Loss of p53 and the Rb family is frequently found together with activating Ras mutations in human cancers. Using primary cells, we have investigated how these signals influence cell growth (cell size and mass). We saw that constitutively active Ras can drive growth in the absence of extrinsic factors via the canonical growth pathway, PI3K/AKT/TOR. However, we found that oncogenic Ras stimulates growth relatively poorly. We found that Rb, a key regulator of cell cycle progression, has an additional role in controlling cell growth. While loss of p53 is not capable of promoting cell growth, loss of Rb is sufficient to drive cell growth in the absence of growth factor signalling. Additionally, we showed that effects of the Rb family on growth pathways and the cell cycle can be uncoupled. Interestingly, growth driven by Rb loss does not appear to be the result of PI3K/AKT/TOR signalling, but seems to be via a distinct pathway - acting together with Ras to drive strong sustained growth.