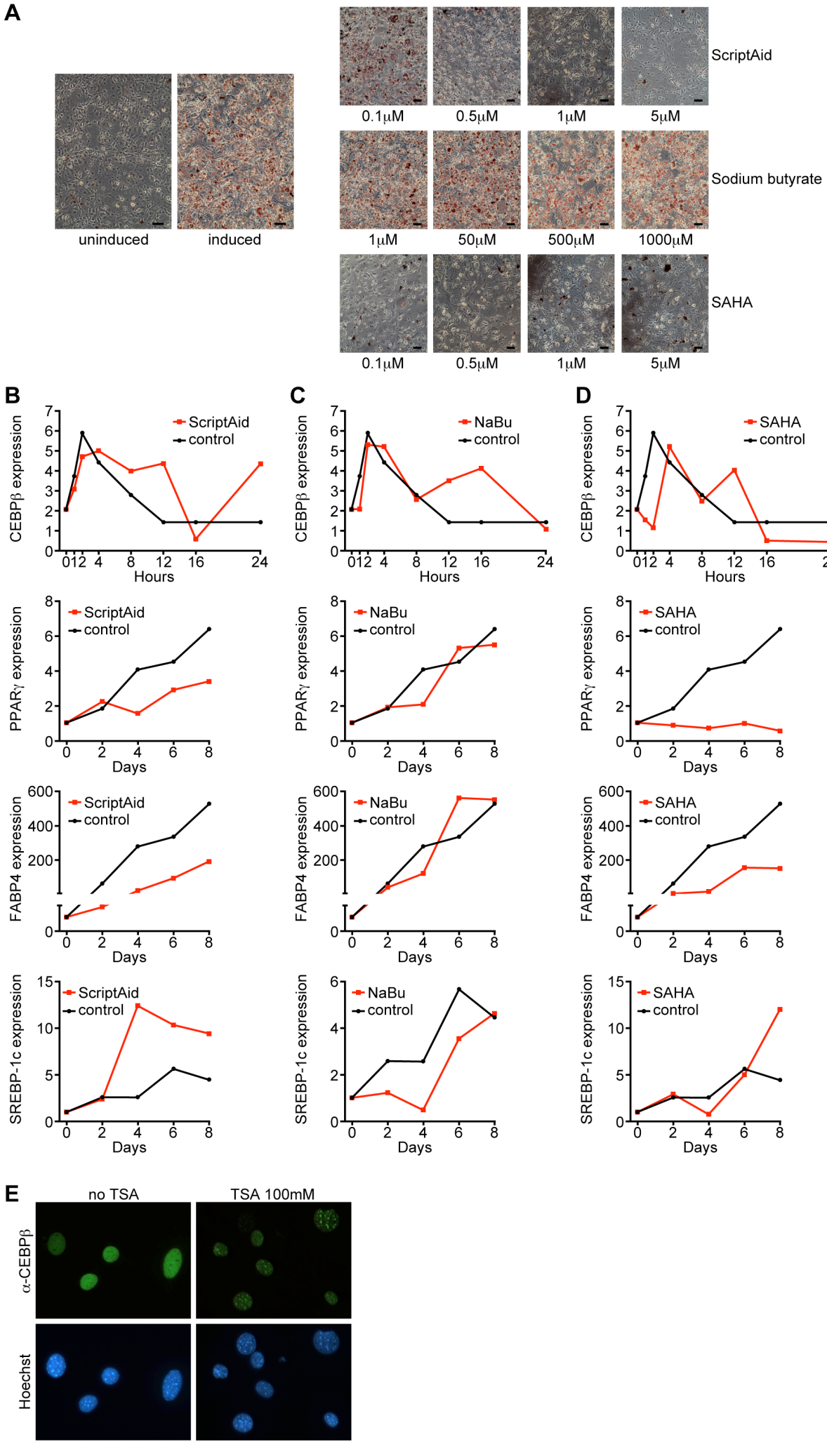
Supplemental Figure 1: Different HDAC inhibitors block adipogenesis in 3T3-L1 cells and TSA induces redistribution of C/EBPβ in transcriptionally inactive chromatin regions. (A-D) Treatment of pre-adipocytes (3T3-L1 cells) with a hormone inducer cocktail for 8 days leads to effective adipogenesis, which can be monitored by staining with ORO. The HDAC inhibitors Scriptaid and SAHA block adipocyte differentiation in a dose-response manner. Sodium butyrate, a short chain fatty acid, does not inhibit adipogenesis. Transcriptional profiling of adipogenic marker genes shows that treatment of 3T3-L1 cells with Scriptaid or SAHA (but not sodium butyrate) blocks the activation of the transcription of PPARγ but does not reduce the extent of the upregulation of SREBP-1c or C/EBPβ transcription. (E) Immunofluorescence shows redistribution of C/EBPβ in the nucleus of TSA-treated 3T3-L1 cells after 18 h of induction of differentiation. We hypothesize that HDAC inhibitors, such as TSA can block adipogenesis in 3T3-L1 cells induced to differentiate by affecting the acetylation state of C/EBPβ, consequently causing C/EBPβ to be sequestered in transcriptional inactive chromatin regions.


A
uninduced
induced
ScriptAid
0.1μM
0.5μM
1μM
5μM
Sodium butyrate
1μM
50μM
500μM
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SAHA
0.1μM
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B
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CEBPβ expression
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FABP4 expression
SREBP-1c expression
Hours
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ScriptAid
NaBu
SAHA
control
E
no TSA
TSA 100mM
α-CEBPβ
Hoechst