平成 30 年 11 月 1 日
November 1, 2018

大学院学生各位
To All Graduate Students

平成 **30** 年度

基盤医学特論 開講通知

Information on Special Lecture Tokuron 2018

題目（Title）： Postnatal movement restriction deteriorates locomotion and sensorimotor circuitry: implications for developmental coordination disorders

講師（Teaching Staff）: J-Olivier Coq, PhD

Institute of Neuroscience de la Timone (INT) in Marseille, France

日時：平成 30 年 11 月 19 日(月) 18 時 00 分より（90 分）

Time and Date: from 18:00, Monday, November 19, 2018

場所： 鶴友会館 2 階大会議室

Room: Kakuyu Kaikan (Alumni Hall) 2F, Large Conference Room

言語： 英語

Language: English

※関係講座・部門等の連絡担当者：佐藤義朗
＊＊講座 総合周産期母子医療センター/小児科学（内線 2294）
Contact: Yoshiaki Sato, Center for Maternal-Neonatal Care / Department of Pediatrics (Ext. 2294)

事前の申込みは不要です。No Registration Required.

医学部学務課大学院係
Student Affairs Division, School of Medicine

Abstract

Motor control and body representations in the central nervous system are built, i.e., patterned, during development by sensorimotor experience and somatosensory feedback/reafference. In the absence of brain damage, children with developmental coordination disorders (DCD) display deficits in planning, executing and controlling movements, concomitant with deficits in executive functions. Thus, are early sensorimotor atypicalities at the origin of long-lasting abnormal development of brain anatomy and functions? We hypothesize that degraded locomotor outcomes in adulthood originate as a consequence of early atypical sensorimotor experiences that induce developmental disorganization of sensorimotor circuitry. In this lecture I will show that postnatal sensorimotor restriction (SMR), through hind limb immobilization from birth to one month, led to enduring digitigrade locomotion with ankle-knee overextension, degraded musculoskeletal tissues (e.g., gastrocnemius atrophy), and clear signs of spinal hyperreflexia in adult rats, suggestive of spasticity; each individual disorder likely interplaying in self-perpetuating cycles. I will also show that 28 days of daily SMR degraded the topographical organization of somatosensory hind limb maps, reduced both somatosensory and motor map areas devoted to the hind limb representation and altered neuronal response properties in the sensorimotor cortex several weeks after the cessation of SMR. We found no neuroanatomical histopathology in hind limb sensorimotor cortex, yet increased glutamatergic neurotransmission that matched clear signs of spasticity and hyperexcitability in the adult lumbar spinal networks. Thus, even in the absence of a brain insult, movement disorders and brain dysfunction can emerge as a consequence of reduced and atypical patterns of motor outputs and somatosensory feedback that induce maladaptive neuroplasticity. Our results may contribute to understanding the inception and mechanisms underlying neurodevelopmental disorders, such as DCD.