SUPPLEMENTAL DATA

Rapamycin and Glucose-TOR Signaling in Plants

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Supplementary Table 1. Primers used for constructs.

Name	**Forward primer**	**Reverse primer**
S6K1	ATGGTTTCCTCTCAGCGTCC	CAAAGTAGTTGTGGACTGGTGA
S6K2	ATGGTTTCTTCTCAGTGTTCTGTTG	CAAGTTGGATGTGGTCCGAT
FKP12	ATGGGAGTGCAGGTGGAA	TTCCAGTTTTAGAAGCTCCACAT
FKP15.1	ATGATGAGCTCTGCATCCG	AAGCTCATTCTTTGATTTCGCT
FKB15.2	ATGGCGAGCAAGATGAGTCT	TAGCTCGTCATTTCCATATCCC
FKP20.1	ATGGGTGATGCAATCGATTT	TTTAGCTTTGCCTTTGCCTTT
FKP42	ATGGATGAATCTCTGGAGCATC	ATCTGCTTTAACTCTGTGGCG
FKP62	ATGGATGCTAATTTCGAGATGC	AGACCTCAGGTGCTCATTGC
FKP65	ATGGAAGACGATTTCGACACG	TGCCTTGGTGTCAATACTCATC
FKP72	ATGGCGGTAGGCGATCAG	TGTAAATTTGGCGCTCACAA

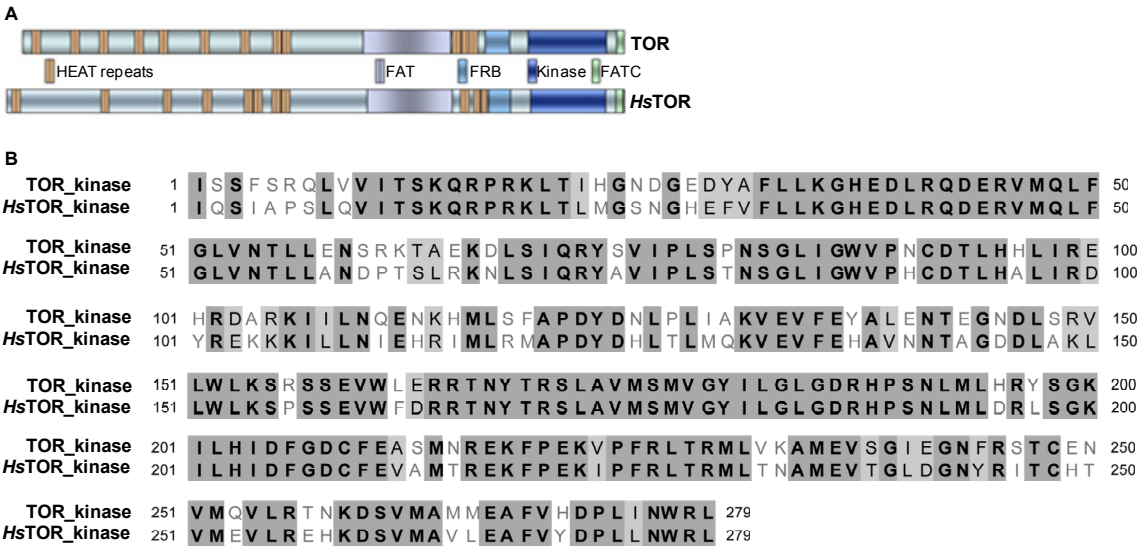

Figure S1. **TOR PKs are highly conserved in human and *Arabidopsis*.** A, Domain organization of human and *Arabidopsis* TOR PKs. HEAT repeats: Huntingtin, Elongation factor 3, subunit of protein phosphatase 2A and TOR1; FAT: FRAP, ATM and TRRAP domain; FRB: FKP12-rapamycin binding domain. FATC: Carboxy-terminal FAT domain. B, Alignment of the catalytic kinase domain of human and *Arabidopsis* TOR proteins.

