

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human BMAL1 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2B} Clone # 1029536
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human BMAL1 Met105-Glu303 Accession # O00327
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

ELISA Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

BMAL1 (Brain and Muscle ARNT-like 1; ARNTL) is an essential core component in circadian clock machinery which is regulatory mechanism for circadian rhythms. Circadian clock genes include three period proteins (PER1, PER2 and PER3), two cryptochromes (CRY1 and CRY2), CLOCK, NPAS2 and BMAL proteins, wherein BMAL1 has a potential to heterodimerize with CLOCK or NPAS2 genes; and this neocomplex drives transcription from E-box elements (5'-CACGTG-3') found in circadian-responsive genes's promoters. PER/CRY proteins negatively regulate CLOCK/BMAL1 dimer-mediated transcription, thereby forming the feedback loop that regulates the timing of clock gene transcription. BMAL1 also associates with GNB2L1/RACK1 and PRKCA in a nuclear complex, wherein GNB2L1 and PRKCA are recruited to the complex in a circadian manner. BMAL1 undergoes acetylation (Lys-538), phosphorylation and sumoylation (Lys-259) upon dimerization with CLOCK and acetylation facilitates CRY1-mediated repression. CLOCK-BMAL1 double mutations within PAS domains leads to synergistic desensitization to high levels of CRY on repression of CLOCK-BMAL1 transcriptional activity of PER1 and, disrupt circadian rhythmicity. Clock genes functions primarily as tumor suppressors and their abbarant expression is observed in malignant pleural mesothelioma, colorectal and breast cancer.

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