8th Annual Canadian Neuroscience Meeting

Presented by:

Canadian Association for Neuroscience

Association canadienne des neurosciences

Press releases and Information for the Media

Annual Meeting: May 25 - 28, 2014

Bonaventure Hilton Montréal, 900 Rue de la Gauchetière Ouest, Montréal, QC H5A 1E4

http://can-acn.org/meeting2014

Public Lectures: May 24th, 3PM (French) and 4PM (English)

Montreal Neurological Institute, Jeanne Timmins Amphitheatre, 3801 University Street, Montreal

http://can-acn.org/2014-public-lecture

Media contact: **Julie Poupart**, info@can-acn.org, 514-912-2405

8th Annual Canadian Neuroscience Meeting

Presented by:

Canadian Association for Neuroscience - Association canadienne des neurosciences

Here you will find general information about our Association, which is an association of neuroscientists dedicated to the promotion of all fields of neuroscience research. Our press releases follow.

CAN-ACN Mission

The purpose of the Canadian Association for Neuroscience is:

1- To promote communication among neuroscientists throughout Canada.

2- To represent the interests of Canadian neuroscientists at national and international levels.

3- To promote research in all disciplines contributing to the understanding of the nervous system.

4- To contribute to the advancement of education in the Neurosciences.

5- To provide for and assist in the dissemination to the general public of the results of current Neuroscience research and its significance in relation to health and disease.

6- To raise funds and to provide income for the above purposes.

The Canadian Association for Neuroscience is a registered **not-for-profit association**.

CAN-ACN Leadership - Executives

President: **Samuel David**, PhD, McGill University

Vice-President (President-Elect): **Douglas Munoz**, PhD, Queen's University

Treasurer: **Ellis Cooper**, PhD, McGill University

Media contact: **Julie Poupart**, info@can-acn.org, 514-912-2405

Secretary: **Katalin Toth**, PhD, Université Laval

Chair of the nominations committee: **Yves De Koninck**, PhD, Université Laval

2014 Meeting Organisation

Chair of the 2014 Scientific Program Committee: **Sheena Josselyn**, PhD, University of Toronto

Co-Chair of the 2014 Scientific Program Committee: **Kurt Haas**, PhD, University of British Columbia

CAN-ACN Administration

Association Secretariat & Conference Management:

De Armond Management Ltd. secretariat@can-acn.org

Marischal De Armond and **Jude Ross**

Communications Director and webmaster:

Julie Poupart, PhD - info@can-acn.org

Membership Information

CAN-ACN membership is open to students, post-doctoral fellows and principal investigators actively engaged in neuroscience research in Canada and around the world.

2014 Public Lectures

Two public lectures, presented in English and in French have been organised on May 24th 2014. They are open to all people interested in neuroscience. Admittance is free.

2014 Press releases

You will find our press releases in the following pages. Our press releases are published on the EurekAlert! Website, the online, global news service operated by AAAS. Press release are available in English and French.

Information about: 2014 CAN-ACN Public lecture May 24th, 4PM

How Life Experiences Impact On Mental Health

Traumatic life experiences can get "under our skin", and influence how we feel and behave. Dr. Michael Meaney will present recent research that helps us understand how life experiences influence brain function and behavior. Recent studies show that life experiences, such as traumatic events or childhood social environment, alter the 'epigenetic signals' on the DNA that control the way genes act. In this way, our genes adapt to meet the daily challenges we face, and thus influences on our mental health.

Professor Meaney's research has revealed how the early life environment influences the development of vulnerability for multiple forms of chronic illness, including mental disorders. His studies show that variations in the early social environment, especially those associated with parental care, can alter the activity of genes that regulate our responses to stress and various forms of learning and memory.

About Dr. Michael Meaney

James McGill Professor, McGill University
Director of the program for the Study of Behaviour, Genes and Environment
Associate Director of the Douglas Institute Research Centre.

A full profile of Dr. Meaney is available here:
http://www.douglas.qc.ca/researcher/michael-meaney

Time: **Saturday, May 24 2014 - 4 PM**

This event will be immediately preceded by a lecture in French by Dr. Gustavo Turecki, at 3 PM - Both researchers will be available for questions.

Venue: Jeanne Timmins Auditorium, **Montreal Neurological Institute**,
3801 University street, Montreal

Free admittance - ALL WELCOME !

Media contact: **Julie Poupart**, info@can-acn.org, 514-912-2405

Information - Conférence publique 2014, 24 mai, 15h

Comment le vécu affecte la santé mentale

Les expériences traumatiques de la vie nous marquent et influencent nos émotions et nos comportements. Le Dr Gustavo Turecki présentera des résultats de recherche récents qui nous aident à comprendre l'influence de l'expérience sur le fonctionnement du cerveau et le comportement.

