

mTORC2/AKT/HSF1/HuR constitute a feed-forward loop regulating Rictor expression and tumor growth in glioblastoma

Holmes *et al.*

Supplementary Information

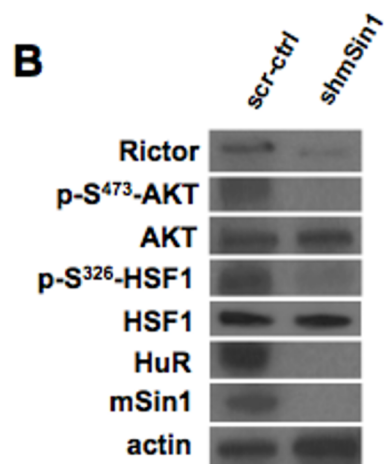
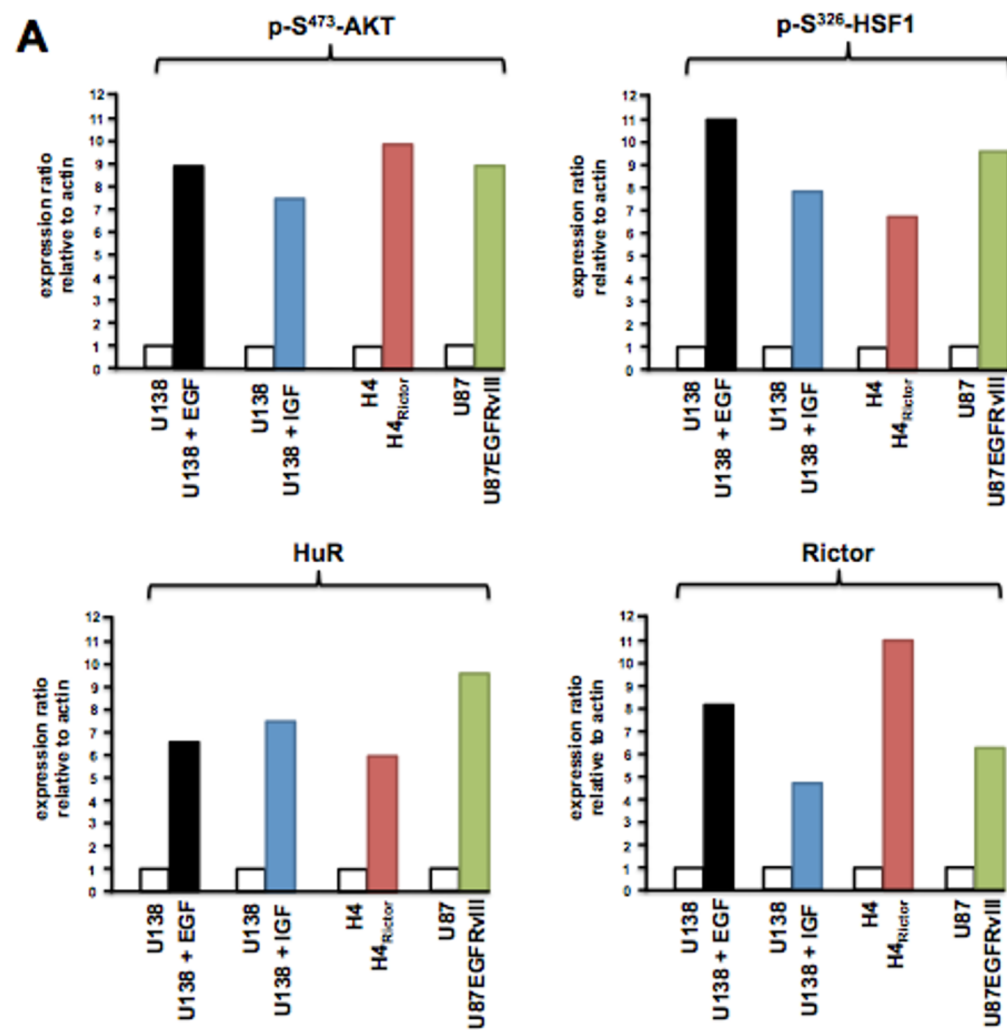
Supplemental figure legends

