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PER2 Rabbit pAb

Catalog # AP100266

Product Information

Application	IHC-P, IHC-F, IF
Primary Accession	O15055
Other Accession	O15055
Reactivity	Rat, Mouse
Predicted	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	136579
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human PER2/Period circadian protein 2
Epitope Specificity	731-830/1255
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Contains 1 PAC (PAS-associated C-terminal) domain. Contains 2 PAS (PER-ARNT-SIM) domains.
SUBUNIT	Homodimer. Component of the circadian core oscillator, which includes the CRY proteins, CLOCK or NPAS2, BMAL1 or BMAL2, CSNK1D and/or CSNK1E, TIMELESS, and the PER proteins. Interacts directly with PER1 and PER3, and through a C-terminal domain, with CRY1 and CRY2. Interaction with CSNK1D or CSNK1E promotes nuclear location of PER proteins. Interacts, via its second PAS domain, with TIMELESS in vitro. Interacts with NFIL3 (By similarity). Interacts with PML (By similarity).
Post-translational modifications	Phosphorylated by CSNK1E and CSNK1D. Phosphorylation results in PER2 protein degradation.
DISEASE	Defects in PER2 are a cause of familial advanced sleep-phase syndrome (FASPS) [MIM:604348]. FASPS is characterized by very early sleep onset and offset. Individuals are 'morning larks' with a 4 hours advance of the sleep, temperature and melatonin rhythms.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	PER2, a mammalian homologue of the Drosophila period gene, shares a 40% homology with PER1 including the protein dimerization PAS domain. PER2 is a circadian regulator that may act as a transcription factor. It behaves as a negative element in circadian transcriptional loop. PER2 does not appear to bind DNA, suggesting indirect transcriptional inhibition. Expression oscillates with a 24 hour rhythm in the suprachiasmatic nucleus (SCN) and the whole eyes. Oscillations are maintained under constant darkness and are responsive to changes of the light/dark cycles. There is a 4 hour time delay between PER1 and PER2 oscillations. The expression rhythms appear to originate from retina.

Additional Information

Gene ID	8864
Other Names	FASPS; FASPS1; mKIAA0347; mPer2; PER2_HUMAN; PER2; hPER2; Circadian clock protein PERIOD 2; KIAA0347; PER2_MOUSE; period circadian regulator 2; period (Drosophila) homolog 2; period homolog 2 (Drosophila); period circadian clock 2
Dilution	IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	PER2
Synonyms	KIAA0347
Function	Transcriptional repressor which forms a core component of the circadian clock. The circadian clock, an internal time-keeping system, regulates various physiological processes through the generation of approximately 24 hour circadian rhythms in gene expression, which are translated into rhythms in metabolism and behavior. It is derived from the Latin roots 'circa' (about) and 'diem' (day) and acts as an important regulator of a wide array of physiological functions including metabolism, sleep, body temperature, blood pressure, endocrine, immune, cardiovascular, and renal function. Consists of two major components: the central clock, residing in the suprachiasmatic nucleus (SCN) of the brain, and the peripheral clocks that are present in nearly every tissue and organ system. Both the central and peripheral clocks can be reset by environmental cues, also known as Zeitgebers (German for 'timegivers'). The predominant Zeitgeber for the central clock is light, which is sensed by retina and signals directly to the SCN. The central clock entrains the peripheral clocks through neuronal and hormonal signals, body temperature and feeding-related cues, aligning all clocks with the external light/dark cycle. Circadian rhythms allow an organism to achieve temporal homeostasis with its environment at the molecular level by regulating gene expression to create a peak of protein expression once every 24 hours to control when a particular physiological process is most active with respect to the solar day. Transcription and translation of core clock components (CLOCK, NPAS2, BMAL1, BMAL2, PER1, PER2, PER3, CRY1 and CRY2) plays a critical role in rhythm generation, whereas delays imposed by post- translational modifications (PTMs) are important for determining the period (tau) of the rhythms (tau refers to the period of a rhythm and is the length, in time, of one complete cycle). A diurnal rhythm is synchronized with the day/night cycle, while the ultradian and infradian rhythms have a period shorter and longer than 24 hours, respectively. Disruptions in the circadian rhythms contribute to the pathology of cardiovascular diseases, cancer, metabolic syndrome and aging. A transcription/translation feedback loop (TTFL) forms the core of the molecular circadian clock mechanism. Transcription factors, CLOCK or NPAS2 and BMAL1 or BMAL2, form the positive limb of the feedback loop, act in the form of a heterodimer and activate the transcription of core clock genes and clock-controlled genes (involved in key metabolic processes), harboring E-box elements (5'-CACGTG-3') within their promoters. The core clock genes: PER1/2/3 and CRY1/2 which are transcriptional repressors form the negative limb of the feedback loop and interact with the CLOCK|NPAS2-BMAL1|BMAL2