Les études récentes montrent que le vécu, comme les événements traumatiques, ou l'environnement social durant l'enfance, modifie les «épigénétiques» sur l'ADN, signaux qui contrôlent l'expression des gènes. Nos gènes s'adaptent ainsi aux défis de tous les jours, ce qui affecte aussi notre santé mentale.

Le Dr Turecki vise, par ses recherches, à comprendre comment le cerveau change quand des personnes se sentent désespérées et suicidaires. Il étudie particulièrement comment notre vécu modifie le fonctionnement du cerveau par des processus que l'on nomme épigénétiques.

À propos du Dr Gustavo Turecki
Directeur, Groupe McGill d'études sur le suicide, Institut Douglas
Co-directeur, Banque de cerveaux Douglas –Bell Canada
Chef, Programme des troubles dépressifs, Institut Douglas
Directeur, Réseau québécois de recherche sur le suicide (RQRS)
Professeur, Université McGill

Voir son profil complet:
http://www.douglas.qc.ca/researcher/gustavo-turecki

Heure: **Samedi 24 mai 2014, 15h**

Cette conférence précède celle du Dr. Michael Meaney, en anglais, à 16h - Les Drs Turecki et Meaney répondront ensemble aux questions du public.

Lieu: Auditorium Jeanne Timmins, **Institut neurologique de Montréal**,
3801 rue Université, Montréal

Entrée gratuite - Bienvenue à tous !

Media contact: **Julie Poupart**, info@can-acn.org, 514-912-2405

Press release- Published May 7th 2014-

Eurekalert! link: http://www.eurekalert.org/pub_releases/2014-05/cafn-slb050714.php

Stephanie L. Borgland and Brian E. Chen both winners of 2014 CAN Young Investigator Awards

Awards recognize excellence in neuroscience research in a young neuroscientist

The Canadian Association for Neuroscience is proud to announce that Dr. Stephanie Borgland, from the Hotchkiss Brain Institute at University of Calgary, and Dr. Brian Chen, from the Centre for Research in Neuroscience at McGill University, are both winners of CAN Young Investigator Awards for 2014. Drs. Brian E Chen and Stephanie L Borgland were both judged equally deserving of this distinction, which recognizes excellence in neuroscience research in a young neuroscientist. They will receive their awards during the opening ceremony of the upcoming Canadian Neuroscience Meeting on May 25 2014

Brian Chen: How to build a brain

Dr. Brian Chen seeks to understand how the instructions to wire up a brain are encoded in our DNA. His research uses a combination of high-resolution imaging techniques with advanced molecular genetics to look inside the brains of living animals while their neurons form connections. Dr. Chen's research provides insight into how the brain's wiring is encoded in the DNA, and how these instructions malfunction in mental disorders.

Dr. Chen's research accomplishments demonstrate his ability to address central questions in the field of neuroscience. His research has helped deepen our understanding of the underlying genetic causes of mental retardation, caused by diseases such as Fragile X, Down's and Rett's syndrome, and autism. His important research has been published in high impact journals such as Nature, Cell and Nature Neuroscience.

Media contact: **Julie Poupart**, info@can-acn.org, 514-912-2405

Stephanie Borgland: Why we eat too much

Dr. Stephanie L Borgland studies the neuroscience behind addiction and obesity. Most people that become obese overeat despite the knowledge that the consequences will be harmful. Similarly, a key feature of addiction is the inability to stop drug use despite negative consequences. The mechanisms in the brain underlying abnormally heightened motivation leading to obesity and addiction may be similar. Dr. Borgland and her research team focus on the neural mechanisms that underlie eating for reasons other than hunger.

Dr. Borgland is internationally known for her innovative work in neuroscience and many of her discoveries have been published in top journals in neuroscience (including Nature Neuroscience, Neuron, The Journal of Neuroscience and Biological Psychiatry). Her lab uses a combination of techniques to explore how areas of the brain involved in evaluating reward and motivating behaviour are rewired by consumption of high fat foods. The laboratory has made exciting discoveries on how peptides that signal satiety ("I'm full"), such as insulin and leptin, modulate dopamine neurons involved in reinforcement and motivation. This understanding is of key importance to understand why we start and stop eating.

Full profiles of Drs. Chen and Borgland, including references to representative research publications can be found on the Canadian Association for Neuroscience website, at :

http://can-acn.org/brian-e-chen-2014-can-young-investigator-awardee
http://can-acn.org/stephanie-l-borgland-2014-can-young-investigator-awardee

About the CAN Young Investigator Award

The Canadian Association for Neuroscience Young Investigator Award is given yearly to recognize outstanding research achievements by a young neuroscientist at the early stage of his or her career. The winner is chosen by the CAN Nominations Committee.

