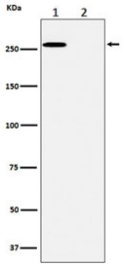
Phospho-ACACA (Ser79) Antibody / ACC1 Phospho Antibody - Metabolic Stress Marker [clone 28A12] (RQ8337)

Catalog No.	Formulation	Size
RQ8337	Antibody in PBS with 0.02% sodium azide, 50% glycerol and 0.4-0.5mg/ml BSA	100 ul

Recombinant RABBIT MONOCLONAL

Bulk quote request

Availability	1-3 business days
Species Reactivity	Human, Mouse, Rat
Format	Purified
Host	Rabbit
Clonality	Recombinant Rabbit Monoclonal
Isotype	Rabbit IgG
Clone Name	28A12
Purity	Affinity chromatography
UniProt	Q13085
Applications	Western Blot : 1:500-1:2000
Limitations	This Phospho-ACACA (Ser79) Antibody / ACC1 Phospho Antibody - Metabolic Stress Marker is available for research use only.



Phospho-ACACA (Ser79) Antibody for WB. Western blot analysis of ACACA / Acetyl-CoA carboxylase 1 phosphorylation in human A431 cell lysate using Phospho-ACACA (Ser79) Antibody - metabolic stress marker (clone 28A12). Lane 1: untreated lysate, Lane 2: lysate treated with lambda protein phosphatase. A band is detected at approximately 260 kDa in the untreated sample, consistent with phosphorylated ACC1, while signal is abolished following phosphatase treatment, confirming phosphorylation-dependent detection at Ser79 and supporting specificity for the inhibited form of ACC1 associated with suppressed lipogenesis.

Description

Acetyl-CoA carboxylase 1 (ACACA), also known as ACC1, is a cytosolic, biotin-dependent enzyme that catalyzes the conversion of acetyl-CoA to malonyl-CoA, representing the rate-limiting step in de novo fatty acid synthesis. Phosphorylation of ACC1 at serine 79 (Ser79) is a critical inhibitory modification that directly suppresses enzymatic

activity and reduces lipogenic flux. This phosphorylation event serves as a central regulatory mechanism linking cellular energy status to lipid metabolism. Phospho-ACACA (Ser79) Antibody enables detection of this functionally important modification, providing a direct readout of metabolic suppression and lipid synthesis inhibition.

Phospho-ACACA antibody, also referred to as phospho ACC1 antibody or ACACA Ser79 antibody in the literature, specifically recognizes ACC1 only when phosphorylated at Ser79. This site-specific modification decreases malonyl-CoA production, thereby limiting substrate availability for fatty acid synthesis while indirectly promoting fatty acid oxidation through relief of CPT1 inhibition. As a result, detection of Ser79 phosphorylation reflects a coordinated metabolic shift from anabolic lipid production toward energy utilization.

ACC1 Ser79 phosphorylation is rapidly induced under a wide range of metabolic stress conditions, including nutrient deprivation, glucose limitation, hypoxia, oxidative stress, and pharmacologic activation of energy-sensing pathways. These stimuli converge on signaling networks that suppress lipogenesis and promote cellular adaptation to energy deficit. Increased phospho-ACC1 signal therefore serves as a sensitive biomarker of metabolic stress and pathway activation, particularly in systems undergoing rapid changes in energy demand.

At the signaling level, phosphorylation of ACC1 at Ser79 is primarily mediated by AMP-activated protein kinase (AMPK), a master regulator of cellular energy homeostasis. Activation of AMPK leads to immediate inhibition of ACC1 activity, effectively shutting down fatty acid synthesis during low-energy states. This tight coupling between AMPK activation and ACC1 phosphorylation makes phospho-ACC1 a widely used downstream readout of AMPK pathway engagement, while still allowing interpretation within the broader context of metabolic stress responses.

In cancer and other proliferative contexts, suppression of ACC1 Ser79 phosphorylation is often required to sustain high rates of fatty acid synthesis needed for membrane biogenesis and tumor growth. Conversely, increased phosphorylation at this site is associated with reduced lipogenic capacity and impaired proliferation. Detection of phospho-ACC1 in tumor tissues can therefore provide insight into metabolic heterogeneity, therapeutic response, and pathway activity within the tumor microenvironment.

Unlike total ACC1 detection, which reflects protein abundance, phosphorylation-specific detection at Ser79 provides direct functional information about enzyme inhibition and metabolic state. This distinction is essential for interpreting lipid metabolism, as elevated ACC1 expression does not necessarily indicate active lipogenesis if the enzyme is phosphorylated and inactive. Because ACC2 is regulated at a distinct phosphorylation site, Ser79-specific antibodies are selective for ACC1 and do not detect ACC2, ensuring precise target specificity.

This Phospho-ACACA (Ser79) Antibody (clone 28A12) is designed to detect ACC1 only when phosphorylated at Ser79 and does not recognize non-phosphorylated ACC1. It is well suited for research applications focused on metabolic stress, lipogenesis inhibition, and cellular energy regulation, providing a robust tool for studying metabolic control at the level of enzyme activity.

This antibody is part of the ACACA antibody collection, where additional Acetyl-CoA Carboxylase 1 antibodies can be explored.

Application Notes

Optimal dilution of the Phospho-ACACA (Ser79) Antibody / ACC1 Phospho Antibody - Metabolic Stress Marker should be determined by the researcher.

Immunogen

A synthetic peptide derived from the area surrounding phosphorylated serine 79 of human ACC was used as the immunogen for the Phospho-ACACA antibody.

Storage

Store the Phospho-ACACA antibody at -20oC.

Alternate Names

phospho ACACA antibody, phospho ACC1 antibody, ACACA Ser79 antibody, ACC1 pSer79 antibody, phospho acetyl-CoA carboxylase antibody, metabolic stress marker antibody