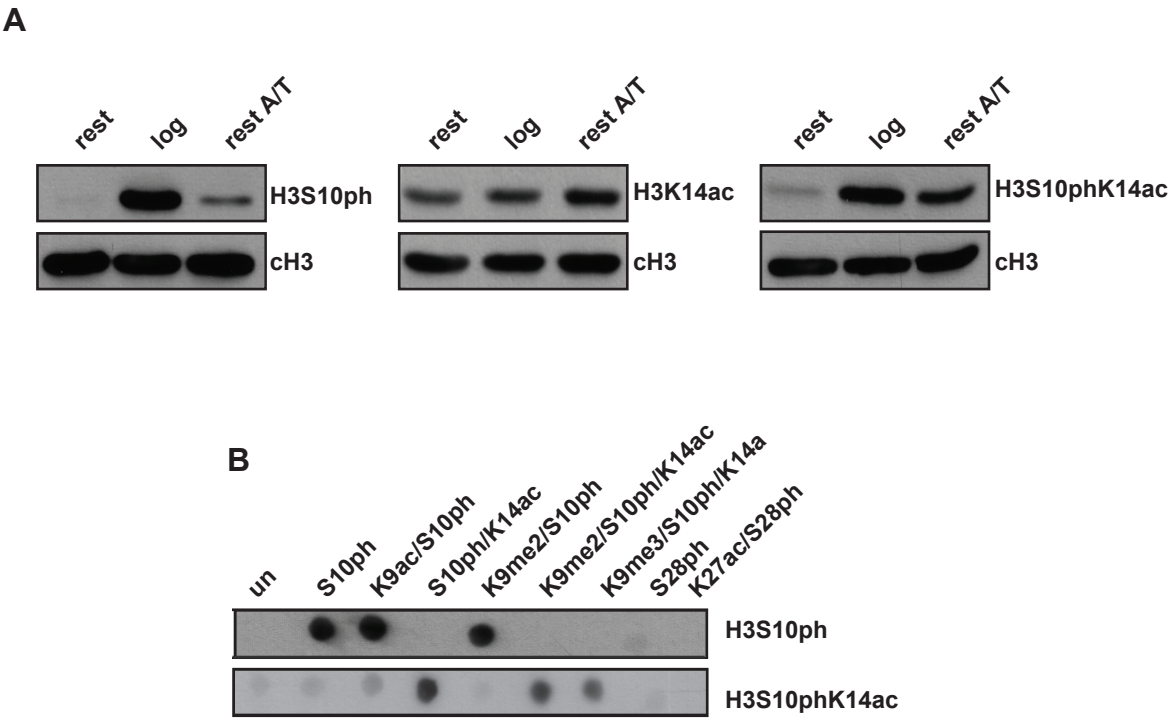
Supplementary Figure 1. (A) Histone H3 modifications in resting and proliferating Swiss 3T3 fibroblasts. Histones were extracted from serum-deprived (rest) or proliferating (log) Swiss 3T3 cells and as positive control from resting 3T3 cells treated with TSA and anisomycin for 3 hours (rest A/T). Histones were separated by SDS-PAGE and probed on immunoblots with antibodies specific for H3K14ac, H3S10ph, H3S10phK14ac and as loading control with an antibody specific for the C-terminus of histone H3. **(B)** Antibody test by Dot blot. Peptides resembling the N-terminus of histone H3, either unmodified (un) or bearing various single or multiple modifications such as acetylation (ac), phosphorylation (ph) and di- or tri-methylation (me2 or me3) were spotted on nitrocellulose membranes. Antibodies specific for the histone marks H3S10ph (upper panel) and H3S10phK14ac (lower panel) were tested for their epitope specificity.

Supplementary Figure 2. Effects of H89 on anisomycin dependent stimulation of p21 expression. Proliferating Swiss 3T3 mouse fibroblasts were treated for 3 hours with 188.5nM anisomycin or 165.5nM TSA or both together without or with pre-treatment with 10μM H89 for 15 minutes. Total RNA was extracted and reverse transcribed cDNA was used for p21 expression analysis by qRT PCR. Expression of p21 mRNA is shown relative to the GAPDH control.

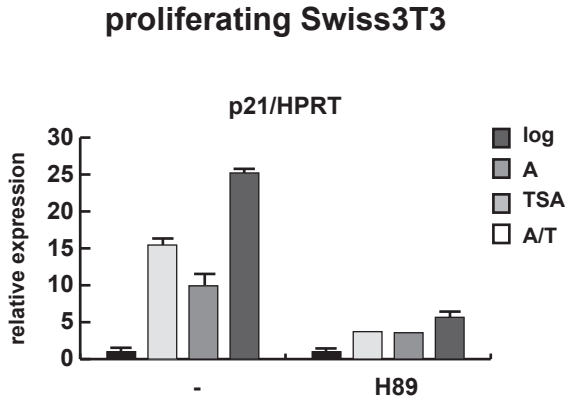
Supplementary Figure 3. siRNA mediated knockdown of MSK1, PP2A and 14-3-3 ζ in T98G cells. Proliferating T98G cells were transfected with control siRNA oligonucleotides and siRNA oligonucleotides specifically the target mRNA. **(A)** Cells transfected with MSK1 siRNA or a control siRNA were arrested by serum deprivation and were either left untreated (rest) or stimulated with 188.5nM anisomycin and 165.5nM TSA (A/T) for 1 hour. MSK1 expression was analyzed by qRT-PCR and is shown relative to the GAPDH control. **(B)** Cells transfected with MSK1 siRNA or a control siRNA were arrested by serum deprivation and were either left untreated (rest) or were stimulated with 188.5nM anisomycin (A) or 165.5nM TSA (T). Expression of MSK1 and p21 was analyzed by qRT-PCR and is shown relative to the GAPDH control. **(C)** Proliferating T98G cells transfected with siRNAs specific for the 2 isoforms of the catalytic subunit of PP2A (PP2Aa and PP2Ab) or a control siRNA were left untreated (rest) or were stimulated with 188.5nM anisomycin and 165.5nM TSA (A/T). Expression of PP2Aa and PP2Ab was analyzed by qRT-PCR and is shown relative to the GAPDH control. **(D)** Cells transfected with 14-3-3 ζ siRNA or a control siRNA were arrested by serum deprivation and were either left untreated (rest) or stimulated with 188.5nM anisomycin and 165.5nM TSA (A/T) for 1 hour. 14-3-3 ζ expression was analyzed by qRT-PCR and is shown relative to the GAPDH control.