heterodimer inhibiting its activity and thereby negatively regulating their own expression. This heterodimer also activates nuclear receptors NR1D1/2 and RORA/B/G, which form a second feedback loop and which activate and repress BMAL1 transcription, respectively. PER1 and PER2 proteins transport CRY1 and CRY2 into the nucleus with appropriate circadian timing, but also contribute directly to repression of clock-controlled target genes through interaction with several classes of RNA-binding proteins, helicases and others transcriptional repressors. PER appears to regulate circadian control of transcription by at least three different modes. First, interacts directly with the CLOCK-BMAL1 at the tail end of the nascent transcript peak to recruit complexes containing the SIN3-HDAC that remodel chromatin to repress transcription. Second, brings H3K9 methyltransferases such as SUV39H1 and SUV39H2 to the E-box elements of the circadian target genes, like PER2 itself or PER1. The recruitment of each repressive modifier to the DNA seems to be very precisely temporally orchestrated by the large PER complex, the deacetylases acting before than the methyltransferases. Additionally, large PER complexes are also recruited to the target genes 3' termination site through interactions with RNA-binding proteins and helicases that may play a role in transcription termination to regulate transcription independently of CLOCK-BMAL1 interactions. Recruitment of large PER complexes to the elongating polymerase at PER and CRY termination sites inhibited SETX action, impeding RNA polymerase II release and thereby repressing transcriptional reinitiation. May propagate clock information to metabolic pathways via the interaction with nuclear receptors. Coactivator of PPARA and corepressor of NR1D1, binds rhythmically at the promoter of nuclear receptors target genes like BMAL1 or G6PC1. Directly and specifically represses PPARG proadipogenic activity by blocking PPARG recruitment to target promoters and thereby inhibiting transcriptional activation. Required for fatty acid and lipid metabolism, is involved as well in the regulation of circulating insulin levels. Plays an important role in the maintenance of cardiovascular functions through the regulation of NO and vasodilatatory prostaglandins production in aortas. Controls circadian glutamate uptake in synaptic vesicles through the regulation of VGLUT1 expression. May also be involved in the regulation of inflammatory processes. Represses the CLOCK-BMAL1 induced transcription of BHLHE40/DEC1 and ATF4. Negatively regulates the formation of the TIMELESS-CRY1 complex by competing with TIMELESS for binding to CRY1.

Cellular Location

[Isoform 1]: Nucleus. Cytoplasm {ECO:0000250|UniProtKB:O54943}. Cytoplasm, perinuclear region {ECO:0000250|UniProtKB:O54943}. Note=Nucleocytoplasmic shuttling is effected by interaction with other circadian core oscillator proteins and/or by phosphorylation. Translocate to the nucleus after phosphorylation by CSNK1D or CSNK1E. Also translocated to the nucleus by CRY1 or CRY2. PML regulates its nuclear localization {ECO:0000250|UniProtKB:O54943}

Tissue Location

Widely expressed. Found in heart, brain, placenta, lung, liver, skeleatal muscle, kidney and pancreas. High levels in skeletal muscle and pancreas. Low levels in lung. Isoform 2 is expressed in keratinocytes (at protein level)

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.