Pancreatic β-cells express insulin receptors (Insr) at higher levels than most other cell types, but the consequences of β-cell insulin resistance remain enigmatic. Here, we used the Ins1Cre allele to delete Insr specifically in β-cells, but not in the brain. Both female and male knockout Insrf/f;Ins1cre/wt;ntng+/− and heterozygous Insrf/ft;Ins1cre/ft;ntng+/− mice were compared to Ins1Cre− littermate controls to define the roles of Insr, and β-cell insulin resistance. Action potential and calcium oscillation frequencies were increased in single Insr knockout β-cells from female, but not male mice. Consistent with this, isolated islets from female knockout Insrf/f;Ins1cre/ft;ntng+/− and heterozygous Insrf/ft;Ins1cre/ft;ntng+/− exhibited significantly elevated insulin release to high glucose in perfusion experiments compared with Insrwt/ft;Ins1cre/ft;ntng+/− controls. Similarly, insulin secretion measured in vivo following i.p. glucose challenge and during a 350 mg/dl hyperglycemic clamp was consistently higher in female lacking β-cell Insr, but not male mice. Glucose tolerance was significantly improved in female Insrf/f;Ins1cre/ft;ntng+/− and Insrf/ft;Ins1cre/ft;ntng+/− mice when compared to controls at 9, and 39 weeks. There were no significant differences between groups of male mice, or between groups of high fat-fed mice, suggesting the possibility that global insulin resistance obscuring these effects. No differences in insulin tolerance were observed. RNAseq on FACS-purified, recombinant β-cells revealed significant differences NFκB signaling and Ras signaling, processes we and others have previously implicated in β-cell proliferation. However, deletion of Insr did not alter β-cell mass up to 9 months of age, nor did it impair 4 day hyperglycemia-induced proliferation, suggesting possible redundancy with Igf1r. Collectively, our data suggest that loss of β-cell Insr alone is sufficient to drive post-prandial hyperinsulinemia, thereby improving glucose homeostasis in otherwise insulin sensitivity mice. These findings are consistent with the concept that glucose stimulated insulin release normally inhibits its own secretion during hyperglycemic conditions and point to the Insr as a modulator of insulin secretion.