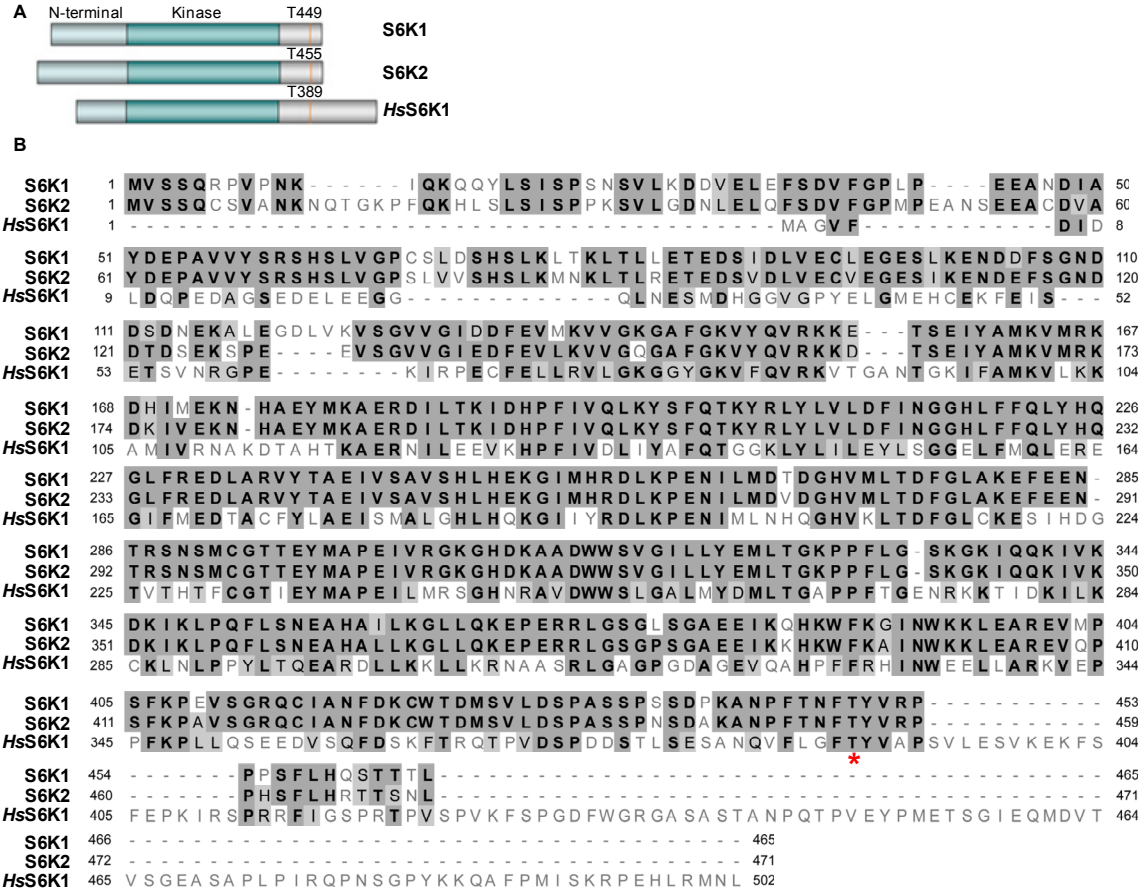

Figure S2. S6Ks are highly conserved in human and *Arabidopsis*. A, Domain organization of human and *Arabidopsis* S6 kinases. B, Alignment of human and *Arabidopsis* S6 kinases. Asterisk indicates the conserved TOR phosphorylation sites.

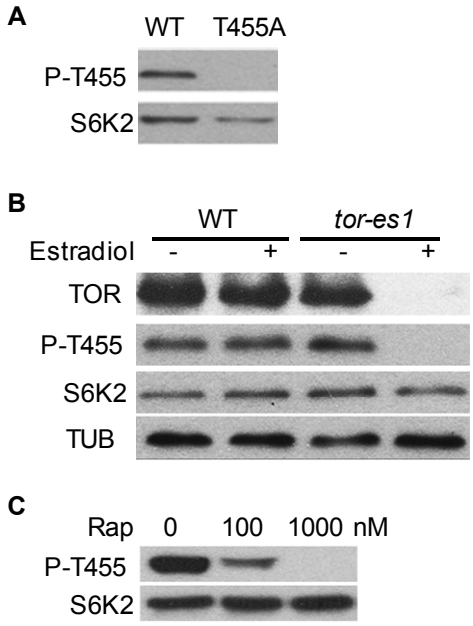

Figure S3. T455 phosphorylation of S6K2. A, T455 phosphorylation specificity of S6K2. FLAG-tagged WT or mutant S6K2 (T455A) was transiently expressed in protoplasts. Total proteins were analyzed by protein blot analysis using anti-p-T389 (P-T455) or anti-FLAG (S6K2) antibody. B, T455 phosphorylation is abolished in the *tor* mutant. WT or conditional *tor* mutant protoplasts expressing FLAG-tagged S6K2 were treated without or with estradiol (10 μM). Total proteins were analyzed by protein blot analysis probed with anti-TOR (TOR), anti-p-T389 (P-T455) or anti-FLAG (S6K2) antibody. Tubulin (TUB) was used as a loading control. C, T455 phosphorylation of S6K2 is inhibited by rapamycin (Rap).

