BMAL1 binds CLOCK,NPAS2 forming BMAL1:CLOCK,NPAS2 heterodimer

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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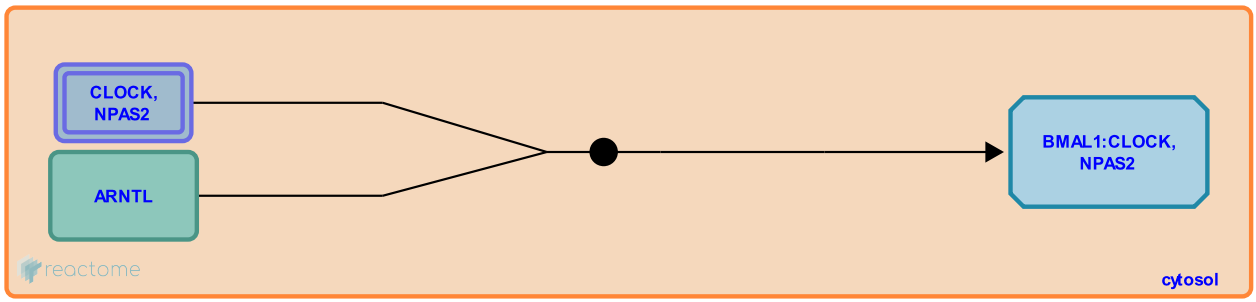
Stable identifier: R-HSA-400228

Type: binding

Compartments: cytosol

Inferred from: Bmal1 binds Clock,Npas2 forming Bmal1:Clock,Npas2 heterodimer (Mus musculus)



BMAL1 (ARNTL), CLOCK, and NPAS2 are basic helix-loop-helix transcription factors. In humans BMAL1 has been demonstrated to form a heterodimer with CLOCK. In mouse, BMAL1 can form a heterodimer with either CLOCK or NPAS2. By analogy with other basic helix-loop-helix proteins the basic domain binds DNA, in this case the E-box motif, and the helix-loop-helix domains interact to form the heterodimer. BMAL1 and CLOCK/NPAS2 are codependently phosphorylated by unknown kinases after dimerization. The phosphorylation enhances transactivation activity and is inhibited by PER:CRY complexes. Both CLOCK and NPAS2 are expressed in the suprachiasmatic nucleus of the hypothalamus and act redundantly there. The tissue distributions of CLOCK and NPAS2 do not entirely overlap, however. For example, NPAS2 but not CLOCK is found in forebrain.

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Editions

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