Rapamycin-sensitive phosphorylation of target proteins by the mTORC1 complex controls T-cell growth, survival and proliferation, which largely accounts for the extensive application of rapamycin and its analogues in post-transplantation immunosuppression. Although the best-characterised of these downstream targets control translation, several other candidate targets can influence proliferation, so that the effects of rapamycin on T-cell proliferation could be mediated through several different pathways. The non-protein-coding gene GAS5 (Growth Arrest Specific transcript 5) is both necessary and sufficient for normal growth arrest in both untransformed and leukaemic T-cells. In addition, most of the inhibition of T-cell proliferation produced by rapamycin is mediated through GAS5. GAS5 transcripts accumulate on growth arrest in stationary phase and after rapamycin treatment, and this accumulation provides a functional link between the control of translation and the control of proliferation and survival. The GAS5 gene does not encode a functional protein and its effects may be mediated by novel and specific direct interactions with steroid receptors and/or by the snoRNAs (Small Nucleolar RNAs) encoded in its introns.