Ageing is the major risk factor for numerous killer diseases including cardiovascular disease, dementia and cancer. Importantly, mutations in single genes that can extend lifespan in laboratory animals, do so by keeping them healthy and youthful for longer. The best understood interventions capable of extending healthy lifespan are dietary restriction, and reductions in the target-of-rapamycin (TOR) and insulin/IGF signaling (IIS) pathways. Because our knowledge about the mechanisms of ageing stem from genetic studies, we therefore applied a novel pharmacological approach employing rapamycin, a well-studied biochemical inhibitor of the TOR pathway. We have shown that rapamycin robustly extends lifespan in *Drosophila*. Examination of the underlying mechanisms revealed that the TORC1 branch of the TOR pathway was accountable for the observed longevity effects. More precisely, lifespan extension was associated with up-regulation of autophagy and down-regulation of translation. Furthermore, lifespan extension by rapamycin was independent of the FOXO transcription factor, the major anti-ageing down-stream effector of IIS signaling. This suggests that IIS and rapamycin act independently in mediating longevity. Rapamycin also further extended the longevity of flies with a lifespan already maximized by DR, indicating additive effects. In conclusion, it seems that autophagy together with down-regulation of translation, act as a rejuvenating process underlying lifespan extension by rapamycin. It is currently unclear if autophagy and translation act in concert or separately in this anti-ageing intervention.