Supplementary Figure S8

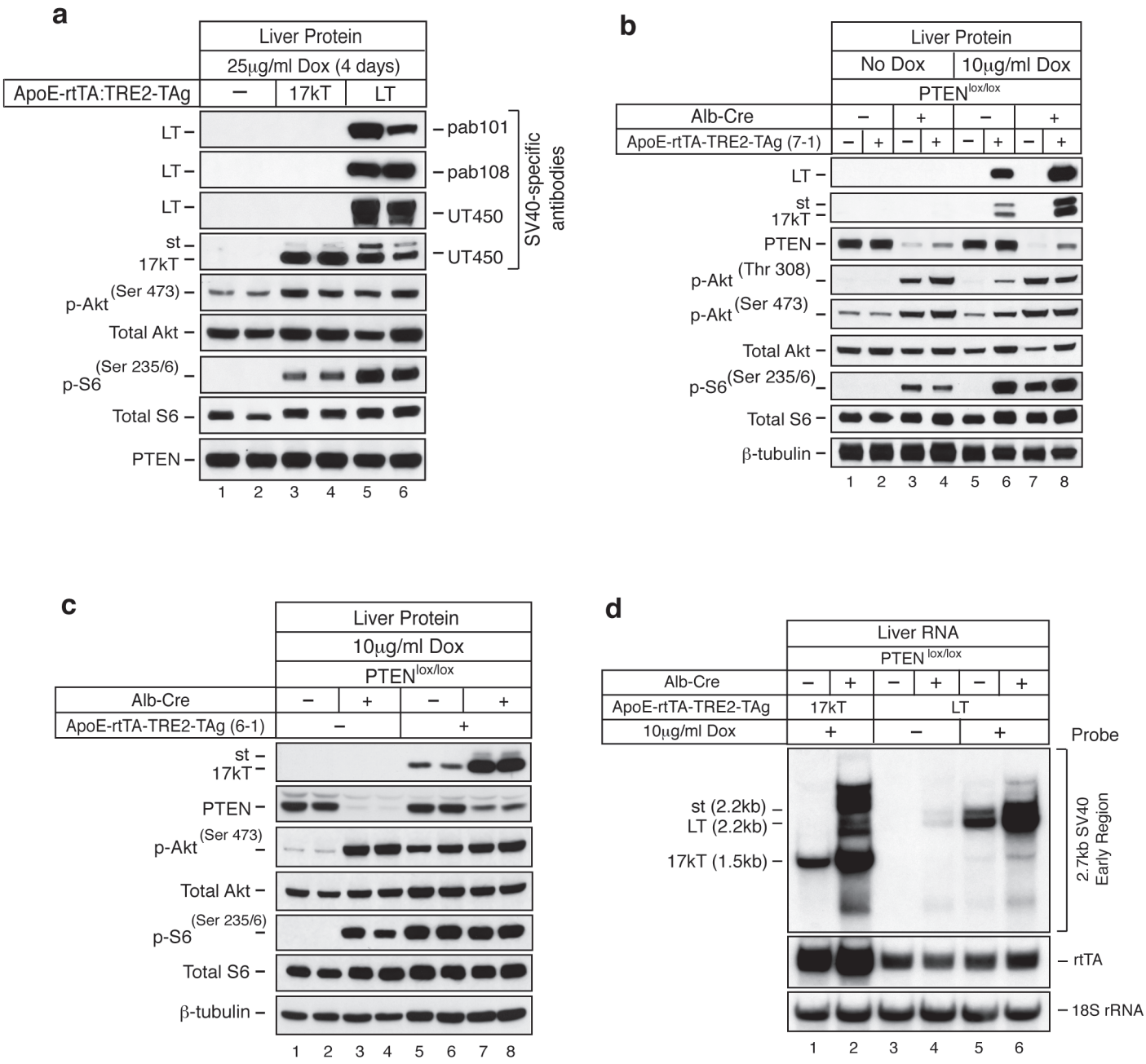

Figure S8. Acceleration of LT-dependent HCC and 17kT-induced hyperplasia in the absence of PTEN is due to enhanced translation and transcription of the SV40 oncoproteins rather than "super-activation" of PI3K or mTOR. **(a)** Immunoblot analysis showing activation of PI3K/Akt and mTOR/S6 signaling in livers expressing 17kT and LT. Note that while the magnitude of Akt phosphorylation is equivalent in livers of both transgenic lines, the magnitude of S6 phosphorylation is greater in livers expressing LT. Also note that 17kT and LT both stimulate signaling without altering expression of Akt, S6 or PTEN. **(b, c)** Immunoblot analysis of viral oncoprotein expression and PI3K and mTOR signaling in liver lysates from 7-1 (b) and 6-1 (c) transgenic mice with and without PTEN. Note that expression of the viral oncoproteins is increased in livers of both lines in the absence of PTEN, but that the viral oncoproteins and loss of PTEN fail to synergize to "super-phosphorylate" Akt or S6. **(d)** Northern blot analysis of SV40 transcript abundance in LT- and 17kT-expressing livers in the presence or absence of PTEN. Note that the abundance of viral transcripts is increased in the absence of PTEN in 6-1 mice treated with dox (compare lanes 1 and 2) and in livers of 7-1 mice in the absence (lanes 3 and 4) and presence (lanes 5 and 6) of dox. In both lines, PTEN-deficiency has little to no effect on rtTA transcript abundance.