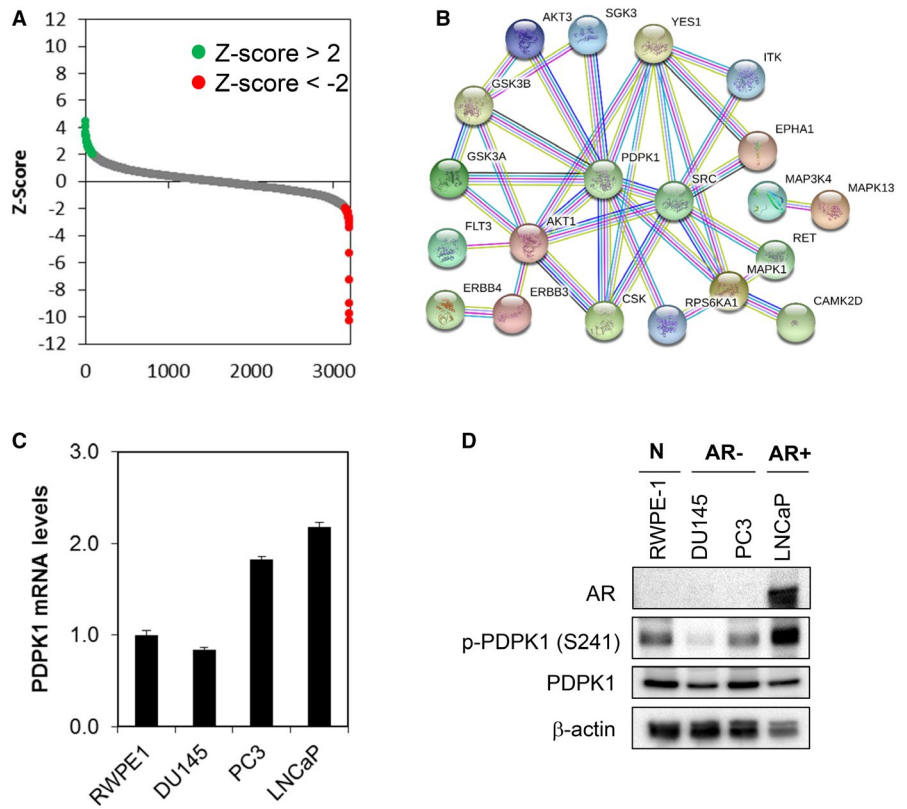


Erratum

In Nalaindran et al,¹ the published article contains errors in Figures 1 to 3. The correct figures are shown below. The authors confirm all results and conclusions of this article remain unchanged.

FIGURE 1 Kinome-wide shRNA library screen identifies PDPK1 as putative target regulating the survival of PCa cells. A, Kinase shRNA screen scatter plot. Z-scores are plotted on the y-axis against 3109 corresponding shRNAs on the x-axis. The red and green circled dots represent shRNA hits, which the former inhibited cell proliferation and the latter promoted cell proliferation. B, Protein-protein interaction network of the PDPK1 target proteins. C and D, PDPK1 is expressed in a subset of PCa cells and RWPE 1 non-transformed prostate epithelial cells. PDPK1 mRNA expression was evaluated by qPCR with GAPDH as housekeeping gene. The level of PDPK1 protein expression was detected by immunoblotting with β -actin as loading control



