Insulin activates the nutrient sensitive complex mTOR complex 1 (mTORC1), which requires binding of Rheb-GTP to mTOR. Activated mTORC1 phosphorylates the substrates S6K, 4EBP1 and PRAS40, but only when bound to raptor. PRAS40 is preferentially bound to raptor when mTORC1 is unstimulated, however insulin increases the ability of mTORC1 to bind all three substrates, which then exhibit cross-competition for raptor. We find that multiple factors underlie insulin upregulation of mTORC1 substrate binding. First, removal of mTOR from mTORC1 greatly enhances 4E-BP binding to raptor with no further increase by insulin. Second, the catalytic activity of mTORC1 is required to enhance substrate binding. Excess Rheb-GTP, despite activating signalling, does not increase substrate binding to mTORC1, whereas catalytic site inhibitors of mTORC1 prevent increased 4E-BP1 binding to mTORC1, by inhibiting PRAS40 release. Third, activation of Akt is required, independently of Akt action on TSC; in the presence of excess Rheb-GTP, inhibition of Akt nevertheless greatly reduces 4E-BP1 binding without altering S6K phosphorylation. Some but not all of Akt stimulated 4E-BP1 binding is mediated by PRAS40 release; although depletion of PRAS40 increases 4E-BP1 binding to unstimulated mTORC1, this increase is eliminated by inhibition of Akt, acting at a site to be determined.