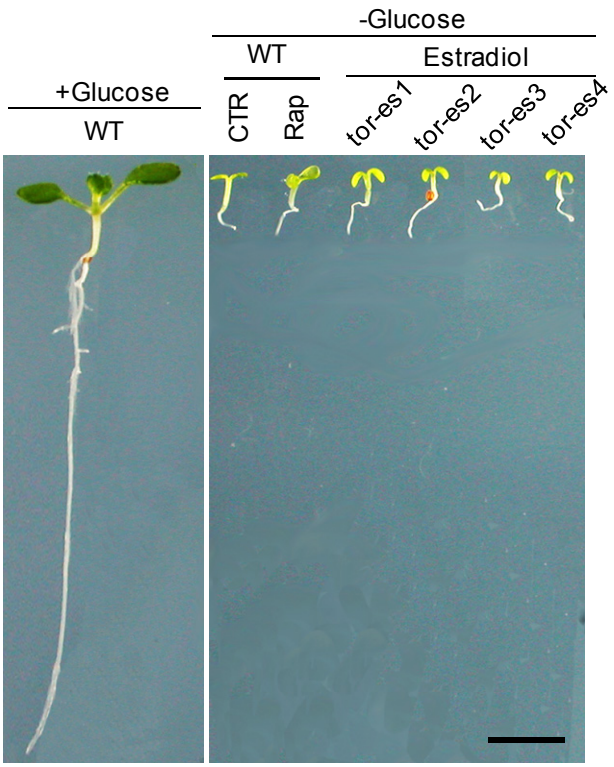

Figure S4. Growth of *Arabidopsis* seedlings relies on glucose. WT and estradiol-inducible *tor* mutants were grown in liquid MS medium without any sugar or with 30 mM glucose for 9 days. Scale bar: 5 mm.

