

Liste de Symposium

Symposium I: Neuronal processes of attention and action and their use in artificial intelligent systems

Abstract: We have recently reached a level of understanding of brain processes far beyond the simple analysis of sensory information and motor control. The knowledge of the complex tasks that our brain performs is bringing the attention of the scientific community on how to exploit this knowledge in applicative fields as artificial intelligence.

We now know that the cortical control of reach-to-grasp actions involves specific parieto-frontal circuits that perform the sensorimotor transformations needed to correctly execute the action under sensory guidance. We also begun to know how other processes involved in the reach-to-grasp actions are controlled by the brain. Different neuronal networks encode the salience of visual stimuli and control the spatial shifts of attention; other neural networks are involved in orchestrating gaze direction and arm movements, and in coding of goals of the action.

Three speakers will share their neurophysiological knowledge about these processes in the primate brain. The fourth speaker will show how this knowledge is useful in building artificial intelligent systems able to perform autonomously the sensorimotor transformations required for a robot to interact with objects in space.

Key Words: Hand actions, Hierarchy of goals, Attentional shifts, Eye-arm coordination, Robotics

1. ***Chairman:* Galletti Claudio**, Bologna, Italy
2. **Duhamel Jean-René**, Bron, France: A fronto-parietal network for attentional control
3. **Patrizia Fattori**, Bologna, Italy: Neuronal processes of action in the superior parietal lobule of primate brain
4. **Leonardo Fogassi**, Parma, Italy: The role of inferior parietal and premotor cortices in coding action goals
5. **Angel Pasqual del Pobil**, Castellon, Spain: Sensorimotor integration in robotic reach-to-grasp actions

Symposium II: Cannabinoids and brain reward processes

Abstract: The endogenous cannabinoid system is a newly discovered neuromodulatory system comprised of receptors, endogenous cannabinoids (endocannabinoids), and enzymes responsible for the synthesis and degradation of endocannabinoids. In this symposium, recent evidence will be presented that suggests that the endocannabinoid system has important roles in signalling of rewarding events. 1) Dr. Fattore will present self-administration data showing that cannabinoids agonists are reinforcing in laboratory animals and will present recent results obtained in an animal model of cannabinoid relapse; 2) Dr. Rodriguez de Fonseca will present data showing that the endogenous cannabinoid system modulates the rewarding effects of other drugs and will focus on the interactions between endocannabinoids and ethanol; 3) Dr. Errami will present data showing that cannabinoids participate in the control of food intake by interacting with the central serotonin system and hypothalamic peptides.. Whereas this set of findings strongly supports a positive role of endocannabinoids in reward functions, 4) Dr. Panagis will present data showing that, paradoxically, in intracranial self-stimulation procedures, the endogenous cannabinoid system appears to counteract brain stimulation reward. At the end of the symposium, a general discussion will take place on the role of the endogenous cannabinoid system in reward processes and on the possible implications for the development of new medication for the treatment of psychiatric diseases.

Keywords: cannabis, addiction, food, brain stimulation reward, motivation

1. ***Chairman:* Solinas Marcello**, Poitiers, France

2. **Fattore Liana**, Monserrato-Cagliari, Italy: Reinforcing effects of cannabinoids in animal models of addiction
3. **de Fonseca Fernando Rodriguez**, Málaga, Spain: Interactions between cannabinoids and alcohol
4. **Mohamed Errami**, Tétouan, Morocco: Effects of cannabinoids on food intake: interactions with the hypothalamic serotonin and peptides systems
5. **Panagis Giorgos**, Crete, Greece: Paradoxical counter-reward effects of cannabinoids on brain stimulation reward

Symposium III: Cellular regulation of adult-born neurons and their stem cells

Abstract: This symposium will delve into several aspects of the cellular mechanisms underlying adult neurogenesis. New neurons continue to be born in the adult subventricular zone (SVZ) of the lateral ventricle. The SVZ contains neural stem cells, which give rise to neuroblasts migrating along a restricted pathway to the olfactory bulb (OB), where they differentiate into interneurons. All steps of this process are submitted to different forms of cellular regulation. Neural stem cells are regulated in their proliferation, differentiation and self-renewal properties. Nathalie Spassky is interested in the involvement of primary cilia in these processes. Afsaneh Gaillard will show how neuropeptide Y can regulate proliferation of these cells but also change their destiny by diverting them from their regular pathway of migration to the striatum. The differentiation of neurons is itself a regulated process. Isabelle Caillé is interested in the involvement of the Fragile X Mental Retardation Protein, the protein which is absent in the Fragile X syndrome, in the differentiation, integration and plasticity of the newly-formed interneurons into the OB network. Constance Scharff works on adult neurogenesis in the bird brain, the system on which the seminal studies of adult neurogenesis were carried on. She will show how adult-born neurons of a song control nucleus are preferentially integrated into clusters, thus raising the intriguing possibility that intercellular communications are necessary for cell survival.

This symposium will thus bring new information about different levels of cellular regulation of adult neurogenesis from neural stem cell proliferation to neuron differentiation and survival.

Key words: Adult neurogenesis, neural stem cell, subventricular zone, olfactory bulb

1. **Chairman: Caillé Isabelle**, Paris, France
2. **Spassky Nathalie**, Paris, France: Cellular and molecular ciliary mechanisms underlying the proliferation and differentiation of adult neural stem cells
3. **Gaillard Afsaneh**, Poitiers, France: Neuropeptide Y stimulates neurogenesis in the adult subventricular zone and promotes migration of newly generated neurons to the striatum
4. **Isabelle Caillé** (chairman): The fragile X mental retardation protein regulates the integration and plasticity of adult-born olfactory bulb interneurons
5. **Scharff Constance**, Berlin, Germany: Recruitment of adult-born new neurons into neuronal clusters in 'cortical' song control region.

Title of symposium IV: Development of the cerebellum: from neurogenesis to axon guidance and dendritic differentiation

Abstract: Brain complexity develops through a succession of transitions between cell states: from the specification of the different types of neurons and glia to their acquisition of mature phenotypic traits. The cerebellum is a classical model in which all the different open questions concerning brain development have been addressed. The scope of the present symposium is to present original data concerning some of these fundamental questions such as: what controls midline crossing by some precerebellar axon populations, do glial cells and neurons have common progenitors, how neurons acquire the specific form of their dendritic tree? C. Sotelo

will first present the classical framework of cerebellar development. A. Chedotal will present how conditional inactivation of the Robo3 guidance molecule in precerebellar neurons affects their projection in the cerebellum and how it affects motor function. M. Wassef and F Rossi will describe the origins of the interstitial cerebellar white matter progenitors that give rise to interneurons and oligodendrocytes and the control of their differentiation. I. Dusart will illustrate how transcription factors control early stages of Purkinje cell dendritic differentiation.

The participants have deployed a variety of experimental approaches (mouse genetics technology, in vivo transplantation, lentiviral approaches...) to understand cerebellar development that should be of interest to all developmental neuroscientists. This symposium should complete the well known scheme of cerebellar development provided a century ago by integrating several new molecular and cellular aspects.