Figure S1. **A).** Expression levels of phospho-S⁴⁷³-AKT, phospho-S³²⁶-HSF1, HuR and Rictor from Figure 3A. Quantification of band intensities was determined by densitometry. **B).** mSin1 knockdown blocks EGF-induced AKT/HSF1/HuR/Rictor signaling. U138 cells stably expressing either a scr-crtl (scrambled sequence negative control) or mSin1-shRNA targeting construct, were treated with EGF (100 µg/ml, 8 h) and expression levels of the indicated proteins determined by immunoblotting.

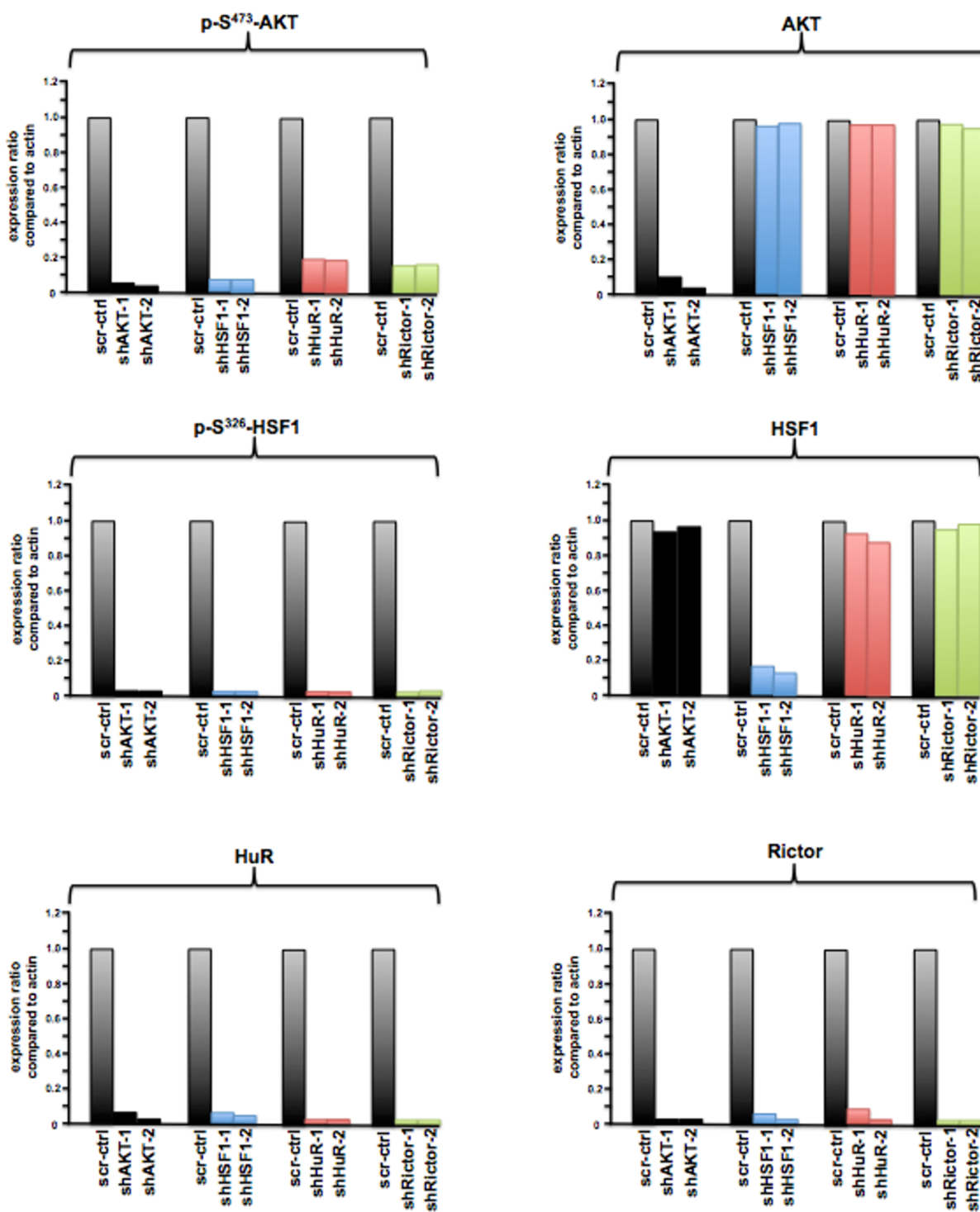
Figure S2. Quantification of expression levels of phospho-S⁴⁷³-AKT, total-AKT, phospho-S³²⁶-HSF1, total-HSF1, HuR and Rictor from Figure 4A.

