Delayed Sleep Phase Syndrome (DSPS) is a circadian rhythm sleep disorder characterized by later sleeping and waking times than standard [1]. In addition to the lag in sleeping and waking schedule, individuals with DSPS often report fragmented patterns of sleep: brief periods of sleep during the night and extended naps during the day. Recent studies have identified a candidate allele in the CRY1 gene coding for a repressor of the transcription factors CLOCK and BMAL1 in the circadian clocks of mammals. The Cry1 protein is composed of a highly conserved N-terminal photolyase homology region and a more divergent C-terminal tail [2]. The N-terminal’s photolyase region is responsible for Clock/Bmal1 repression [3] and the C-terminal protein tail has been linked to nuclear translocation and lengthening via phosphorylation [4], *yet* *the process by which this protein tail interacts with the Clock/Bmal1 transcription factors in the circadian rhythm cycle are still unknown.*

My **primary goal** is to understand the role of phosphorylation of the Cry1 protein in the regulation of circadian cycles. I will test the **hypothesis** that phosphorylation events in the Cry1 protein tail increase the length of the circadian cycle by structurally regulating the Cry1 protein’s affinity to transcription factors. My **long-term goal** is to understand the processes by which the Cry1 affects circadian rhythm patterns.

**Aim #1**: Identify and understand the protein sequences conserved with the CRY1 gene that are necessary for common circadian clock transcriptional regulation.

**Approach**: I will use online databases Pfam and SMART to align the conserved protein sequences of Cry1 of human and mouse. I will then use CRISPR-Cas9 to create transgenic mice lines with a 72 base pair deletion on the Cry1 gene on chromosome 10: Cry1-/Cry1- and Cry1+/Cry1-. Transgenic and wild-type mice will then be reared under equal environmental conditions and their sleep-wake cycles will be observed.

**Hypothesis**: Deletions within the Cry1 gene will result in the lengthening of sleep-wake cycle of the affected mice lines.

**Rationale**: The molecular clock in mammals is controlled through a negative feedback loop with the transcriptional repressor protein, Cry1. The absence of Cry1 protein at normal levels should result in the inefficient regulation of the clock complex and the lengthening of the sleep-wake cycle[5].

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