Keywords: cerebellum, axon guidance, neurogenesis, dendritic differentiation

1. ***Chairman: Sotelo, Constantino***, Alicante, Spain
2. **Chedotal, Alain**, Paris, France: ROBO3: A genetic tool to study the development and function of precerebellar neurons
3. **Wassef, Marion**, Paris, France: Origin and Mash1 dependence of the precursors of GABAergic interneurons and glia in the embryonic cerebellum
4. **Rossi, Ferdinando**, Turin, Italy: The genesis of cerebellar GABAergic interneurons
5. **Dusart, Isabelle**, Paris, France: Transcription factors involved in the development of Purkinje cell dendrites

Symposium V: CHEMOKINES: new modulators in brain and pituitary functions

Abstract: Chemokines are small secreted proteins belonging to the cytokine family which were initially discovered for their chemoattractant properties for immune cells. Recently it was shown that chemokines and their GPCR receptors can be expressed in the brain by both glial cells and neurons, opening up a new era of research in brain research. Thus chemokines have been shown to regulate neural stem cell migration during brain development, modulate neuronal excitability, hormone release and to play a key role in the pathogenesis of various neurodegenerative diseases and pain. Some of these recent advances of chemokine functions will be highlighted in this broad appeal symposium which aims to introduce this emerging field in Neuroscience. S. MBAREK will illustrate the importance of the chemokine SDF1/CXCL12 in an animal model of adaptation to heavy metal pollution and desertic conditions on drinking behaviour. T. FLORIO will present evidence for the role of chemokines on pituitary functions. S. MELIK PARSADANIANTZ will show the importance of the chemokine MCP1/CCL2 in neuropathic pain, and finally W. ROSTENE will examine the implication of chemokines in the modulation of brain dopamine neurons involved in locomotion and addiction.

Keywords: Chemokines, Pain, Environment, Neurodegenerative diseases, Neuroendocrinology

1. ***Chairman: Rostene William***, Paris, France
2. **Mbarek Sihem**, Sidi Thabet Tunis, Tunisia: Implication of SDF1/CXCL12 in drinking behaviour
3. **Florio Tullio**, Genova, Italy: Chemokines and pituitary functions
4. **Melik Parsadaniantz Stéphane**, Paris, France: Chemokines and pain
5. **Rostene William**, Paris, France: Chemokines and regulation of central dopaminergic systems

Symposium VI: Cocaine and brain plasticity: new paths for treating addiction

Abstract: Cocaine abuse places an enormous economical and social burden on society. The purpose of this symposium is to report important new advances that couple our understanding

of the behavioural effects of cocaine with its anatomical, biochemical and molecular correlates. Four established investigators will integrate their most recent discoveries in neuronal intracellular signalling to delineate the molecular mechanisms that participate to cocaine addiction.

Dr. Fumagalli (Italy) will show the role of BDNF in the action of cocaine in conjunction with the effects of stress on cocaine-induced BDNF-mediated intracellular signaling activation. Dr. Valjent (France) will present recent findings from mice knockout studies on the upstream modulators of psychostimulant-induced ERK pathway and the consequences of this activation in the acquisition of behavioral effects of cocaine. Dr. Gainetdinov (USA) will discuss how regulation of G-protein-coupled receptor function and/or activity affect cocaine addiction, by employing animals with knockout of beta adrenergic receptor kinases or beta-arrestins. Dr. Beaulieu (Canada) will address how Akt/GSK3 signaling responses contribute to the effects of cocaine and other psychostimulants.

By dissecting out specific candidates and intracellular pathways, this symposium will uncover novel, and previously unappreciated, molecular mechanisms underlying cocaine addiction in an attempt to define new paths to treat addiction.

Keywords: psychostimulants, neurotrophic factors, MAP Kinases, G-protein coupled receptors

1. **Chairman 1: Fabio Fumagalli**, Milan, Italy
2. **Chairman 2: Emmanuel Valjent**, Stockholm, Sweden
3. **Fabio Fumagalli**, Milan, Italy: Stress and cocaine mutually interact to modulate the activation of BDNF intracellular signaling
4. **Emmanuel Valjent**, Stockholm, Sweden: Role of the ERK signaling pathway in the actions of psychostimulants
5. **Raul Gainetdinov**, Durham, United Kingdom: G-protein coupled receptor desensitization machinery and cocaine actions
6. **Jean-Martin Beaulieu**, Beauport (Québec), Canada,: Akt/GSK3 signaling in the effects of psychostimulants

Symposium VII: How does my brain decide? From decision to regret

Abstract: All animals have developed rapid and efficient decision-making processes to solve complex and conflicting, sometimes life-threatening, choice situations. These processes have reached an unprecedented degree of complexity in both nonhuman primates and humans, perhaps at the price of an increased vulnerability to decision-making dysfunctions, as observed in several mental and personality disorders. The goal of the proposed symposium is to present recent research from leading neuroscientists on the neural basis of normal decision-making. Hagai Bergman (INMED, Marseille, France) will present recent evidence showing that the primate basal ganglia asymmetrically encode the values of rewarding and aversive events. Thomas Boraud (UMR CNRS 5227, Bordeaux, France) will formulate a novel concept of action selection that does not postulate the existence of a central supervisor, as in traditional models, but a direct competition between motor effectors within the cortex-basal-ganglia loop. Ahmed Moustafa (Rutgers University, Newark, USA) will discuss recent neurocomputational models of the basal ganglia and how they approximate real-life action selection and execution. Finally, Angela Sirigu (UMR CNRS 5229, Lyon, France) will focus her talk on uniquely-human, higher-order decision processes, such as counterfactual reasoning and regret, and show how these processes are implemented in the human orbitofrontal cortex.

Keywords: dopamine – orbitofrontal cortex – basal ganglia – action selection – neurocomputation.

1. **Chairman: Serge Ahmed**, Bordeaux, France
2. **Hagai Bergmann**, Marseille, France: Asymmetric Evaluation of Values in the Basal Ganglia

3. **Thomas Boraud**, Bordeaux, France: Decision making as a competition mechanism in the cortex-basal ganglia loop circuits: electrophysiological and fMRI evidences
4. **Angela Cirigu**, Bron, France: The role of the ventromedian prefrontal cortex in decision making and for experiencing the emotion of regret
5. **Ahmed Moustafa**, Newark, NJ, USA: On neurocomputational models of the basal ganglia

Symposium VIII: Adult-born hippocampal neurons and memory

Abstract: The dogma of “once the development of the central nervous system ended, generation of neurons was impossible” has been challenged by the discovery that new neurons are created in specific regions of the adult mammalian brain. One of these regions is the dentate gyrus of the hippocampus, a key structure in memory. This symposium will concentrate on the functional role of adult-born hippocampal neurons in memory. It will bring together recent data of the reciprocal inter-relationship between neurogenesis and learning (Laroche, Dalla), of the qualitative contribution of new neurons to memory (Abrous) and of the inflammation impaired generation of new neurons in memory deficits (Petry).

