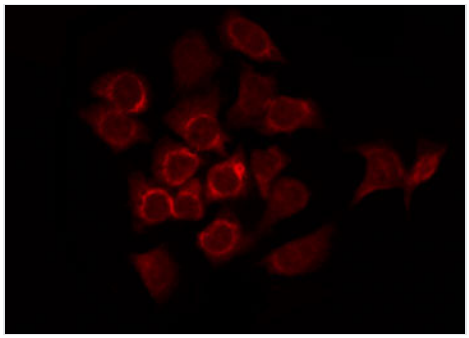
FKHR Antibody – Rabbit Polyclonal

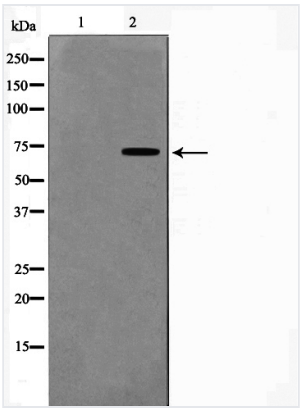
PRODUCT OVERVIEW

Product Name	FKHR, Polyclonal Antibody
Full Product Name	FKHR Antibody
Product Gene Name	anti-FKHR antibody
Product Synonym Gene Name	FOXO1
Product Synonym Names	FKH 1; FKH1; FKHR; Forkhead (Drosophila) homolog 1 (rhabdomyosarcoma); Forkhead box O1; Forkhead box protein O1; Forkhead box protein O1A; Forkhead in rhabdomyosarcoma; Forkhead, Drosophila, homolog of, in rhabdomyosarcoma; FoxO transcription factor; foxo1; FOXO1_HUMAN; FOXO1A; OTTHUMP00000018301
Clonality	Polyclonal
Isotype	IgG
Host	Rabbit
Species Reactivity	Human, Mouse, Rat
Form/Format	Liquid Phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Purity/Purification	The antiserum was purified by peptide affinity chromatography using SulfoLink Coupling Resin.
Concentration	1mg/ml (lot specific)
Specificity	FKHR antibody detects endogenous levels of total FKHR
Preparation and Storage	Store at -20 degree C. Stable for 12 months from date of receipt.
Antibody/Peptide Pairs	FOXO1A peptide (MBS9617524) is used for blocking the activity of FOXO1A antibody (MBS9601967)
Molecular Weight	Observed: 69 kDa Predicted: 70 kDa
3D Structure	ModBase 3D Structure for Q12778
Supply Chain Verification	Manufactured in a lab with traceable raw materials manufactured on site. Coordinated product portfolio of antibodies, pairs, conjugates, recombinant proteins, and immunoassay materials available, please inquire.
Immunogen	A synthesized peptide derived from human FKHR
Subcellular Location	Cytoplasm. Nucleus. Shuttles between cytoplasm and nucleus.
Tissue Specificity	Ubiquitous.
Predicted Cross Reactivity	**Pig, Bovine, Dog, Chicken, Xenopus**
Similarity	Pig (100%), Bovine (100%), Dog (100%), Chicken (100%), Xenopus (100%)
Conjugation	Unconjugated
ISO Certification	Manufactured in an ISO 9001:2015 Certified Laboratory.
Applications Tested/Suitable for anti-FKHR antibody	Western Blot (WB), Immunohistochemisty (IHC), Immunofluorescence (IF), Immunocytochemistry (ICC), ELISA (EIA)
Application Notes for anti-FKHR antibody	WB: 1:500-1:2000 IHC: 1:50-1:200 IF/ICC: 1:100-1:500

