Proto-oncogene c-KIT is the gene encoding the receptor tyrosine kinase protein known as tyrosine-protein kinase KIT, CD117 (cluster of differentiation 117) or mast/stem cell growth factor receptor (SCFR).

Multiple transcript variants encoding different isoforms have been found for this gene.

KIT was first described by the German biochemist Axel Ullrich in 1987 as the cellular homolog of the feline sarcoma viral oncogene v-kit.

Kit is a cytokine receptor that belongs to the type III receptor tyrosine kinase family. It is structurally similar to platelet-derived growth factor recpetors (PDGFRs), colony stimulating factor-1 receptor and fms-like tyrosine kinase. Kit signaling is plays important role in a number of physiological processes including erythropoiesis, lymphopoiesis, mast cell development and function, megakaryopoiesis, gametogenesis and melanogenesis. Sequence alterations in the c-kit gene are found to be associated with different cancers including hematopoietic malignancies, gastrointestinal stromal tumors, germ cell tumors, small-cell lung cancer and pancreatic cancer. The primary ligand for kit receptor is stem cell factor (SCF). It is also known as Kit ligand, steel factor or mast cell growth factor. SCF is a glycosylated, non-covalent homodimer. Alternative splicing and proteolytic cleavage results in soluble and membrane bound forms of the protein. that binds to two KIT monomers. Binding of SCF to KIT results in the dimerization of the receptor and its autophsphorylation. The residues that are known to get phosphorylated upon ligand binding include Tyr568, Tyr570, Tyr703, Tyr721, Tyr730, Tyr823, Tyr 900 and Tyr936. Signaling events downstream of the KIT receptor are well studied. Among the signaling cascades that are activated are the Ras/Raf/MEK/MAPK and the PI3K/AKT/RPS6K pathways. KIT stimulation is also known to activate the JAK/STAT and PLC/PKC signaling pathways. Among the other key proteins that are regulated by KIT are the kinases BTK, TEC, LYN, SRC, FYN and JNK. Regulation of KIT receptor tyrosine kinase occurs through many mechanisms. Activated KIT receptors are degraded via CBL, a E3 ubiquitin-protein ligase. CBL induces the degradation of the receptor via the proteasome or lysosome. KIT can also be dephosphorylated and inactivated by the protein tyrosine phosphatase Shp1. Also, activation of protein kinase C results in a negative feedback loop, wherein it phosphorylates specific serine residues leading to the inactivation of KIT.