Keywords: adult, neurogenesis, hippocampus, memory, plasticity

1. **Chairman: Abrous DN**, Bordeaux, France
2. **Dr S Laroche**, Orsay, France: Synaptic plasticity, neurogenesis and memory
3. **Dr C Dalla**, Athens, Greece and New Jersey, USA: Associative learning enhances the survival of new neurons in the male and female hippocampus
4. **Dr Abrous DN**, Bordeaux, France: Adult-born neurons and spatial relational memory
5. **Petry Klaus, PhD**, Bordeaux, France: Experimental Autoimmune Encephalomyelitis induces spatial learning deficits associated with inhibition of hippocampal neurogenesis

Symposium IX: Mormyrid fishes in myth, membranes and molecules

Abstract: The Nile is home to many mormyrids, which are weakly electric fishes, and to the electric catfish, *Malapterurus electricus*, which is strongly electric. The mormyrids use their electric organs to probe their environment and to communicate with conspecifics. The electric catfish's much stronger discharge is used to stun prey and repel predators. The ancient Egyptians utilized the Nile fishery as a major food source, and depictions of fishing and fish including mormyrids and *Malapterurus*, which do not appear to have changed in the last ~3000 years, are common in tomb freezes and paintings. The classical fish, *Oxyrhynchus*, and a catfish, *Phagrus*, probably *Bagrus sp.*, figure in the Osiris legend, possibly because of their ability to shock (Brier). Although not known to be electric by contemporary Egyptian fishermen, electric discharges of modest size mormyrids are clearly perceptible. While *Bagrus sp.* are not electric, the electric catfish produces a painful shock even when nearly dead, and the two may have been confused. Further presentations will describe how the cells of the electric organ generate the organ discharge and how the organs are controlled (Bennett). In electric fishes, electric organs express one Na channel isoform and skeletal muscles another, whereas nonelectric fishes use both in muscle (Zakon). Active electroreception of the environmental impedance involves numerous specializations in the electrosensory lobes that are beginning to account for the remarkable sensitivity (Maler). The Nile fishes provide an intellectual tour from ancient history, field biology, gene expression in excitable membranes and systems neuroscience.

Keywords: Mormyridae / electric fish / electric organ / electrosensory system / Osiris

1. **Chairman: Michael V L Bennett**, Bronx, NY, USA
2. **Prof. Bob Brier**, Brookville, NY, USA: Electric fish and the ancient Egyptian Myth of Osiris
3. **Michael V L Bennett**, Bronx, NY, USA: How mormyrids can shock you

4. **Harold Zakon**, Austin, Texas USA: Modified gene expression in electric organs
 5. **Leonard Maler**, Ottawa, ON, Canada: How fish sense electric fields
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Symposium X: Neural dynamics, learning and functional recovery

Abstract: Complex neural dynamics are at the basis of information processing in the brain, and allow the emergence of intelligent behaviour. Neural dynamics allows us and many other animal species to acquire new perceptual and cognitive skills, to keep in memory tremendous knowledge, and overall to develop new behavioural capacities. These neural changes occur with experience, i.e. practice of a task or repeated exposure to an event, and are crucial for the survival of animal species which need to adapt to new environmental constraints. The dynamics of neural activity is also at the basis of the amazing brain capacity to recover function after injury. But such a complex problem is of course difficult to investigate, for the neural changes during learning or after injury, occur at various spatio-temporal scales. This symposium focuses on these two main domains of neural plasticity, learning and functional recovery. It puts together the contribution of experimental and theoretical approaches of the mechanisms of neural dynamics. Two speakers will address the neural changes during a particular type of learning termed associative learning (Dr. A. Brovelli) and in relation with recovery of hearing after cochlear implants (Dr. P. Barone). These studies use functional imaging techniques to demonstrate that brain networks involved in perceptual processing, or in higher order functions such as linking sensory information with behaviour based on reinforcement, undergo dynamical changes. The other two speakers use computational approaches to understand the neural dynamics in large populations of neurons and in large networks. Dr. G. Deco uses a computational approach to model the dynamics of neuronal networks of attention, whereas Dr. V. Jirsa considers the neural dynamics during what is typically termed “the resting period”. Both of these studies use computational modelling built on architecture derived from the brain’s structure and connectivity. Overall, the symposium emphasizes the need to develop computational frameworks for understanding the relations between different levels of investigation of neural dynamics.

Keywords: Neural plasticity; cerebral cortex; Brain imaging; modelling

1. **Chairman: Driss Boussaoud**, Marseille, France
 2. **Andrea Brovelli**, Marseille, France: Learning from own and others’ actions: a fMRI study
 3. **Gustavo Deco**, Barcelona, Spain: A Computational Neuroscience Approach to Attention, Memory, and Decision-Making
 4. **Viktor Jirsa**, Marseille, France: What does the brain do when it does nothing?
 5. **Pascal Barone**, Toulouse, France: Brain imaging and recovery of speech comprehension in cochlear implanted deaf subjects
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Symposium XI: Comparative Neuroendocrinology: novel neurochemical systems

Abstract: In the history of neurotransmission, new actors like neuropeptides or even new concepts like co-transmission often arose from neuroendocrine field before being accepted for the whole brain. Conversely, neuroendocrine regulations have received new insight from the discovery of novel chemical signalling in the central nervous system. Both movements are still present in recent research and benefit particularly from comparative approaches. Recent examples will be illustrated in this symposium involving five groups from Mediterranean or related countries:

After introduction by A. Calas (France), JM Conlon (UAE) will show how many species of frogs synthesize in their skins a wide range of neuroendocrine and cytolytic peptides that are released into skin secretions in high concentrations and how the traditional assumption that these peptides serve to protect the animal against predators and pathogenic microorganisms should be revisited.

Then H. Tostivint (France) will develop the comparative aspects of urophysial peptides: Urotensin II (UII) is a neuropeptide which has been initially characterized from the urophysis of fish. Subsequent studies have shown that UII exists in all vertebrate classes. Recently, the occurrence of a second UII isoform, named URP, has been reported. This talk will describe the evolutionary history of the UII gene family.

In the third talk, MF. Franzoni (Italy) will illustrate the role of endocannabinoid system in the control of energy intake in bonyfish and its interplay with other orexigenic/anorexigenic factors.

Finally, M Tena-Sempere (Spain) will expose recent discoveries on a new family of neuroendocrine peptides (kisspeptins). He will show that kisspeptins seemingly constitute a *first* and *continuous kiss* that is essential to switch on and keep in motion key aspects of reproductive maturation and function in mammals, from rodents to primates including humans.

Keywords: Comparative neuroendocrinology; novel neuromediators

1. **Chairman: André CALAS**, Bordeaux, France
2. **J. Michael Conlon**, Al-Ain, United Arab Emirates: Neuroendocrine and cytolytic peptides in frog skin: what do they all mean?
3. **Hervé Tostivint**, Paris, France: Molecular evolution of the urotensin II gene family
4. **Maria Fosca Franzoni**, Torino, Italy: Role of the endocannabinoid system in the control of bonyfish feeding response
5. **Manuel Tena-Sempere**, Córdoba, Spain: The kisspeptin system as essential player in the neuroendocrine control of reproduction in mammals

Symposium XII: Physiological involvement of neurogenic niches: from stem cells to clinics

Abstract: The existence of neural stem cells (NSC) in adult mammals is demonstrated by primary culture of dissociated adult tissue in the presence of the specific mitogens EGF and bFGF. This assay produces growing spherical masses of cells that display seminal capacities for proliferation, self-renewal and differentiation into the three characteristic cell types of nervous tissue, along with resistance to tumorigenesis. *In vivo*, neurogenesis *per se*, *i.e.* production of new neurons, occurs only in a small subset of NSC-endowed structures named “neurogenic niches”, the best known of which are the olfactory bulb, fed through migration from the subventricular zone NSCs, and the hippocampus. A decade of investigations has led to the concept that the primary role of mammalian neural stem cells *in vivo* is not post-lesional repair but experience-dependent plasticity, such as during memory processes in the hippocampus (Speaker 1). The production of oligodendrocytes from NSC is still more restricted than neurogenesis. Functional recovery from demyelinating lesions can though occur under multimodal sensory overstimulation or specific pharmacological treatment of adult rodents (Speaker 2). These discoveries raised high hopes for clinical nervous tissue repair. For this purpose, the major challenge now consists in identifying the nature of tissue-specific extracellular signals that determine *in vivo* rate and lineage commitment of NSC progeny. This issue begins to be successfully addressed by combining pharmacological and genetic imaging tools (Speaker 3). Expected application in regenerative medicine is illustrated by successful post-lesional autologous transplantation of olfactory mucosa-derived stem cells in human patients (Speaker 4).

