcca; reverse: acgagcccaatgattggac

Data not shown, Yahi et al.



Data not shown: Expression of HP1 isoforms in regenerating muscle.
Tibialis anterior muscles of mice were injected with cardiotoxin (*as described in Duquet A. et al, EMBO Rep, 2006*), and regeneration was monitored for a period of 4 days. HP1 isoforms (α, β, γ), Muscle Creatine Kinase (MCK), and α-Tubulin (loading control) were detected by western blot. rest: control mice.

Supplemental data 2, Yahi *et al*.



Supplemental data 2: Proposed model for the dual role of HP1 proteins during muscle differentiation.

In proliferating myoblasts, HP1 proteins repress MyoD target genes, while in differentiating myoblasts they switch onto E2F target promoters to silence them. Note that in muscle system, proliferation inhibition or cell cycle exit is a pre-requisite to terminal differentiation. Thus, down-regulation of HP1 (or that of Suv39h1) in myoblasts results in a defect in E2F target genes silencing, and consequently in differentiation inhibition.

HAT: Histone acetyltransferase.