The eighth Annual Canadian Neuroscience meeting takes place May 25 - 28 2014 at the Hilton Bonaventure in Montreal. Two public lectures, open to all, will take place May 24th.

Media contact: **Julie Poupart**, info@can-acn.org, 514-912-2405

Press release- **Embargoed until May 25th 2014, 7:00 PM**
http://www.eurekalert.org/emb_releases/2014-05/cafn-pat052114.php

Promising approach to slow brain degeneration in a model of Huntington's disease uncovered

Mechanism uncovered could also help preserve neuron function in Alzheimer's disease, traumatic brain injury and other neurodegenerative conditions

Research presented by Dr. Lynn Raymond, from the University of British Columbia, shows that blocking a specific class of glutamate receptors, called extrasynaptic NMDA receptors, can improve motor learning and coordination, and prevent cell death in animal models of Huntington disease. As Huntington disease is an inherited condition that can be detected decades before any clinical symptoms are seen in humans, a better understanding of the earliest changes in brain cell (neuronal) function, and the molecular pathways underlying those changes, could lead to preventive treatments that delay the onset of symptoms and neurodegeneration. "After more than a decade of research on the pre-symptomatic phase of Huntington disease, markers are being developed to facilitate assessment of interventional therapy in individuals carrying the genetic mutation for Huntington disease, before they become ill. This will make it possible to delay onset of disease," says Dr. Raymond. These results were presented at the 2014 Canadian Neuroscience Meeting, the 8th annual meeting of the Canadian Association for Neuroscience - Association Canadienne des Neurosciences (CAN-ACN), held in Montreal, May 25-28.

The neurotransmitter glutamate has long been known to promote cell death, and its toxic effects occur through the action of a family of receptors known as the NMDARs (N-methyl-D-Aspartate ionotropic glutamate receptors). Unfortunately, treating disorders of the nervous system by blocking NMDARs has not been successful because such treatments have numerous side effects. A recent hypothesis based on work from many scientists suggests that NMDARs located in different regions at the surface of neurons may have opposite effects, which would explain why blocking all NMDARs is not a good treatment option. A synapse is a structure that allows one neuron to connect to another neuron and pass an electrical or chemical signal between them. Many receptors for neurotransmitters are located in synapses, as these are the main area where these chemical signals are transmitted. However, receptors can also be found outside the synapse, and in this case are called extra-synaptic receptors. Many recent studies have revealed that NMDARs located at synapses act to increase survival signaling and promote learning and memory, whereas extra-synaptic NMDARs shut off survival signaling, interfere with learning mechanisms, and increase cell death pathways.

Dr. Raymond and her team were able, by using a drug that selectively blocks extra-synaptic NMDARs early, before the appearance of any symptoms, to delay the onset of Huntington-like symptoms in a

mouse model of the disease. These promising results could lead to new treatment avenues for Huntington patients, and delay the appearance of symptoms. "The drug we used, memantine, is currently being used to treat moderate-stage Alzheimer disease patients. Our results suggest that clinical studies of memantine and similarly-acting drugs in Huntington disease, particularly in the pre-symptomatic stage, are warranted,"says Dr. Raymond.

Extra-synaptic NMDARs have also been shown to be involved in other neurodegenerative diseases, such as Alzheimer disease, and in damage caused by traumatic brain injury and some forms of stroke. These results therefore suggest novel treatment avenues for many conditions in which neurons degenerate and die, a new way to protect neurons before the appearance of symptoms of neurodegeneration.

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This research was supported by: Canadian Institutes of Health Research, Huntington Society of Canada, Cure Huntington Disease Initiative, and Michael Smith Foundation for Health Research.

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For news media only:

Please contact Julie Poupart, Communications Director for the Canadian Association for Neuroscience, for further information, to receive a press pack, or to request an interview with a neuroscientist: info@can-acn.org. Press passes for the Canadian Neuroscience Meeting, May 25-28 2014, in Montreal, are available for accredited journalists.

Presidential Lecture - Scientific Presentation Abstract:

Mechanisms and neuroprotective strategies in neurodegeneration: Huntington disease can lead the way- By Lynn Raymond, University of British Columbia