Keywords: neuroplasticity - intercellular communication - cell renewal – neural repair - neurogenesis

1. **Chairman 1: Moyse Emmanuel**, Marseille, France
2. **Chairman 2 : Najimi Mohamed**, Beni-Mellal, Maroc
3. **Rampon Claire**, Toulouse, France: Adult hippocampal neurogenesis: functional implications for memory processing.

4. **Cayre Myriam**, Marseille, France: Mobilization of endogenous neural stem cells for myelin repair
5. **Malva Joao**, Coimbra, Portugal: Regulation of adult neural stem cells by neuroactive messengers
6. **Feron François**, Marseille, France: The use of human olfactory mucosa stem cells in regenerative medicine.

Symposium XIII: NeuroGenomics, Discovering New Genes and Pathophysiological Mechanisms with Phosphoproteomics and Transcriptomics

Abstract: Discovery of protein function within signaling networks reveals therapeutic targets for controlling neuroendocrine cell growth and secretion, and enhancing cell survival in inflammatory and neurodegenerative disease. Protein targets are ‘discoverable’ in multiple ways—through identifying loss- and gain-of-function phenotypes, by assignment to signaling networks that convey apoptotic, proliferative, secretory or plasticity signals to the cell nucleus, or through their direct roles in cellular effector function. Youssef Anouar will describe how gene products selected for their differential regulation during hormonal signaling are systematically assessed for function and pharmacology using microarray and siRNA technologies. Michael Comb will describe the discovery of the growth factor phosphoproteome in human cells and its implications for a comprehensive understanding of neuroendocrine signaling and its regulation. Maria Malagon will discuss the uses of bioinformatics and differential display to identify neuroendocrine function in paralogous protein families that illuminate mechanisms of secretion and their control points. Nancy Gough will describe bioinformatic and information management tools and practices that are accelerating the ability of the individual research laboratory to harness this new information to enhance experimental design, interpretation, and impact; and potentially create a new pharmacology for cell signaling. This symposium aims to illustrate how transcriptomic, proteomic and phosphoproteomic approaches to the discovery of novel secretory and signaling components can be integrated into a bioinformatic analysis of neuroendocrine response networks underlying normal function, homeostatic adaptation, and dysfunction in disease. It is relevant to all practitioners of cellular and molecular neuroscience working in vitro and in whole animals.

Keywords: phosphoproteomics ; PhosphoScan ; expression profiling ; neurogenomics ; cell signaling

1. **Chairman: Lee E. Eiden, Ph.D.**, Bethesda, MD, USA
2. **Youssef Anouar, Ph.D.**, Mont-Saint-Aignan, France: From microarray to siRNA-- Systematic discovery of novel, functionally relevant proteins related to control of calcium in secretory cells
3. **Michael J. Comb, Ph.D.**, Danvers, MA, USA: Mapping phosphoprotein network activation in cellular responses to growth hormone signaling.
4. **Maria M. Malagon, Ph.D.**, Cordoba, Spain: Bioinformatics and differential display reveal novel neuroendocrine functions for paralogous pituitary golgin proteins
5. **Nancy R. Gough, Ph.D.**, Washington, D.C., USA: Science’s signaling: A global bioinformatics network and its role in signal transduction research and drug discovery.

Symposium XIV: Neuroimaging in Pediatrics, Neurology and Psychiatry: from clinical phenomenology to neurobiological phenotypes.

Abstract: Most neurological and psychiatric disease etiology is multifactorial (Tsuang and Faraone 1995) and does not follow a simple Mendelian mode of inheritance (Gottesman and Shields, 1967; McGue and Gottesman, 1989). Thus, clinicians and neuroscientists are faced with complex disorders. Hereditary multifactorial disorders are complex due to imprinting, mitochondrial inheritance, phenocopies, genetic heterogeneity, variable clinical expression,

age at onset, and incomplete penetrance (Dean, 2003). These complicating factors conspire to make brain disorders among the most daunting challenges of modern neuroscience and genetics. Indeed, the complexity of the human brain -approximately 70% of all genes are expressed in brain (Hariri and Weinberger, 2003)- requires an effort to “de-puzzle” this puzzle into empirical biological subprocesses. From this perspective, neuroscience researchers are looking at intermediate phenotypes that are more closely related to genotype than behavior (Castellanos and Tannock, 2002). Phenotypes play a role in discovering alternative controlled quantitative indices of disease liability, that predict the risk of disease. In addition, it may provide new targets for the early intervention/prevention. For example, blood pressure and cholesterol levels have proved fruitful as endophenotypes for heart disease (Almasy and Blangero, 2001). Several studies have commented on the primordial role intermediate phenotypes may play in hereditary complex brain disorders and the role neuroimaging could play in searching for these potential biomarkers. For example, In 2003, Hariri and Weinberger stated that neuroimaging studies have established important physiological links between functional genetic polymorphisms and robust differences in information processing within distinct brain regions and circuits that have been linked to the manifestation of various disease states such as Alzheimer's disease, schizophrenia and anxiety disorders. In their review on intermediate phenotypes and genetic mechanisms of psychiatric disorders, Meyer-Lindenberg and Weinberger, (2006) have stated that functional and structural neuroimaging is bringing about a conceptual change in the way in which biological intermediate phenotypes are viewed and pursued in psychiatry and behavioral genetics by enabling a previously inaccessible level of biological characterization and validation of genetic effects. In essence, neuroimaging subsumes various modalities of data acquisition, for example functional magnetic resonance imaging (fMRI), anatomical magnetic resonance imaging (aMRI) and diffusion tensor imaging (DTI), each of which provides unique but complementary information about brain function, structure and connectivity. Combining such modalities with genetic, neuropsychological and clinical information may have a primordial role in “de-puzzling” some of the neurological and psychiatric diseases puzzle, hence paving the way to early diagnosis and the development of more precisely tailored medications.