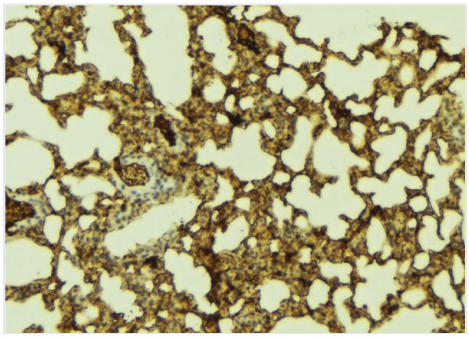
Related Product Information for anti-FKHR antibody	Description: This gene belongs to the forkhead family of transcription factors which are characterized by a distinct forkhead domain. The specific function of this gene has not yet been determined; however, it may play a role in myogenic growth and differentiation. 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 STK4/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. Mediates the function of MLIP in cardiomyocytes hypertrophy and cardiac remodeling (By similarity). Subunit Structure: Interacts with LRPPRC. Interacts with RUNX2; the interaction inhibits RUNX2 transcriptional activity and mediates the IGF1/insulin-dependent BGLAP expression in osteoblasts Interacts with PPP2R1A; the interaction regulates the dephosphorylation of FOXO1 at Thr-24 and Ser-256 leading to its nuclear import. Interacts (acetylated form) with PPARG. Interacts with XBP1 isoform 2; this interaction is direct and leads to FOXO1 ubiquitination and degradation via the proteasome pathway (By similarity). Interacts with NLK. Interacts with SIRT1; the interaction results in the deacetylation of FOXO1 leading to activation of FOXO1-mediated transcription of genes involved in DNA repair and stress resistance. Binds to CDK1. Interacts with the 14-3-3 proteins, YWHAG and YWHAZ; the interactions require insulin-stimulated phosphorylation on Thr-24, promote nuclear exit and loss of transcriptional activity. Interacts with SKP2; the interaction ubiquitinates FOXO1 leading to its proteosomal degradation. The interaction requires the presence of KRIT1. Interacts (via the C-terminal half) with ATF4 (via its DNA-binding domain); the interaction occurs in osteoblasts, regulates glucose homeostasis via suppression of beta-cell proliferation and subsequent decrease in insulin production. Interacts with PRMT1; the interaction methylates FOXO1, prevents PKB/AKT1 phosphorylation and retains FOXO1 in the nucleus. Interacts with EP300 and CREBBP; the interactions acetylate FOXO1. Interacts with SIRT2; the interaction is disrupted in response to oxidative stress or serum deprivation, leading to increased level of acetylated FOXO1, which promotes stress-induced autophagy by stimulating E1-like activating enzyme ATG7. Interacts (acetylated form) with ATG7; the interaction is increased in response to oxidative stress or serum deprivation and promotes the autophagic process leading to cell death. Interacts (via the Fork-head domain) with CEBPA; the interaction increases when FOXO1 is deacetylated. Interacts with WDFY2. Forms a complex with WDFY2 and AKT1 (By similarity). 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 (By similarity). 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.
Other Notes	Small volumes of anti-FKHR antibody vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply.
Searchable Terms for FKHR purchase	MBS9601967 is an antibody targeting FKHR. Also known as FKHR Antibody. Reactive in Human, Mouse, Rat. Host: Rabbit.



Immunofluorescence (IF) of anti-FKHR antibody
MBS9601967 staining HeLa by IF/ICC. The sample were fixed with PFA and permeabilized in 0.1% Triton X-100, then blocked in 10% serum for 45 minutes at 25 degree C. The primary antibody was diluted at 1/200 and incubated with the sample for 1 hour at 37 degree C. An Alexa Fluor 594 conjugated goat anti-rabbit IgG (H+L) Ab, diluted at 1/600, was used as the secondary antibody.



Western Blot (WB) of anti-FKHR antibody
Western blot analysis of FKHR expression in HeLa whole cell lysates, The lane on the left is treated with the antigen-specific peptide.



Immunohistochemistry (IHC) of anti-FKHR antibody
MBS9601967 at 1/100 staining Mouse lung tissue by IHC-P. The sample was formaldehyde fixed and a heat mediated antigen retrieval step in citrate buffer was performed. The sample was then blocked and incubated with the antibody for 1.5 hours at 22 degree C. An HRP conjugated goat anti-rabbit antibody was used as the secondary.

ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for FKHR. It may not necessarily be applicable to this product.

NCBI GI #	9257222
NCBI GeneID	2308
NCBI Accession #	NP_002006.2
UniProt Primary Accession #	Q12778
UniProt Secondary Accession #	O43523; Q5VYC7; Q6NSK6
UniProt Related Accession #	Q12778
GenBank Nucleotide #	NM_002015.3
NCBI Official Full Name	forkhead box protein O1
NCBI Official Synonym Full Names	forkhead box O1
NCBI Official Symbol	FOXO1
NCBI Official Synonym Symbols	FKH1; FKHR; FOXO1A
UniProt Protein Name	Forkhead box protein O1
UniProt Synonym Protein Names	Forkhead box protein O1A; Forkhead in rhabdomyosarcoma
UniProt Gene Name	FOXO1

UniProt Synonym Gene Names	FKHR; FOXO1A
NCBI Protein Information	forkhead box protein O1
NCBI Summary for FKHR	This gene belongs to the forkhead family of transcription factors which are characterized by a distinct forkhead domain. The specific function of this gene has not yet been determined; however, it may play a role in myogenic growth and differentiation. Translocation of this gene with PAX3 has been associated with alveolar rhabdomyosarcoma. [provided by RefSeq, Jul 2008]
UniProt Comments for FKHR	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 STK4/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. Mediates the function of MLIP in cardiomyocytes hypertrophy and cardiac remodeling ().
Research Articles on FKHR	**1.** In particular, we discuss molecular mechanisms that might determine the switch between pro-apoptotic and pro-survival effects of FOXO1 and their interplay with specific differentiation programs.
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