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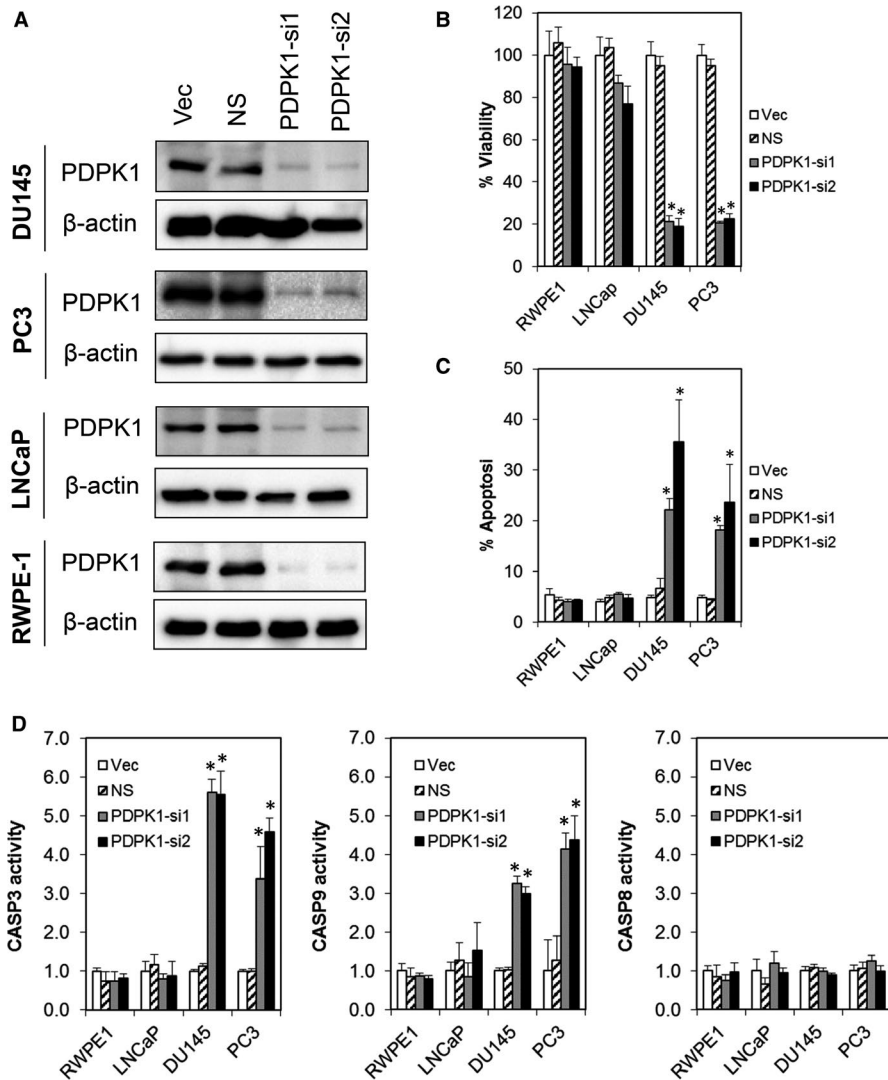
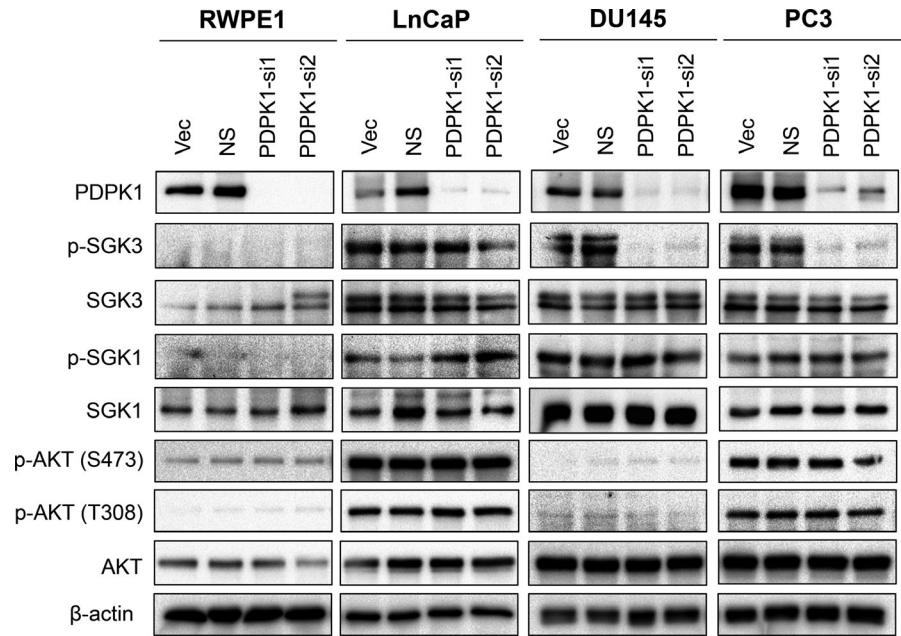


FIGURE 2 Depletion of endogenous PDPK1 induces tumour-specific cell death in PCa cells. A, Effective PDPK1 knock-down was achieved by two independent shRNA constructs targeting PDPK1 (PDPK1-si1 and PDPK1-si2). Lysates were harvested at 72 h post-lentiviral transduction and analysed by immunoblotting. B and C, PDPK1 depletion selectively inhibited the proliferation and induced apoptosis in AR-negative DU145 and PC3 PCa cells but not in AR-positive LNCaP or RWPE-1 non-transformed prostate epithelial cells. Cell viability and apoptosis were measured using CellTiter-Glo[®] assay and annexin V/7-AAD flow cytometry at 72 h post-transduction. D, Depletion of endogenous PDPK1 induced caspase 3 and 9 activities. Caspase 3, 8 and 9 activities were evaluated by CaspaseGlo assay at 72 h post-transduction. Bars represent means \pm SD of three independent experiments. (*) indicates statistical significance compared with NS control cells ($P < 0.01$, Student's *t* test)

FIGURE 3 Depletion of endogenous PDPK1 reduces SGK3 phosphorylation. PDPK1 depletion down-regulated SGK3 phosphorylation in AR-negative DU145 and PC3 cells, but not in AR-positive LNCaP PCa cells or RWPE-1 non-transformed prostate epithelial cells. The protein expression and phosphorylation of AKT, SGK1 and SGK3 Lysates were analysed by immunoblotting with β -actin and served as loading controls



REFERENCE

1. Nalairndran G, Hassan Abdul Razack A, Mai C-W, et al. Phosphoinositide-dependent Kinase-1 (PDPK1) regulates serum/glucocorticoid-regulated Kinase 3 (SGK3) for prostate cancer cell survival. *J Cell Mol Med.* 2020;24:12188–12198. <https://doi.org/10.1111/jcmm.15876>