1. ***Chairman: Dr. Boualem Mensour***, Montreal, Quebec, Canada
2. **Dr. Adrianna Mendrek**, Montreal, Quebec, Canada: Sexual dimorphism in brain structure and function: General population versus neuropsychiatric disorders
3. **Dr. Adham Mancini**, Montreal, Quebec, Canada: Functional and anatomical magnetic resonance imaging: From Pediatrics to Psychiatry
4. **Dr. Stephane Potvin**, Montreal, Quebec, Canada: Substance abuse in schizophrenia: Neurobiological correlates
5. **Dr. Cherine Fahim**, Montreal, Quebec, Canada: Anatomical magnetic resonance imaging: from neurology to pedo-psychiatry

Symposium XV: Neuro-immune interactions in health and disease

Abstract: The classical view of the immune and the nervous systems as separated entities underlying the concept of the brain as an immune-privileged site has been modified. Thus, it has become apparent the two systems share several signalling molecules and their receptors. Like in the nervous system, synapses can form between immune cells, and the molecular interplays in recognition between the two faces of the synapse – immune versus neural - are under intense studies. In addition, both innate and adaptive immune responses can be elicited within the nervous system to eliminate infectious agents, but special mechanisms prevail to prevent permanent damage to irreplaceable neurons.

This symposium will high-light modern concepts on ion channels expressed both in neurons and in immune cells, and how these can be novel targets for treatment of neuro-immune disorders. How pathogens can be controlled to persist in neurons and how molecules thereby

released can affect synapses to cause functional disturbances such as pain will be discussed. Recent studies, which indicate that molecules released during immune responses can affect synaptic activities in neuronal circuits and play a role in the pathogenesis in epilepsy, will also be covered. Although most studies have dealt with immune cell effects on the nervous system, it is clear that there are bi-directional interactions. The last presentation of the symposium will, as an example of this, be devoted to how the brain can control the immune system under stress.

Keywords: Brain, immune system, ionic channels, epilepsy, virus, stress

1. **Chairman: Krister Kristensson**, Stockholm, Sweden
2. **Rym Benkhalifa**, Tunis, Tunisia: The Neuro-immune dialogue: ion channels as targets for therapy
3. **Krister Kristensson**, Stockholm, Sweden: Control of pathogens in the immune-privileged neuron
4. **Pr Chahnez Charfi Triki**, Sfax, Tunisia: Neuro-immune interactions in epilepsy
5. **Yasser El-Wazir**, Ismailia, Egypt,: Brain control of the immune system under stress

Symposium XVI: Neuronal loss by programmed cell deaths: new players in neurodegenerative diseases

Abstract: Aging-related neurodegenerative diseases represent one of the main health problems in the world because of the over-all increase of elderly population. Discovery of apoptosis, considered until recently as a sole programmed cell death (PCD), raised much hope for intervention in neurodegenerative diseases. However, inhibiting apoptosis turned out to be not as efficient as expected. It is currently thought that the reasons behind this inefficiency might lie in developmentally-regulated inhibition of apoptosis in adult neurons. Peripheral hormones such as leptin emerge as putative regulators of neuronal differentiation-associated PCD.

Morphological and biochemical features of neuronal death in degenerative diseases of the adult nervous system are reminiscent of the recently discovered new PCD phenotypes: apoptosis-like death and programmed necrosis. The main mediator of these PCDs is Apoptosis-Inducing Factor (AIF), which after calpain-mediated cleavage, translocates from mitochondria to nucleus to trigger these PCDs. However, although inhibition of AIF function appears more efficient than blocking apoptosis by caspase inhibition, this still does not completely prevent neuronal death. What are then the additional effectors of neuronal cell death? Activation of lysosomal enzymes such as cathepsins, mediates transformation of a serine protease inhibitor LEI into an endonuclease, L-DNase-II which is involved in DNA fragmentation and cell death in the absence of caspase- and calpain activation. Although understanding how this process is controlled and what are the exact impacts of L-DNase-II activation on the phenotype of cell death remain to be established, AIF and L-DNase-II appear as new effectors of neuronal death that might become attractive therapeutic targets.

Keywords: Programmed Cell death (PCD), neurodegenerative disorders, non-apoptotic neuronal death, programmed necrosis, Apoptosis Inducing Factor (AIF)

1. **Chairman: Slavica Krantic**, Marseille, France
2. **Remi Quirion**, Montreal, Canada: Cell death problem in neurodegenerative diseases
3. **Slavica Krantic**, Marseille, France: Regulation of neuronal death in the course of differentiation: peripheral hormones as the new players
4. **Santos A. Susin**, Paris, France: Programmed cell death: apoptotic versus necrotic
5. **Alicia Torriglia**, Paris, France: DNase II: a comutator between apoptosis and alternative PCD

Symposium XVII: Neuron-glia communications in the normal and pathological brain

Abstract: Growing evidence obtained from several groups has demonstrated the existence of bidirectional communication between astrocytes and neurons. It has been shown that astrocytes respond to synaptic activity with intracellular calcium elevations, and that this might trigger the release of intercellular signaling molecules –called gliotransmitters- that can influence neuronal excitability and synaptic transmission. These results have led to the concept of the Tripartite Synapse, which represents a novel view of the synaptic physiology in which astrocytes are key partners of neuronal synaptic elements.

A great effort is being made to identify the molecular and cellular mechanisms underlying the release of gliotransmitters, and their physiological consequences on neuronal excitability as well as synaptic transmission and synaptic plasticity. These issues will be covered by the present proposal. Specifically, Stéphane Oliet will discuss the contribution of glial cells to NMDA receptor activity through the release of D-serine.

Claudia Verderio will present data on an unconventional mechanism of secretion from astrocytes, based on release of extracellular vesicles such as exosomes. These microvesicles can represent a cargo system for trophic/signaling molecules involved in intercellular communication.

Alfonso Araque will discuss the existence of endocannabinoid-mediated neuron-astrocyte communication, its physiological consequences on synaptic transmission and on long-term potentiation of transmitter release.

Robert Zorec will talk about reactive astrocytes which develop after trauma or during neurodegeneration and how the traffic of several subcellular organelles is affected in reactive astrocytes, which may likely contribute to the detrimental influences on neurons in trauma and in neurodegeneration.

Key words: Astrocyte, synapse, vesicle, D-serine, endocannabinoids

1. **Chairman: Mothet Jean-Pierre**, Bordeaux, France
2. **Oliet Stéphane**, Bordeaux, France: contribution of glial cells to NMDA receptor activity
3. **Verderio Claudia**, Milano, Italy: Astrocyte-derived microvesicles: possible role in intercellular communication
4. **Araque Alfonso**, Madrid, Spain: Astrocytes Regulate Synaptic Transmission
5. **Zorec Robert**, Zaloška, Slovenia: Reactive Astrocytes, Vesicle Traffic and Regulated Exocytosis

Symposium XVIII: Neuroprotective strategies and their application into the clinic

Abstract: Because of the ageing of the world population, the importance of neurodegenerative diseases is increasing dramatically.

Adenosine and adenosine A_{2A} receptors for their role in neurodegeneration and neuroprotection phenomena, are emerging as new target for the treatment of pathologies like ischemia, Huntington's and Parkinson's diseases. Furthermore, preclinical results show how the neuroprotection afforded by A_{2A} receptors blockade combines neuronal and neuro-inflammatory actions, suggesting a broad range of action for these receptors.

