Supplementary Information
for

The *PRKD1* promoter is a target of the KRas-NF-κB pathway in pancreatic cancer

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Figure S1, related to Figure 3

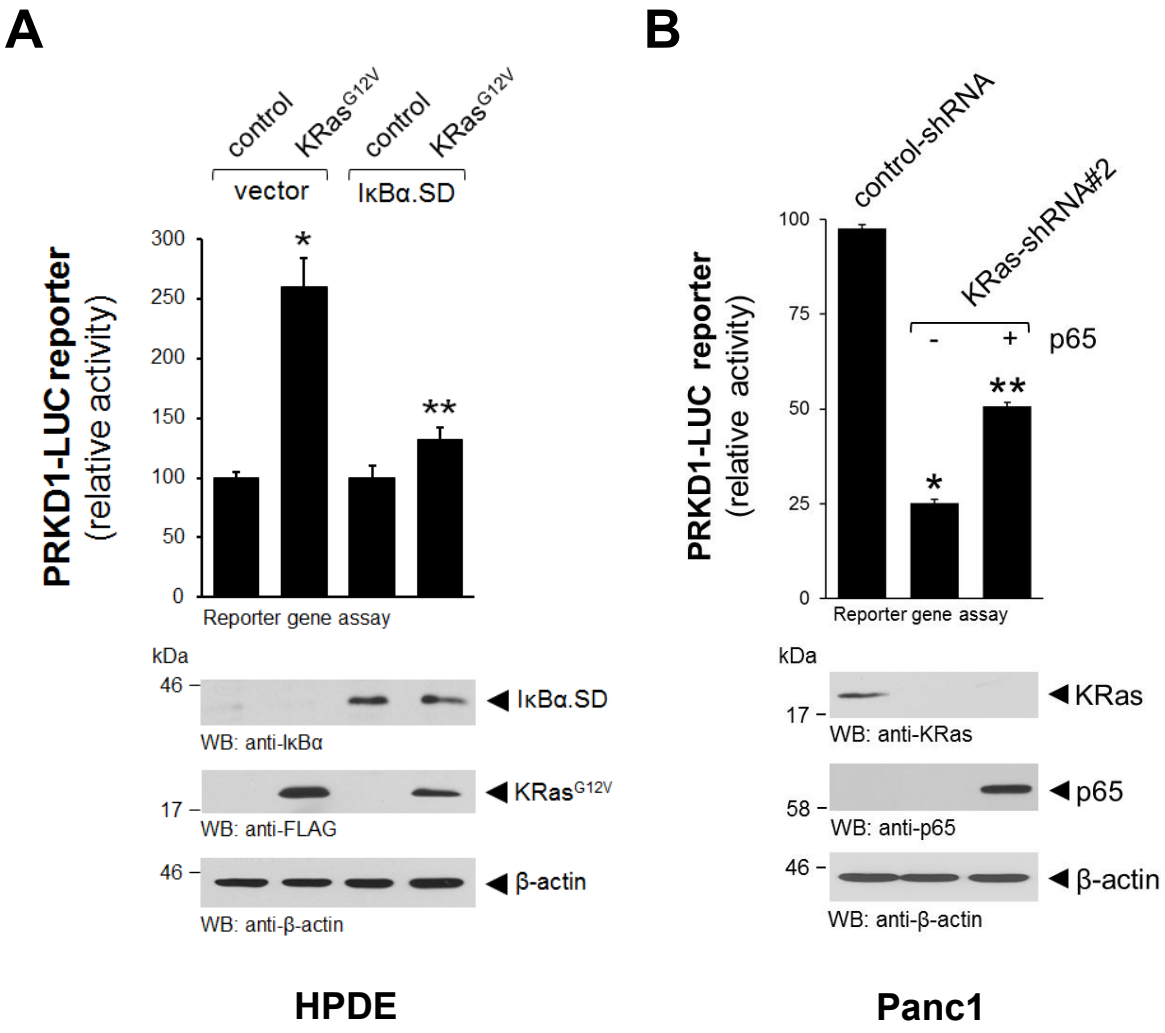
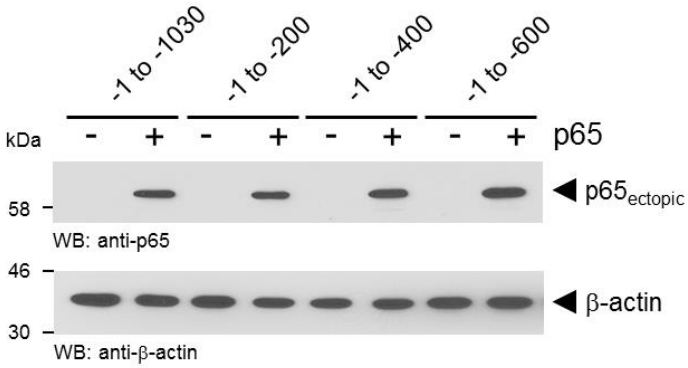


Figure S1, relates to Figure 3: **Oncogenic Kras induces *PRKD1* expression via the canonical NF-κB pathway. A:** HPDE cells were co-transfected with vector control, IκBα.SD or KRas^{G12V} and *PRKD1*-luciferase and renilla-luciferase reporters. 24 hours after transfection cells were lysed, and reporter gene assays performed. Probing lysates for IκBα (anti-IκBα), KRas^{G12V} (anti-FLAG) or β-actin (anti-β-actin) served as expression or loading controls. **B:** Panc1 cells were transfected with control-shRNA or shRNA targeting KRas, as well as vector control or p65 and *PRKD1*-luciferase and renilla-luciferase reporters, as indicated. 48 hours after transfection cells were lysed, and reporter gene assays performed. Probing lysates for KRas (anti-KRas), p65 (anti-p65) or β-actin (anti-β-actin) served as expression or loading controls.

Figure S2, related to Figure 4

A



B

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CTTCTAC
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Figure S2, relates to Figure 4: **Mapping of the NF-κB site in the *PRKD1* promoter region -1 to -1030. A:** Control blots (input control) for Fig. 4A. Cell lysates were analyzed by Western blot for overexpression of p65 (anti-p65), as well as for β-actin (anti-β-actin; loading control). **B:** Nucleotide sequence of the *PRKD1* promoter (-1 to -1030) fused to a luciferase reporter. Blue labeling indicates the fragment identified in Fig. 4A as relevant for p65-mediated activation of the *PRKD1* promoter. The potential reverse oriented NF-κB binding site within this fragment is indicated in red.