mTOR is a central regulator of cell growth, proliferation, survival and metabolism. mTOR inhibition is increasingly used in oncology and is a promising therapeutic approach in cardiology. mTOR was recently shown to regulate stress response in adult myocardium primarily via the mTORC1 substrate, 4E-BP1. However, mTOR functions during early postnatal cardiac development remain to be elucidated. Here we showed that mTOR inactivation in early postnatal mouse myocardium induced a lethal dilated cardiomyopathy caused by defects in cardiomyocyte growth and survival associated with subsequent fibrosis. In contrast to adult myocardium, we demonstrated a functional role for both mTOR complexes in juvenile heart as mTORC1 and mTORC2 activities are impaired, as shown by hypophosphorylation of the translation inhibitor 4E-BP1 and of the cardioprotective kinase AKT at Ser 473, thereby leading to an accumulation of its pro-apoptotic targets p53 and Bax. Altogether, our results demonstrate that mTOR is a key regulator of cardiomyocyte growth and viability in early postnatal myocardium through 4E-BP1 and AKT. Our findings also suggest potential cardiotoxicity of new generation of mTOR catalytic-site inhibitors developed for antineoplastic therapies.