Figure S3. Polysome distribution of Rictor and actin mRNAs from U138 cells transduced with empty vector or constitutively active HSF1 (HSF1-CA) and treated with siRNAs targeting AKT as indicated. Performed as in figure 1D. Mean and +S.D. are shown. *; $P < 0.05$.

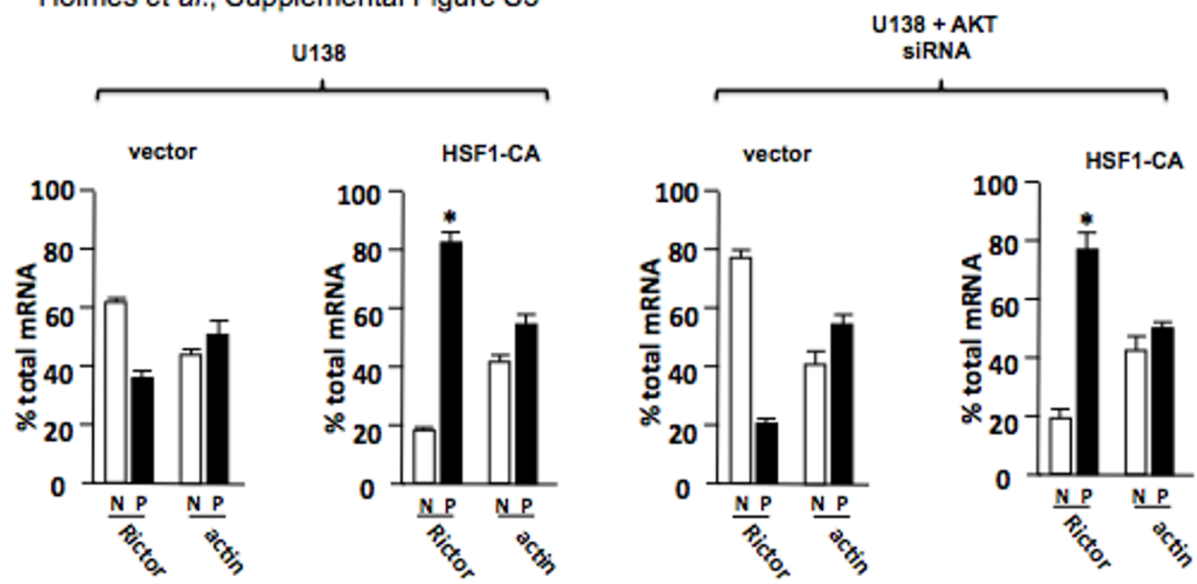
Figure S4. Graphic representations of correlations between the expression levels of the indicated proteins in 34 tumor samples. Levels are expressed as arbitrary units of optical density following quantification. Spearman coefficient r_s values are indicated.



Holmes *et al.*, Supplemental Figure S2



Holmes *et al.*, Supplemental Figure S3



Holmes *et al.*, Supplemental Figure S4