This symposium will debate the therapeutic potential of A_{2A} receptors in different neurodegenerative conditions through a multi-disciplinary approach to understand its functioning (control of cell death, neuronal excitability and plasticity, neuron-glia communication and neuro-inflammation). The symposium proposal spans at many different neuronal levels and involves several scientific disciplines: from molecular biology, behavioral and electrochemical measurements to the clinic. Pedata Felicita (University of Firenze, Italy) will revise the neuroprotective effects of adenosine A_{2A}-receptor antagonists in *in vivo* and *in vitro* models of brain ischemia, with special focus on ischemia-induced microglia activation; Popoli, Patrizia (National Institute of Health, Rome) will critically revise the conflicting data on A_{2A} receptors as a suitable target to develop drugs against Huntington's disease, a chronic

disorder where these receptors seem to mediate both neuroprotection and neurodegeneration; Morelli Micaela (University of Cagliari, Italy) will focus on the mechanisms at the basis of the neuroprotection afforded by A_{2A} receptors in Parkinson's disease, whereas Schwarzschild Michael (MassGeneral Institute for Neurodegenerative Disease, Boston, USA) will discuss on the validity of serum urate, a purine metabolite and potent antioxidant, as a predictor of clinical progression in Parkinson disease.

Keywords: Adenosine A_{2A} receptors, ischemia, Huntington's disease, Parkinson's disease, neurodegeneration

1. ***Chairman: Morelli Micaela***, Cagliari, Italy
2. **Pedata Felicita**, Florence, Italy: Modulation of ischemic brain injury and neuroinflammation by adenosine receptors
3. **Popoli Patrizia**, Rome, Italy: Functions and possible therapeutic relevance of adenosine A_{2A} receptors in Huntington's disease.
4. **Morelli Micaela**, Cagliari, Italy: Inactivation of neuronal forebrain A_{2A} receptors protects dopaminergic neurons in models of Parkinson's disease
5. **Schwarzschild Michael**, Boston, USA: Serum urate as a predictor of clinical and radiographic progression in Parkinson disease

Symposium XIX: Neurotransmitter transporters: Key molecules in synaptic function

Abstract: Once released in the synaptic cleft, neurotransmitters are cleared by enzymatic degradation, diffusion and/or uptake system. This latter is performed using specific transporters and allows the control of the temporal and spatial concentration of released neurotransmitter. These transporters located both at the presynaptic and postsynaptic neurons as well as the surrounding glial cells will adjust the quality of membrane receptor activation and transmission. Besides this documented role, transporters have also been shown to modulate the expression of several elements of the synapse and to be regulated themselves under conditions that alter both intra and extracellular environments. Such recent data remain very interesting in the view of designing therapeutic strategies towards the treatment of pathologies involving these proteins.

The conferences that will be presented in the symposium "Neurotransmitter transporters: Key molecules in synaptic function" will focus on the recent knowledge regarding the behavior of these proteins and on the experimental demonstration of the consequences of their activity and/or expression regulation. As for instance, glutamate transporters activity has recently received much more attention as it is currently established that glutamate uptake is required to prevent glutamate-mediated excitotoxic neuronal injuries and the related pathologies. Famous researchers in this field will present their data obtained from in vitro and in vivo experimental studies in order to highlight the critical role that play these proteins in neurotransmission. The aim of this symposium is to provide a wide but a precise idea regarding the regulation of neurotransmitter membrane transporters and their potential involvement in brain diseases.

1. ***Chairman: Mustapha Najimi, PhD***, Brussels, Belgium
2. **Arturo Ortega, PhD**, Cinvestav, Apartado, México
3. **Guyène Page, PhD**
4. **Takashi Uehara, MD, PhD**, Sugitani, Toyama, Japan
5. **Dr. María A Moro**, Madrid, Spain

Symposium XX: Neurovision: Neuronal Mechanisms of Vision

Abstract: Vision is one of the most prominent and prevalent senses in vertebrates. We will discuss retinal mechanisms leading to specific motion information (Euler), thalamic and cortical processing and the influence of attention (Thiele, Fries), integration of visual information with other sensory modalities to create a unified representation of the external

world (Bremmer) and the sensorimotor transformation of visual information leading to action (Hoffmann).

Keywords: Vision – retina- attention – visuomotor – multimodal

1. ***Chairman*: Klaus-Peter Hoffmann**, Bochum, Germany
2. **Thomas Euler**, Heidelberg, Germany: Retinal direction-selectivity and dendritic processing
3. **Alexander Thiele**, Newcastle upon Tyne, United Kingdom: Acetylcholine contributes to attentional modulation in macaque V1 by muscarinic, not nicotinic receptors
4. **Frank Bremmer**, Marburg, Germany: Multisensory space and motion encoding
5. **Klaus-Peter Hoffmann**, Bochum, Germany: Visuo-motor transformation for eye and arm

Symposium XXI: From Circadian Clock to Human Health

1. **Chairman**: Lakhdar-Ghazal Nouria, Rabat, Morocco
2. **Pévet Paul**, Strasbourg, France: The SCN-endocrine outputs in the multi-oscillatory circadian network
3. **Cooper Howard**, Bron, France: Insights into the presymptomatic phase of Parkinson's Disease: longitudinal study of chronobiological and cognitive deficits in chronic low-dose MPTP-treated monkeys
4. **Ouarour Ali**, Tétouan, Morocco: Daily behavioural rhythmicity and the suprachiasmatic nuclei organization in *Lemniscomys barbarus* : a small diurnal rodent with seasonal reproduction.
5. **Ouichou Ali**, Kénitra, Morocco: Affective responses to different experimental photoperiods in young male Wistar rats
6. **Lakhdar-Ghazal Nouria**, Rabat, Morocco: Circadian rhythms and toxicity
7. **Bentivoglio Marina**, Verona, Italy

Symposium XXII: Dis-inhibition processes in pain sensitization

Abstract: Sensory and pain networks of the spinal cord and trigeminal ganglion are exposed to a balance between excitatory and inhibitory inputs. Dis-inhibition shifts the balance toward excitation, and contributes to network hyperexcitability. Weakened inhibition enables a cross-talk between sensory pathways, which may be the substrate of allodynia characteristic of neuropathic pain. We will consider the alterations of fast ionotropic and slow metabotropic inhibitions on the plasticity of the dorsal horn and trigeminal network, and their overall consequences on pain sensitization.

Daniel Voisin will show that glycinergic dis-inhibition is responsible for dynamic allodynia that turns touch into pain in the trigeminal ganglion. Dis-inhibition activates astrocytes that release D-serine and unmask a NMDA-dependent local, excitatory neural circuit.

Yves de Koninck will present how alterations of chloride homeostasis hampers GABA-A-inhibition in lamina I spinal neurons. He will further show that the transmembrane gradient for chloride ions is actively regulated by BDNF released from microglia after peripheral nerve injury.

Valerio Magnaghi uses GABA-B1 knock-out mice to support the contribution of GABA-B receptors to central nociceptive processing. GABA-B1-deficient mice exhibit morphological and molecular changes in peripheral myelin, specifically affecting nociceptive fibers. The possibility that peripheral alterations contribute to the sensory phenotypes of GABA-B1-deficient mice is further considered.

Marc Landry will show that overexpression of GABA-B binding partners triggers the receptor de-dimerization, and therefore disrupt GABA-B inhibition. He will further indicate how to compensate for this dis-inhibition, and enhance the effectiveness of Baclofen, a GABA-B agonist.

