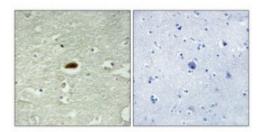


Anti-Per2 antibody





Description	Rabbit polyclonal to Per2.	

Model STJ95028

Host Rabbit

Reactivity Human, Mouse

Applications ELISA, IF, IHC

Immunogen Synthesized peptide derived from human Per2 around the non-

phosphorylation site of S662.

Immunogen Region 610-690 aa

Gene ID 8864
Gene Symbol PER2

Dilution range IHC 1:100-1:300IF 1:200-1:1000ELISA 1:5000

Specificity Per2 Polyclonal Antibody detects endogenous levels of Per2 protein.

Tissue Specificity 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).

Purification The antibody was affinity-purified from rabbit antiserum by affinity-

chromatography using epitope-specific immunogen.

Note For Research Use Only (RUO).

Protein Name Period circadian protein homolog 2 hPER2 Circadian clock protein PERIOD 2

Molecular Weight 120 kDa

Clonality Polyclonal

Conjugation Unconjugated

Isotype IgG

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Concentration 1 mg/ml

Storage Instruction Store at -20°C, and avoid repeat freeze-thaw cycles.

Database Links HGNC:8846OMIM:603426

Alternative Names Period circadian protein homolog 2 hPER2 Circadian clock protein PERIOD 2

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 feedingrelated 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, ARNTL/BMAL1, ARNTL2/BMAL2, PER1, PER2, PER3, CRY1 and CRY2) plays a critical role in rhythm generation, whereas delays imposed by posttranslational 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 ARNTL/BMAL1 or ARNTL2/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 Ebox 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-ARNTL/BMAL1|ARNTL2/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 ARNTL/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-ARTNL/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-ARTNL/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 ARNTL or G6PC. 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-ARNTL/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 Localization

Isoform 1: Nucleus. Cytoplasm Cytoplasm, perinuclear region. 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.. Isoform 2: Nucleus, nucleolus

Post-translational Modifications Acetylated. Deacetylated by SIRT1, resulting in decreased protein stability. Phosphorylated by CSNK1E and CSNK1D. Phosphorylation results in PER2 protein degradation. May be dephosphorylated by PP1. Ubiquitinated, leading to its proteasomal degradation. Ubiquitination may be inhibited by CRY1.