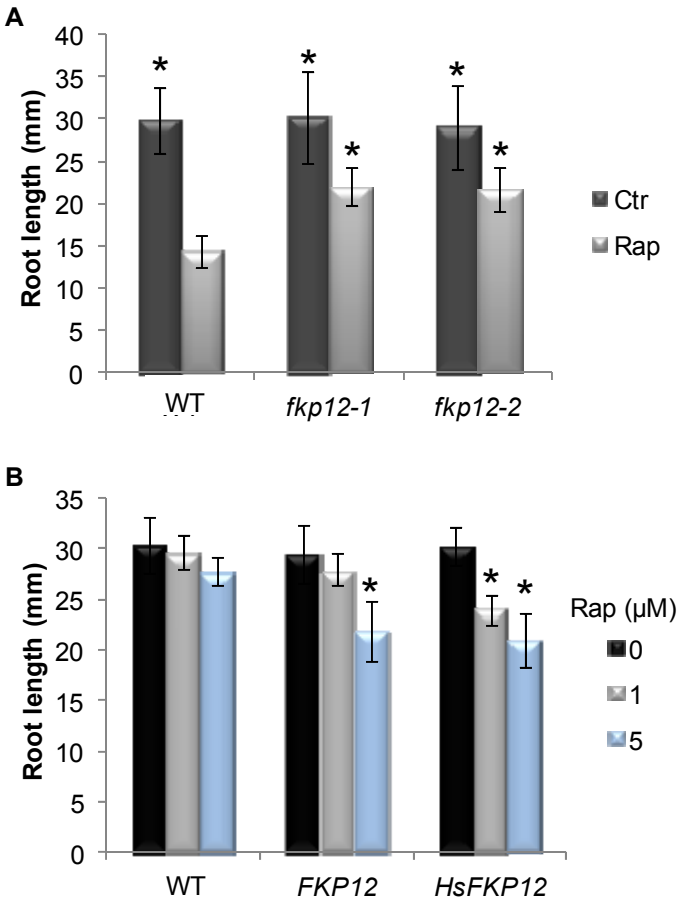

Figure S5. FKP12 is required for inhibition of seedlings growth by rapamycin. A, The inhibition effect of rapamycin on seedlings growth is relieved in *fkp12* mutants. The average root length was measured after 9 days of rapamycin (Rap, 10 μM) treatment. Results represent means ± SD (n=3). * indicates a significant difference with $p<0.05$ when compared with data from wild type (WT) treated with rapamycin, based on the results of an unpaired Student's t test. B, Overexpression of *Arabidopsis* or human FKP12 enhances the rapamycin inhibition on seedlings growth. The average root length was measured after 9 days of rapamycin (Rap) treatment. Results represent means ± SD (n=3). * indicates a significant difference with $p<0.05$ when compared with data from wild type (WT) untreated with rapamycin, based on the results of an unpaired Student's t test.

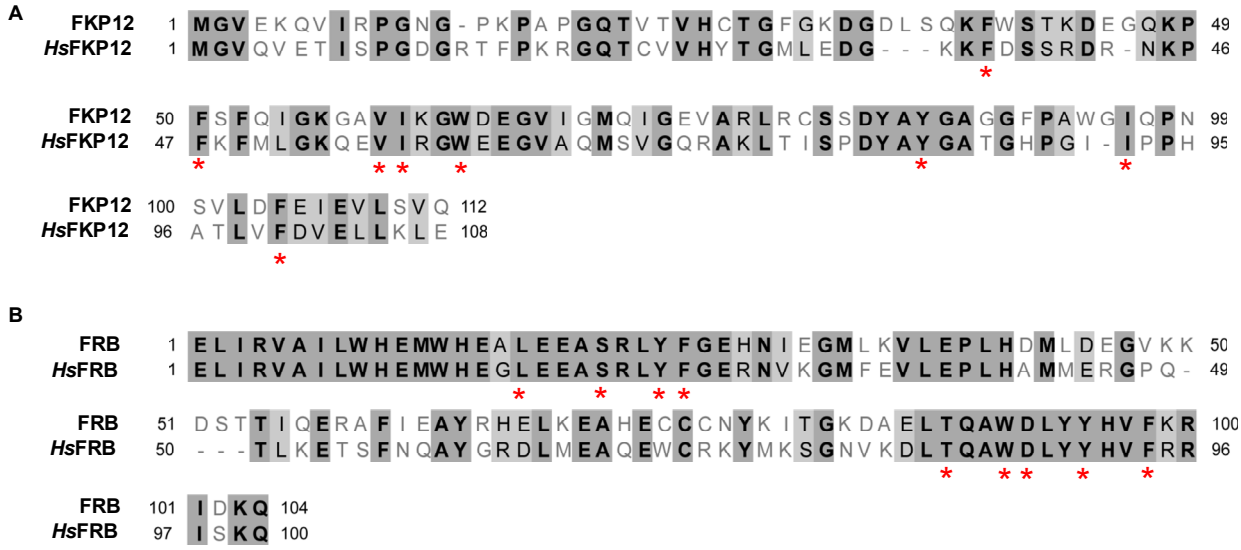

Figure. S6. FKP12s and TOR-FRB domains are highly conserved in human and *Arabidopsis*. Alignment of FKP12 proteins (A) and TOR-FRB domain (B) between human and *Arabidopsis*. Asterisks indicate conserved residues of human FKP12 and FRB that interact with rapamycin (1).

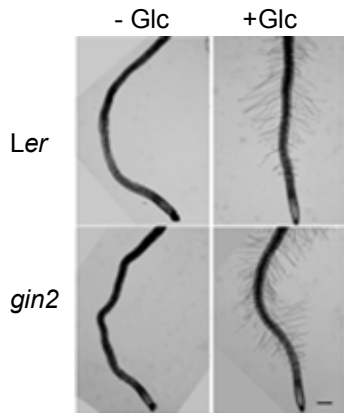

Figure S7. Root hairs are not affected in the *gin2* mutant. L*er* and *gin2* were germinated with or without glucose (30 mM) for 4 days. Scale bar: 200 μm.

REFERENCES

1. Choi, J., Chen, J., Schreiber, S.L., and Clardy, J. (1996) *Science* 273, 239-242