Suppl. Fig. 1. Inhibitor-treatment does not alter Gal4-p65 expression or Gal4-DNA-binding capacity. L929sA cells, stably expressing Gal4-p65, were left untreated or treated with 2000 IU/ml TNF, either or not following a 2-h pretreatment with 10 μM SB203580 and/or 10 μM PD98059. Gal4-p65 expression was checked using an anti-p65 antibody. EMSA experiments were performed, using a specific gal4-probe, to determine DNA-binding capacity.
**Suppl. Fig. 2.** Characterization of a phospho-Ser276 p65 antibody. GST-p65\textsuperscript{12-317} was phosphorylated *in vitro* by either PKA or MSK1 or left untreated. Indicated amounts were spotted on a nitrocellulose paper. Western blot was performed with a phospho-Ser276-specific p65 antibody either or not in the presence of 10 µg/ml of competitor peptide.
Suppl. Fig. 3.
HEK293 cells were transfected as in Fig. 6B. L929sA cells were induced with 2000 IU/ml for 15 min, either or not in combination with 10 µM H89. Total cell lysates were subjected to SDS-PAGE and analyzed for Ser276 phosphorylation using a phospho-Ser276-specific p65 antibody.
Suppl. Fig. 4. MSK1 interacts with the N-terminal domain of p65 *in vitro.*

A. Schematic representation of p65, showing the different constructs. B. As estimated from the expression level shown in the lower panel, equal amounts of $^{35}$S-labeled proteins (p50 RHD, full-length p65 and the various p65 fragments) were incubated with GST, or with the fusion protein of either the MSK1 N-terminal (GST-MSK1-NT) or the C-terminal domain (GST-MSK1-CT) coupled to glutathione-Sepharose 4B beads. After incubation for 1 h at 4°C, the retained proteins were analyzed by SDS-PAGE, followed by PhosphorImager analysis.