Keywords: Pain, spinal cord, dis-inhibition, glycine, GABA

1. ***Chairman:* Marc Landry**, Bordeaux, France
2. **Daniel Voisin**, Bordeaux, France: How glycine inhibitory dysfunction turns touch into pain
3. **Yves De Koninck**, Beauport, Québec, Canada: Neuroimmune-mediated alteration in chloride homeostasis in neuropathic pain; functional consequences
4. **Valerio Magnaghi**, Milan, Italy: Metabotropic GABA-B receptors in the PNS: role in nociception and myelination?
5. **Marc Landry**, Bordeaux, France

Symposium XXIII: Exploring new therapies for Parkinson's disease"

Abstract: Parkinson's disease (PD) is a neurodegenerative disorder characterized by a progressive loss of dopamine cells in the pars compacta of substantia nigra (SNc), thereby inducing a depletion of dopamine concentration at striatal level. The latter is at the origin of the cardinal motor symptoms, akinesia, rigidity and tremor. The restoration of dopaminergic transmission by Levodopa (L-Dopa) has been used successfully for many years but causes long-term motor complications. In this case, deep brain stimulation (DBS) of the subthalamic nucleus has the potential to help restore the motor function of patients. However, for restrictive conditions, all parkinsonian patients cannot be candidate for this neurosurgical therapy. The present symposium will focus on recent research development opening the way for new therapeutical approaches of PD. An overview on pharmacological and surgical treatments will be developed by a neurologist expert (Jose Obeso) followed by the view of a neurosurgeon (Yasin Temel) who practice DBS and study the interaction between DBS and the serotonergic system. The two other speakers will present their recent data on two different experimental approaches targeting glutamate systems by DBS or by using ligands of metabotropic glutamate receptors (Pascal Salin) and the dopamine D5 receptors (Jonathan Chetrit)

Keywords: Parkinson's disease, Basal ganglia, Deep brain stimulation, glutamate metabotropic receptors, serotonergic system

1. ***Chairman:* Abdelhamid Benazzouz**, Bordeaux, France
2. **Jose Obeso**, Pamplona, Spain: What newer pharmacological and surgical treatments are needed for PD?
3. **Yasin Temel**, Maastricht, The Netherlands: Parkinson's disease, subthalamic nucleus and serotonin
4. **Pascal Salin**, Marseille, France: Targeting glutamate systems in the treatment of Parkinson's disease by deep brain stimulation or ligands of metabotropic glutamate receptors
5. **Jonathan Chetrit**, Bordeaux, France: Subthalamic nucleus D5 receptors: role in the pathophysiology and therapy of Parkinson's disease

Symposium XXIV: The purinergic system as a target for the development of novel drugs for acute and chronic CNS disorders

Abstract: The (adenosine)/P1 and (ATP)/P2 purinergic receptors are widely expressed on neurons and all types of glia throughout the CNS, directly participate to neurotransmission and regulate other neurotransmitters' actions. Owing to these widespread roles, dysfunctions of the purinergic system are present in several kinds of acute and chronic diseases. This symposium will bring together 4 internationally known scientists to critically discuss the state of the art of purinergic signaling in the CNS, in view of the future development of novel psychoactive drugs targeting specific P1 or P2 receptor subtypes. **Gloria Cristalli** (Univ Camerino) will review the medicinal chemistry of these receptors with special reference to the availability (or need) of agonists/antagonists acting as potents and subtype-selective agents.

She will also introduce adenosine/P1 receptors as new potential targets for CNS diseases, a topic that will be extensively discussed in prof. Micaela Morelli's symposium. **Rodrigo Cunha** (Univ Coimbra) will critically revise the literature on the involvement of purinergic receptors in cytotoxicity and neurodegeneration and emphasize the cross-talk among these receptors in modulating brain cell survival and neuroinflammation. **Maria Abbraccio** (Univ Milan) will discuss the "druggability" of P2 receptors in human disorders such as trauma/ischemia, pain, Alzheimer's disease and multiple sclerosis. She will also focus on the new P2Y-like receptor GPR17 that may act as a sensor of brain damage. Finally, **Claudia Martini** (Univ Pisa) will review recent developments on the interactions between purinergic and growth factor receptors (GFR), in view of development of "small" purinergic ligands able to foster brain repair and recovery via the modulation of mitogenic signaling activated by GFR.

Keywords: P2 purinergic receptors, neuroinflammation, growth factor receptors, GPR17 receptor, medicinal chemistry

1. **Chairman: Abbraccio, Maria P.**, Milan, Italy
2. **Cristalli, Gloria**, Camerino (MC), Italy: Purinergic Receptor Ligands: State-of-the Art of the Medicinal Chemistry and Application to Neurodegenerative Diseases
3. **Rodrigo, Cunha**, Coimbra, Portugal: Purinergic receptors, cytotoxicity and neurodegeneration
4. **Abbraccio, Maria P.**, Milan, Italy: Extracellular nucleotides as danger signals in the CNS: new perspectives for brain repair
5. **Martini, Claudia**, Pisa, Italy: Purinergic signaling and cell proliferation and differentiation in a neuronal pheochromocytoma PC12 cell model

Symposium XXV: Interactions of synaptic and intrinsic properties in rhythmic motor activities

Abstract: The neural control and generation of rhythmic movements result from interactions between synaptic inputs and intrinsic membrane properties of neurons. One of the major challenges in integrative neurobiology is to understand the specific roles of ionic conductances in controlling not only cellular properties, but ultimately network properties responsible for these rhythmic movements. Modulation and fine tuning of these ionic conductances by synaptic inputs play a key role in shaping the envelope of the output of the network and in adapting the rhythmic pattern in different conditions. This workshop will address membrane and network properties involved in three rhythmic motor activities: mastication, respiration, and locomotion. We will present an up-to-date overview of the recent findings relative to the role of ionic conductances in the operation of rhythmic motor networks. We hope to attract a general audience because of the prevalent role played by these mechanisms in controlling network activity in several motor behaviors. The 5 speakers will review the specific roles played by ionic currents in spinal and brainstem neurons. New findings are surfacing very rapidly in this exciting field and there is a need to integrate the data from different model systems as well as from different neural networks. A KOLTA will discuss the development and the role of the persistent I_{NaP} current (I_{NaP}) in the brainstem circuits involved in mastication and how this current is modulated by the extracellular concentration of Ca^{2+} and the pattern of incoming inputs. F. BROCARD actually paves the way for a new working hypothesis in which I_{NaP} -dependent pacemaker neurons play a key role in locomotor rhythm generation. He will show that most locomotor-related interneurons have pacemaker properties that critically involve the I_{NaP} . He will also describe how the physiological modulation of I_{NaP} in the locomotor network may shed new light on the cellular mechanisms by which the network can operate at different burst rates. JM RAMIREZ will discuss pacemaker properties in neurons involved in the neurogenesis of respiratory activity. He will describe how the dynamic interplay between ion channels, second messenger

pathways, intracellular Ca^{2+} concentrations, neuromodulators, and synaptic inputs leads to burst generation at the cellular and network level. There will be a discussion of how nonlinearity of bursting activity might enable pacemaker neurons to facilitate the onset of excitatory states and to synchronize neuronal ensembles. R DUBUC will discuss the initiation of rhythmic locomotor activity, in particular the role of the descending reticulospinal system. He will show that calcium mediated sustained depolarizations play a key role in building the descending motor command. The sustained depolarizations result from a calcium activated nonselective cationic conductance (I_{CAN}). He will also address the interactions between synaptic inputs and intrinsic properties in these command neurons and more specifically the role of excitatory glutamatergic inputs. Finally, the role of ionic currents and their modulation in generating the activity of spinal cord circuitry will be addressed by A EL MANIRA. The role of specific K^+ currents in determining neuronal firing, synaptic interaction and locomotor pattern generation will be highlighted. He will also discuss how endocannabinoids released within the spinal locomotor circuitry mediate short- and long-term plasticity of the locomotor activity. Altogether, this workshop will provide a cutting edge review of the critical role of ionic currents in shaping cell and network properties that control vertebrate rhythmic motor behaviors.

