In recent studies, we have found that genetic or pharmacologic inhibition of the EGF receptor not only ameliorated the development of diabetic nephropathy in mouse models of type II diabetes but also preserved pancreatic islet function and insulin sensitivity. In contrast, mice with deletion of another member of this receptor family, ErbB4, led to development of obesity, dyslipidemia, hepatic steatosis and glucose intolerance, and NRG4, an ErbB4 ligand, inhibited lipogenesis and promoted adipocyte browning.