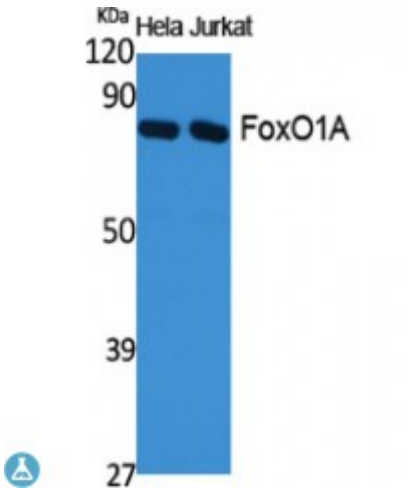
Anti-FoxO1A antibody



Description	Rabbit polyclonal to FoxO1A.

Model	STJ93126
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IF, IHC, WB
Immunogen	Synthesized peptide derived from human FoxO1A around the non-phosphorylation site of S329.
Immunogen Region	270-350 aa
Gene ID	2308
Gene Symbol	FOXO1
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IF 1:200-1:1000ELISA 1:10000
Specificity	FoxO1A Polyclonal Antibody detects endogenous levels of FoxO1A protein.
Tissue Specificity	Ubiquitous.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	Forkhead box protein O1 Forkhead box protein O1A Forkhead in rhabdomyosarcoma
Molecular Weight	78 kDa
Clonality	Polyclonal

Conjugation	Unconjugated
Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:3819OMIM:136533
Alternative Names	Forkhead box protein O1 Forkhead box protein O1A Forkhead in rhabdomyosarcoma
Function	Transcription factor that is the main target of insulin signaling and regulates metabolic homeostasis in response to oxidative stress. Binds to the insulin response element (IRE) with consensus sequence 5'-TT[G/A]TTTTG-3' and the related Daf-16 family binding element (DBE) with consensus sequence 5'-TT[G/A]TTTAC-3'. Activity suppressed by insulin. Main regulator of redox balance and osteoblast numbers and controls bone mass. Orchestrates the endocrine function of the skeleton in regulating glucose metabolism. Acts synergistically with ATF4 to suppress osteocalcin/BGLAP activity, increasing glucose levels and triggering glucose intolerance and insulin insensitivity. Also suppresses the transcriptional activity of RUNX2, an upstream activator of osteocalcin/BGLAP. In hepatocytes, promotes gluconeogenesis by acting together with PPARGC1A and CEBPA to activate the expression of genes such as IGFBP1, G6PC and PCK1. Important regulator of cell death acting downstream of CDK1, PKB/AKT1 and SKT4/MST1. Promotes neural cell death. Mediates insulin action on adipose tissue. Regulates the expression of adipogenic genes such as PPARG during preadipocyte differentiation and, adipocyte size and adipose tissue-specific gene expression in response to excessive calorie intake. Regulates the transcriptional activity of GADD45A and repair of nitric oxide-damaged DNA in beta-cells. Required for the autophagic cell death induction in response to starvation or oxidative stress in a transcription-independent manner.
Cellular Localization	Cytoplasm Nucleus. Shuttles between the cytoplasm and nucleus. Largely nuclear in unstimulated cells. In osteoblasts, colocalizes with ATF4 and RUNX2 in the nucleus . Insulin-induced phosphorylation at Ser-256 by PKB/AKT1 leads, via stimulation of Thr-24 phosphorylation, to binding of 14-3-3 proteins and nuclear export to the cytoplasm where it is degraded by the ubiquitin-proteosomal pathway. Phosphorylation at Ser-249 by CDK1 disrupts binding of 14-3-3 proteins and promotes nuclear accumulation. Phosphorylation by NLK results in nuclear export. Translocates to the nucleus upon oxidative stress-induced phosphorylation at Ser-212 by STK4/MST1. SGK1-mediated phosphorylation also results in nuclear translocation. Retained in the nucleus under stress stimuli including oxidative stress, nutrient deprivation or nitric oxide. Retained in the nucleus on methylation.
Post-translational Modifications	Phosphorylation by NLK promotes nuclear export and inhibits the transcriptional activity. In response to growth factors, phosphorylation on Thr-24, Ser-256 and Ser-322 by PKB/AKT1 promotes nuclear export and inactivation of transactivational activity. Phosphorylation on Thr-24 is required for binding 14-3-3 proteins. Phosphorylation of Ser-256 decreases DNA-binding activity and promotes the phosphorylation of Thr-24 and Ser-319, permitting phosphorylation of Ser-322 and Ser-325, probably by

CDK1, leading to nuclear exclusion and loss of function. Stress signals, such as response to oxygen or nitric oxide, attenuate the PKB/AKT1-mediated phosphorylation leading to nuclear retention. Phosphorylation of Ser-329 is independent of IGF1 and leads to reduced function. Dephosphorylated on Thr-24 and Ser-256 by PP2A in beta-cells under oxidative stress leading to nuclear retention . Phosphorylation of Ser-249 by CDK1 disrupts binding of 14-3-3 proteins leading to nuclear accumulation and has no effect on DNA-binding nor transcriptional activity. Phosphorylation by STK4/MST1 on Ser-212, upon oxidative stress, inhibits binding to 14-3-3 proteins and nuclear export. Acetylated. Acetylation at Lys-262, Lys-265 and Lys-274 are necessary for autophagic cell death induction. Deacetylated by SIRT2 in response to oxidative stress or serum deprivation, thereby negatively regulating FOXO1-mediated autophagic cell death. Ubiquitinated by SRT2. Ubiquitination leads to proteasomal degradation. Methylation inhibits AKT1-mediated phosphorylation at Ser-256 and is increased by oxidative stress. Once in the nucleus, acetylated by CREBBP/EP300. Acetylation diminishes the interaction with target DNA and attenuates the transcriptional activity. It increases the phosphorylation at Ser-256. Deacetylation by SIRT1 results in reactivation of the transcriptional activity. Oxidative stress by hydrogen peroxide treatment appears to promote deacetylation and uncoupling of insulin-induced phosphorylation. By contrast, resveratrol acts independently of acetylation.