Supplementary Figure 4. Role of MSK1 for 14-3-3 ζ recruitment to the p21 promoter. Proliferating T98G cells transfected with 14-3-3 ζ siRNA or a control siRNA were arrested by serum deprivation and were either left untreated (rest) or stimulated with 188.5nM anisomycin and 165.5nM TSA (A/T) for 3 hours. Chromatin was isolated and qRT-ChIP assays were performed with antibodies specific for 14-3-3 ζ. ChIP results are shown as percentage of input.

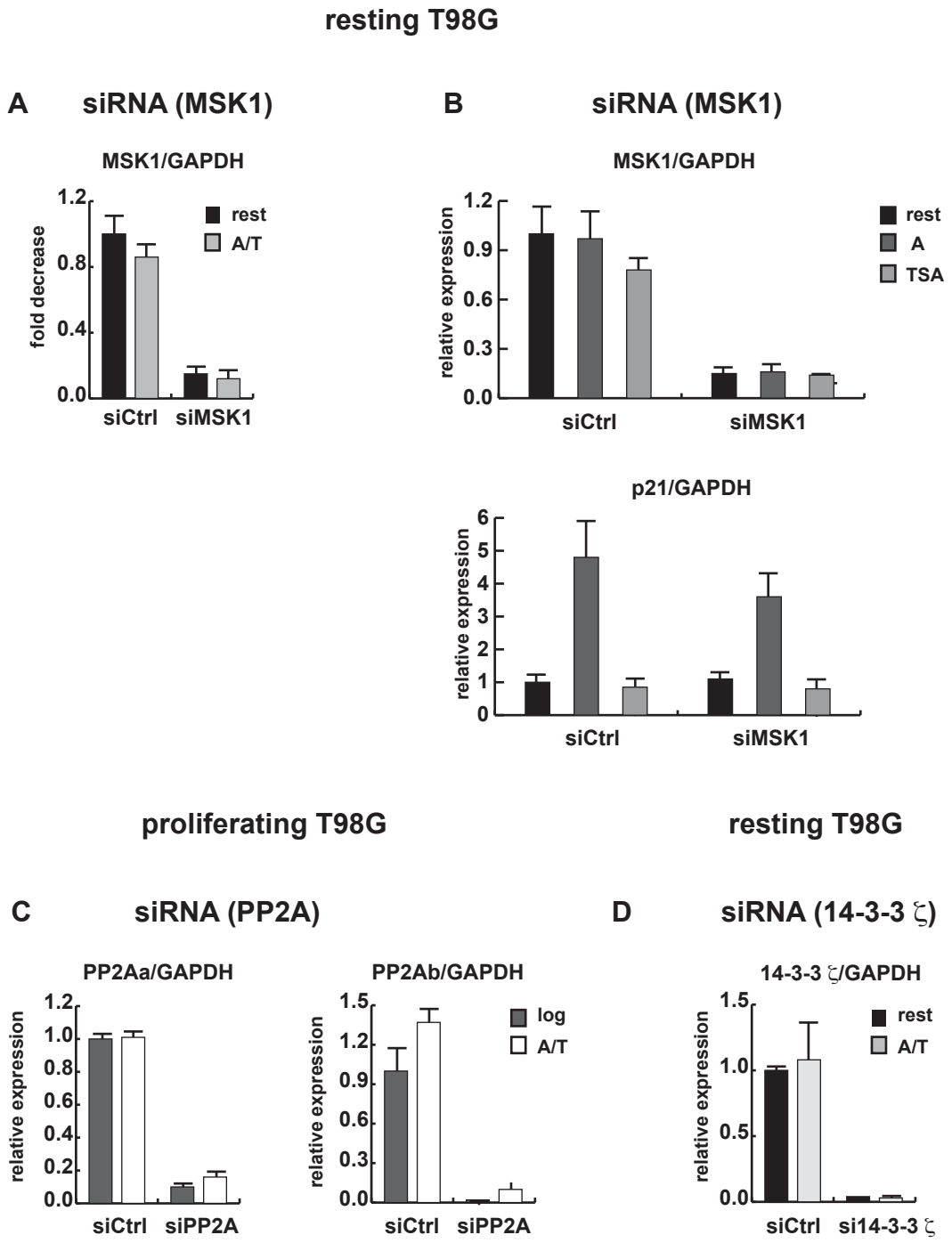
Simboeck et al. Sup. Fig. 1



Simboeck et al. Sup. Fig. 2



Simboeck et al. Sup. Fig. 3



Simboeck et al. Sup. Fig. 4