Leukemia inhibitory factor regulates cyclin-cdk complexes and duration of G1 and G2 transits in mouse embryonic (ES) cells

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Séminaire Interne

Salle de conférence
151, cours Albert Thomas
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Jeudi 5 Février

AM : 09h00 à 12h00

Equipe « Rythmes circadiens »

Introduction : Neuronal Systems to Keep Track of Time
Howard M COOPER

Synchronising Circadian rhythms on Mars and on Earth
Claude GRONFIER

Dissecting out the role of photoreceptors in clock mechanisms
Ouria BENYAHYA

General Introduction – Irradiance detection and temporal integration in the circadian system
Camille RIEUX

Electrophysiological responses to photic stimulation
Elise DROUYER

Pupil responses in humans : a tool to investigate the non-image forming system
Camille RIEUX

Multi-electrode recording in the SCN of Awake behaving animals
Ludovic MURE

Planned future directions
Howard M COOPER

Midi : Pause déjeuner

PM : 14h00 à 17h00

Equipe « cellules souches et cycle cellulaire »

Control of self-renewal and differentiation of mouse and rhesus embryonic stem cells.
Pierre SAVATIER

Etude des mécanismes de contrôle de l'auto renouvellement des cellules embryonnaires souches (cellules ES) chez la souris et chez le primate
Pierre Yves BOURILLOT

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Marielle AFANASSIEFF

Optimisation génétique de la différenciation des cellules ES en cellules pancréatiques
Mélanie MARCHAND

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The propagation of mouse embryonic stem (ES) cells is dependent on leukemia inhibitory factor (LIF), or cytokines of the interleukin-6 family, which can activate the gp130 receptor. Despite LIF-gp130 playing a critical role in controlling the self-renewal of mouse ES cells, little is known on the target genes and the cellular processes that it regulates.

Progression through the cell-cycle is controlled by complexes of cyclins and cyclin-dependent kinases (CDKs). Of prime importance are (*i*) cyclin E forming complexes with CDK2 during the G1 phase of the cell-cycle, and (*ii*) cyclin B1 forming complexes with CDK1 during the G2 phase of the cell-cycle. Cyclin E-CDK2 and cyclin B1-CDK1 complexes are critical determinants, and rate-limiting drivers, of the progression through the G1 and G2 phases, respectively.

Here we show that LIF-gp130 activity up-modulates cyclin E-CDK2, thereby accelerates the transit through the G1 phase. In contrast, LIF-gp130 activity down-modulates cyclin B1-CDK1 and slows down transit through the G2 phase. These data indicate that regulation of cyclin-Cdk complex activities is part of the cellular response triggered by LIF receptor activation in mouse ES cells. Whether the modulation of cyclin-Cdk activities by LIF impacts on self-renewal is currently under investigation.