Seminar/Talk

Nuclear CK1δ as a Critical Determinant of PER:CRY Complex Dynamics and Circadian Period

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The mammalian circadian clock is governed by a feedback loop in which the transcription activator CLOCK:BMAL1 induces expression of its inhibitors, PERs and CRYs, which form a complex with CK1δ, the main circadian kinase. However, the spatiotemporal dynamics of this feedback loop and the precise role of CK1δ remain incompletely understood. Using an inducible overexpression system, we show that nuclear availability of CK1δ is limited by both rapid nuclear degradation and active export of unassembled kinase, while cytoplasmic kinase is readily available for association with PERs. We demonstrate that CK1δ-mediated phosphorylation may disrupt PER2-CRY1 interaction thereby resulting in cytoplasmic PER2 dimers containing substoichiometric amounts of CRY1. Analysis of endogenous PER2 localization in the context of an intact circadian clock reveals that PER2 accumulates in the cytoplasm late in the circadian cycle. Based on these findings, we propose that cytoplasmic accumulation of PER:CRY:CK1δ complexes contributes to the clearance of nuclear PER2, while the CK1δ-dependent release of CRY1 into the nucleus may sustain CLOCK:BMAL1 repression on DNA supporting the transition from the early to the late repressive phase.

Friday, February 20, 2026 10:00am - 11:00am
Moonstone Bldg / Ground floor / Seminar Room E

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