SignalSilence® AMPKα2 siRNA I

For Research Use Only. Not For Use In Diagnostic Procedures.

Species Cross- Reactivity: H

Description: SignalSilence® AMPKα2 siRNA I from Cell Signaling Technology (CST) allows the researcher to specifically inhibit AMPKα2 expression using RNA interference, a method whereby gene expression can be selectively silenced through the delivery of double stranded RNA molecules into the cell. All SignalSilence® siRNA products from CST are rigorously tested in-house and have been shown to reduce target protein expression by western analysis.

Background: AMP-activated protein kinase (AMPK) is highly conserved from yeast to plants and animals and plays a key role in the regulation of energy homeostasis (1). AMPK is a heterotrimeric complex composed of a catalytic α subunit and regulatory β and γ subunits, each of which is encoded by two or three distinct genes (α1, 2; β1, 2; γ1, 2, 3) (2). The kinase is activated by an elevated AMP/ATP ratio due to cellular and environmental stress, such as heat shock, hypoxia and ischemia (1). The tumor suppressor LKB1, in association with accessory proteins such as heat shock, hypoxia and ischemia (1). The upstream kinase and the biological significance of these phosphorylation events have yet to be elucidated (6). The β1 subunit is post-translationally modified by myristoylation and multisite phosphorylation including Ser24/25, Ser96, Ser101, Ser108 and Ser182 (6,7). Phosphorylation at Ser108 of the β1 subunit seems to be required for the activation of AMPK enzyme, while phosphorylation at Ser24/25 and Ser182 affects AMPK localization (7). Several mutations in AMPKγ subunits have been identified, most of which are located in the putative AMP/ATP binding sites (CBS or Bateman domains). Mutations at these sites lead to reduction of AMPK activity and cause glycogen accumulation in heart or skeletal muscle (1,2). Accumulating evidence indicates that AMPK not only regulates the metabolism of fatty acids or skeletal muscle (1,2). Accumulating evidence indicates that AMPK not only regulates the metabolism of fatty acids but also modulates protein synthesis and cell growth through EF2 and TSC2/mTOR pathways, as well as blood flow via eNOS/nNOS (1).

Directions for Use: CST recommends transfection with 100 nM SignalSilence Control siRNA (Unconjugated) α2 siRNA I from CST to knockdown AMPKα2 expression. The upstream kinase and the biological significance of these phosphorylation events have yet to be elucidated (6). The β1 subunit is post-translationally modified by myristoylation and multisite phosphorylation including Ser24/25, Ser96, Ser101, Ser108 and Ser182 (6,7). Phosphorylation at Ser108 of the β1 subunit seems to be required for the activation of AMPK enzyme, while phosphorylation at Ser24/25 and Ser182 affects AMPK localization (7). Several mutations in AMPKγ subunits have been identified, most of which are located in the putative AMP/ATP binding sites (CBS or Bateman domains). Mutations at these sites lead to reduction of AMPK activity and cause glycogen accumulation in heart or skeletal muscle (1,2). Accumulating evidence indicates that AMPK not only regulates the metabolism of fatty acids but also modulates protein synthesis and cell growth through EF2 and TSC2/mTOR pathways, as well as blood flow via eNOS/nNOS (1).

Applications Key: W—Western; IP—Immunoprecipitation; IHC—Immunohistochemistry; CWP—Chromatin Immunoprecipitation; IF—Immuno-fluorescence; F—Flow cytometry; E-P—ELISA-Peptide

Species Cross-Reactivity Key: H—human; M—mouse; R—rat; Hm—hamster; Mm—monkey; Mm—inbred; C—chicken; Dm—D. melanogaster; X—Xenopus; Z—zebrafish; B—bovine

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SignalSilence® AMPKα2 siRNA I

10 µM in 300 µl (100 transfections)

100 nM AMPK
2 siRNA I 48 to 72 hours prior to cell lysis.

For transfection procedure, follow protocol provided by the transfection reagent manufacturer. Please feel free to contact CST with any questions on use. The upstream kinase and the biological significance of these phosphorylation events have yet to be elucidated (6). The β1 subunit is post-translationally modified by myristoylation and multisite phosphorylation including Ser24/25, Ser96, Ser101, Ser108 and Ser182 (6,7). Phosphorylation at Ser108 of the β1 subunit seems to be required for the activation of AMPK enzyme, while phosphorylation at Ser24/25 and Ser182 affects AMPK localization (7). Several mutations in AMPKγ subunits have been identified, most of which are located in the putative AMP/ATP binding sites (CBS or Bateman domains). Mutations at these sites lead to reduction of AMPK activity and cause glycogen accumulation in heart or skeletal muscle (1,2). Accumulating evidence indicates that AMPK not only regulates the metabolism of fatty acids but also modulates protein synthesis and cell growth through EF2 and TSC2/mTOR pathways, as well as blood flow via eNOS/nNOS (1).

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Western blot analysis of extracts from HeLa cells, transfected with 100 nM SignalSilence Control siRNA (Unconjugated) α2 siRNA I or SignalSilence® AMPKα2 siRNA II #6630 (+), using AMPKα2 Antibody confirms silencing of AMPKα2 expression, while the α-Tubulin (11H10) Rabbit mAb is used as a loading control.