Keywords: rhythmic movements, central pattern generators, intrinsic cell properties, network properties

1. ***Chairman: Kolta, Arlette***, Montréal, Québec, Canada: Modulation of INaP mediated bursting in trigeminal main sensory neurons by Ca^{++} and synaptic inputs
2. **El Manira, Abdel**, Stockholm, Sweden: Ion channels and receptors controlling the function and plasticity of the spinal locomotor circuitry
3. **Brocard, Frédéric**, Marseille, France: The locomotor rhythm generation: the pacemaker hypothesis
4. **Ramirez, Jan Marino**, Chicago IL, USA: The Orchestration of the respiratory rhythm: how to integrate multiple modulators and cellular properties
5. **Dubuc, Réjean**, Montréal, Québec, Canada: The co-operation of intrinsic and synaptic properties in brainstem locomotor command neurons

Symposium XXVI: Serotonin neurobiology: new vistas

Abstract: Historically, the serotonergic hypothesis of depression is perhaps one of the most compelling, enduring and best studied disease model in psychiatry. In its original formulation, it postulates a deficit in 5-HT as a primary cause, reversed by antidepressants, which would restore normal function in depressed patients. Indeed, a variety of functional deficits of 5-HT neurotransmission in brain circuits known to regulate emotions, whether primary or secondary, have consistently been associated with aspects of the pathophysiology of MDD. More recently, reports in high risk relatives and MDD patients in remission, suggest that altered serotonergic function may be enduring, persist in remitted patients off medication and rather, represent a vulnerability factor and trait diathesis for MDD.

Serotonin is considered to be crucial for the development of the human and mammalian brain (Gaspar et al., 2003; Cote et al, 2007). An emerging body of experimental evidence has helped gain insight into how dysfunctions of serotonergic tone early in life, may modulate brain pathways development, differentiation and maturation, as well as affect sensitivity to aversive stressors and more generally, emotion regulation in adults. In this evolving model, a disruption in 5-HT homeostasis during critical periods of development, as a result of genetic, environmental, or their interaction, leads to long-term structural and/or functional changes in brain circuits that modulate emotional responses to stress, thereby predisposing vulnerable individuals, in adolescence or early adulthood, in the face of adversity, to maladaptive emotion regulation.

The proposed symposium aims at familiarizing the audience with this emerging new

framework of understanding of serotonergic mechanisms in neuropsychiatric disorders, by inviting speakers, both basic and clinician scientists, whose work illustrates well this change in paradigm.

(1) Dr Gaspar will introduce this workshop and provide an overview of the neurodevelopmental / trophic properties of Serotonin, using molecular techniques and mouse, as template. (2) Vesicular glutamate transporters have first been considered as exclusive markers of glutamatergic transmission. Surprisingly, 5-HT neurons express an atypical vesicular glutamate transporter, named VGLUT3. Dr El Mestikawy will present new data on the role of VGLUT3 in the serotonergic system and on its functional consequences on anxiety. (3) One of the central mechanism of control of the serotonergic system is the somatodendritic 5HT_{1A} receptor, controlling neuronal firing. The 5HT_{1A} receptor is a GPCR, whose transmembrane and intra-cellular trafficking, determines proper signalling and is the subject of multiple molecular regulations. Dr Descarries will provide a summary of his research on agonist-mediated internalization of the somatodendritic 5HT_{1A} receptor, both in vitro and in-vivo. Theoretical speculations about the significance of this process in both health and disease, will be entertained. (4) Finally, Dr Benkelfat will present some recent PET / *in vivo* serotonergic measurements obtained in young adult healthy volunteers, recruited in childhood because of disruptive behaviour at school, illustrating the advantages of studying neurobiological mechanisms in well characterized prospective longitudinal cohort samples.

Keywords: 5HT, 5HT_{1A}, PET neuroimaging, neurodevelopment, impulsivity

1. **Chairman: C Benkelfat**, Montreal, Canada
2. **P Gaspar PhD**, Paris, France: the developmental role of serotonin : lessons from mouse molecular genetics
3. **S ElMestikawi PhD**, Paris, France: expression and function of the vesicular Glutamate transporter (VGlut 3) in serotonergic neurons
4. **L Descarries MD-PhD**, Montreal, Canada: Internalization as a Index of 5-HT_{1A} Serotonin Autoreceptor Sensitivity
5. **C Benkelfat MD**, Montreal, Canada: TPH2 polymorphism, social, psychological and physical adversity during development and brain regional serotonin synthesis capacity in young adult males, followed prospectively since childhood for aggressive behavior at kindergarten

Symposium XXVII: Physiopathology of synaptic plasticity

Abstract: Synaptic plasticity mediates the dynamic changes triggered in neuronal networks by environmental stimuli and individual experiences and is fundamental to memory formation, cognitive and sensory-motor processes. Synaptic plasticity can either be expressed as changes in synaptic strength or as rearrangements of protein composition in the synaptic compartment. Synaptic plasticity impacts on synaptic properties and more generally on the activity of entire neuronal ensembles. A growing body of evidence indicates that pathologic alterations of synaptic plasticity (“synaptopathies”) are also at the origin of numerous brain pathologies.

This symposium will present data obtained in key brains structures (hippocampus, prefrontal cortex, striatum) in both in vitro and ex vivo models using vertical strategies integrating approaches ranging from molecular neuroscience (electrophysiology, real-time dynamic of membrane receptors, Ca²⁺ imaging and biochemistry) to system neuroscience (ex vivo electrophysiology, genetic and behavioral models of psychiatric/neuronal diseases).

The goal of this symposium is to present recent progress uncovering new molecular basis of synaptic plasticity in physiological and pathological conditions and to illustrate how basic research can help set the ground for the development of new therapeutic approaches of synaptopathies and brain pathologies.

Keywords: Synaptic plasticity; neuronal networks; development; maturation; synaptopathies

1. ***Chairman*: Olivier Manzoni**, Bordeaux, France
 2. **Laurent Venance**, Paris, France: Corticostriatal transmission and synaptic plasticity
 3. **Pascale Chavis**, Bordeaux, France: Roles of the extracellular matrix protein Reelin in synaptic pathology and maturation
 4. **Rosa Cossart**, Marseille, France: Neurons orchestrating synapse-driven synchrony in the developing hippocampus
 5. **Laurent Fagni**, Montpellier, France: Synaptopathies linked to glutamate receptor scaffolding proteins
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