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Cry2 Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP6135a

Product Information

Application	WB, IHC-P, E
Primary Accession	Q49AN0
Other Accession	NP_066940
Reactivity	Human, Mouse
Predicted	Canine, Rabbit
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	66947
Antigen Region	564-593

Additional Information

Gene ID	1408
Other Names	Cryptochrome-2, CRY2, KIAA0658
Target/Specificity	This Cry2 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 564-593 amino acids from the C-terminal region of human Cry2.
Dilution	WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.05% (V/V) Proclin 300. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Cry2 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	CRY2
Synonyms	KIAA0658
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 syndromes 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. CRY1 and CRY2 have redundant functions but also differential and selective contributions at least in defining the pace of the SCN circadian clock and its circadian transcriptional outputs. Less potent transcriptional repressor in cerebellum and liver than CRY1, though less effective in lengthening the period of the SCN oscillator. Seems to play a critical role in tuning SCN circadian period by opposing the action of CRY1. With CRY1, dispensable for circadian rhythm generation but necessary for the development of intercellular networks for rhythm synchrony. May mediate circadian regulation of cAMP signaling and gluconeogenesis by blocking glucagon-mediated increases in intracellular cAMP concentrations and in CREB1 phosphorylation. Besides its role in the maintenance of the circadian clock, is also involved in the regulation of other processes. Plays a key role in glucose and lipid metabolism modulation, in part, through the transcriptional regulation of genes involved in these pathways, such as LEP or ACSL4. Represses glucocorticoid receptor NR3C1/GR-induced transcriptional activity by binding to glucocorticoid response elements (GREs). Represses the CLOCK-BMAL1 induced transcription of BHLHE40/DEC1. Represses the CLOCK-BMAL1 induced transcription of NAMPT (By similarity). Represses PPARD and its target genes in the skeletal muscle and limits exercise capacity (By similarity). Represses the transcriptional activity of NR1I2 (By similarity).

Cellular Location

Cytoplasm. Nucleus Note=Translocated to the nucleus through interaction

	with other Clock proteins such as PER2 or BMAL1
Tissue Location	Expressed in all tissues examined including fetal brain, fibroblasts, heart, brain, placenta, lung, liver, skeletal muscle, kidney, pancreas, spleen, thymus, prostate, testis, ovary, small intestine, colon and leukocytes. Highest levels in heart and skeletal muscle.

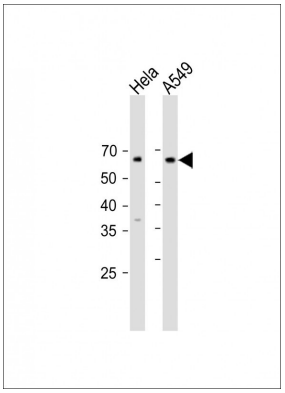
Background

Various biochemical, physiological and behavioural processes display circadian rhythms controlled by an internal biological clock. The central ?gears?driving this clock appear to be composed of an autoregulatory transcription/posttranslation-based feedback loop. Cryptochrome 1 (CRY1) and 2 (CRY2) are DNA-binding flavoproteins that bear some homology to blue-light receptors and photolyases. In Drosophila, CRY is a photoreceptor for the circadian clock where it binds to the clock component TIM in a light-dependent fashion and blocks its function. Mammalian CRY1 and CRY2 function via light-independent interactions with circadian genes CLOCK and BMAL1, as well as with PER1, PER2, and TIM. They seem to act as light-independent components of the circadian clock and likely regulate Per1 transcriptional cycling via interactions with both the activator and its feedback inhibitors. Mutant mice not expressing the Cry1 or Cry2 protein display accelerated and delayed periodicity of locomotor activity, respectively. It appears that the combination of both proteins working together is essential to synchronize the organism to circadian phases. A critical balance between Cry1 and Cry2 is required for proper clock function; in complete darkness, double-mutant mice present with instantaneous arrhythmicity, indicating the absence of an internal circadian clock.

References

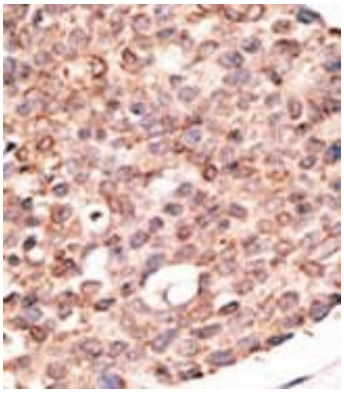
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Images



All lanes: Anti-Cry2 Antibody (C-term) at 1:1000 dilution Lane 1: Hela whole cell lysate Lane 2: A549 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary: Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated (ASP1615) at 1/15000 dilution. Observed band size: 67 KDa Blocking/Dilution buffer: 5% NFDM/TBST.

Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.



Citations

- CRISPR/Cas9 mediated genome editing of Helicoverpa armigera with mutations of an ABC transporter gene HaABCA2 confers resistance to Bacillus thuringiensis Cry2A toxins.

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