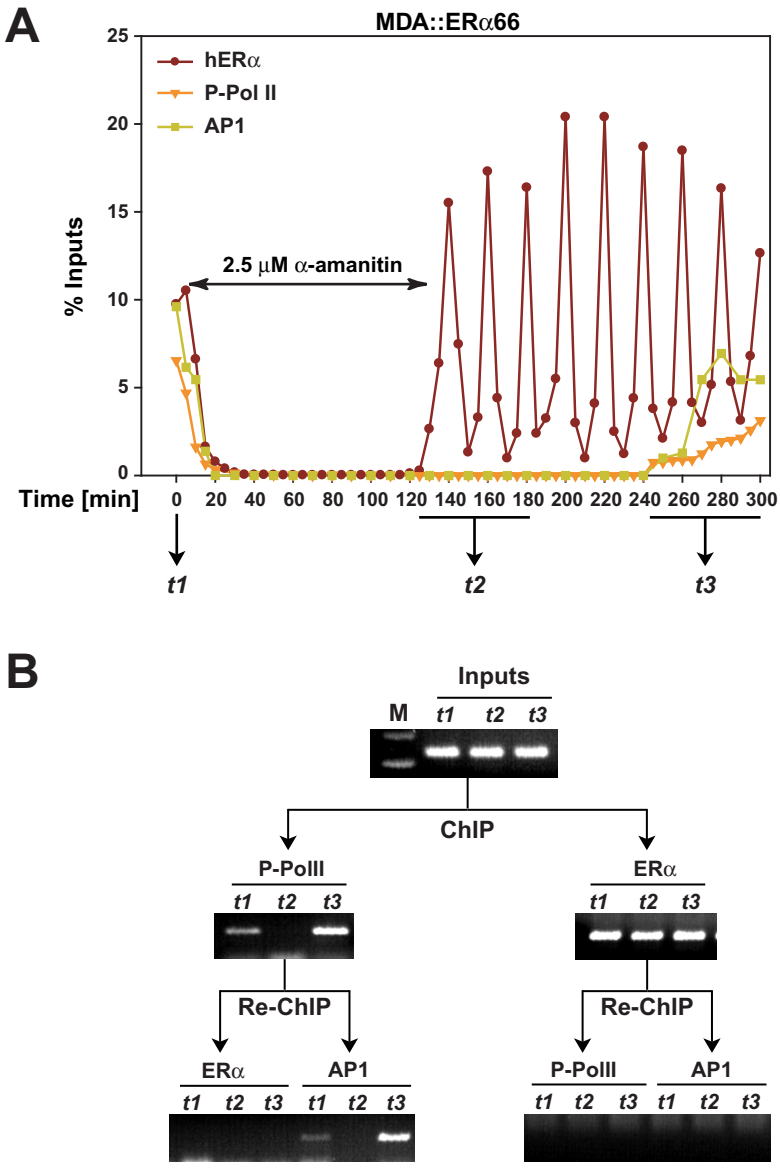

Supplementary Figure S1 Respective roles of ERα and AP1 in generating the basal activity of the pS2 gene. (**A**) After synchronization in G1 phase, MCF-7 cells were treated by 2.5 μM α-amanitin for 90 min. After washings to remove α-amanitin, cells were plated in E2-free media. Chromatin was then prepared from 2.10^6 cells sampled each 5 or 10 mn, and immunoprecipitated with antibodies specific for ERα [HC20 (#sc-543), Santa Cruz, Heidelberg, Germany], P-PolII [Clone CTD4H8 (#05-623), Upstate Biotechnology, Buckingham, England] or AP1 [H-79 (#sc-1694), Santa Cruz]. The presence of the pS2 promoter in ChIPped fractions was quantified by real-time PCR and normalized to inputs. (**B**) Re-ChIP experiments using pooled chromatin prepared at the time points indicated in the Panel **A**. Using the different antibodies in combinations demonstrate that apo-ERα is not associated with activated/phosphorylated Pol II on the pS2 promoter.

Altogether, these assays suggest that AP1, but not ERα, is responsible for the activation of the transcriptional machinery that generated the basal transcriptional activity of the pS2 gene.