Evidence indicates that NMDA-type glutamate receptor (NMDAR)-induced synaptic loss and neuronal dysfunction/death contributes to mechanisms underlying certain neurodegenerative diseases and acute neurological insults. Yet, cell signaling downstream of NMDARs can promote cell survival and plasticity as well as excitotoxicity, which may help explain why general NMDAR inhibitors have failed in clinical trials. A new paradigm developed over the past decade suggests that over-stimulation of extrasynaptic NMDARs triggers stress/death pathways whereas physiological activation of those inside the synapse contributes to cell survival, raising the possibility of neuroprotection based on subcellular localization. This idea has been tested in the inherited, predominantly adult onset, neurodegenerative disorder Huntington disease (HD), which manifests as progressive motor, mood and cognitive impairment. Caused by a polymorphic CAG repeat expansion in the HD gene that encodes an enlarged polyglutamine tract in the protein huntingtin, HD is associated with selective neurodegeneration, principally of striatal GABAergic spiny projection neurons (SPN) and cortical pyramidal neurons. Genetically accurate mouse models have facilitated understanding of HD pathogenesis. In one HD mouse model (YAC128), we have shown an increase in number, activity, and downstream signaling of extrasynaptic NMDARs on SPN beginning in the early postnatal period; selective inhibition of these receptors from an early age ameliorates later stage cell death signaling and also improves motor learning and coordination. Moreover, we and others have identified additional synaptic alterations that occur prior to overt motor manifestations. In particular, we have characterized morphological and electrophysiological changes in cortical-striatal co-cultures from HD mice, a simple model system that can serve as a platform for testing therapeutics. Since HD gene mutation carriers can be identified decades before clinical diagnosis, targeting early changes in cortical-striatal synaptic transmission may significantly delay onset of manifest disease.

Copies of recent publications by Dr. Lynn Raymond are available upon request. Please contact Julie Poupart info@can-acn.org.

Press release- **Embargoed until May 27th 2014, 12:00 PM (noon)**
http://www.eurekalert.org/emb_releases/2014-05/cafn-itp052114.php

Investigating the pleasure centers of the brain: How reward signals are transmitted

Research could help devise treatment strategies for psychiatric diseases such as addiction, Tourette's syndrome and obsessive-compulsive disorder

New research presented today by Dr. Jonathan Britt, from McGill University, helps to better understand how reward signals, such as those produced by addictive drugs, travel through the brain and modify brain circuits. Dr. Britt obtained these results using optogenetics, which use light-responsive proteins to study the activation of neural circuits in distinct locations, allowing the researcher to precisely dissect the roles of different neural circuits in the brain. Dr. Britt's studies have helped reveal circuits that are responsible for habitual behavior, which could be suitable targets for pharmacotherapies designed to treat drug addiction. These results were presented at the 2014 Canadian Neuroscience Meeting, the annual meeting of the Canadian Association for Neuroscience - Association Canadienne des Neurosciences (CAN-ACN) which takes place May 25 - 28th 2014.

One of the most immediate effects of drugs on the brain is an increase in the levels of dopamine, particularly in a region of the brain called the nucleus accumbens. Located near the center of the brain, the nucleus accumbens is connected, by intermingled populations of cells, to many other brain structures having roles in pleasure seeking and drug addiction. The nucleus accumbens is recognized as an integration centre for signals coming from many different brain regions, but the precise role of the different connections, and the means of their integration, resulting in specific behaviours, was until recently impossible to dissect. The advent of optogenetics has made it possible to study the various inputs that come from different regions of the brain, and their positive or negative effects on reward seeking, and their role in drug response in mice and rats.

Dr. Britt has characterized some of the ways that the nucleus accumbens integrates dopamine dependent reinforcement signals with environmental stimuli, which depend on a second neurochemical called glutamate. Glutamate-dependent signals to the nucleus accumbens come from many other brain regions, such as the hippocampus, the amygdala, the thalamus and the prefrontal cortex. Understanding how these different brain regions are interconnected will deepen our understanding of motivation, desire, pleasure seeking and addiction. This research is also applicable to the understanding of conditions such as Tourette's syndrome and obsessive-compulsive disorder.

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"Goal-directed behaviour is regulated by large collection of interconnected brain regions. It is important to understanding how these component parts interact with each other in order to devise treatment strategies for psychiatric diseases such as addiction, Tourette's syndrome and obsessive-compulsive disorder," concludes Dr. Britt.

This research is funded by a grant from the Natural Sciences and Engineering Research Council of Canada (RGPIN-2014-05069).

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Original research presentation abstract:
Dissecting the neural circuits underlying motivated behaviours relevant to reward learning and drug addiction, by Jonathan Britt

The nucleus accumbens plays a major role in the generation of motivated behaviour. It integrates dopaminergic reinforcement signals with glutamate- encoded environmental stimuli. Prominent glutamate afferents to the nucleus accumbens come from the hippocampus, amygdala, thalamus, and prefrontal cortex. Pathway-specific activation of these inputs is known to produce distinct behavioral responses, but mechanistic explanations for these pathway-specific effects are lacking. This talk examines the pathway-specific differences in synaptic properties and innervation patterns between these glutamatergic inputs to the nucleus accumbens. While there are important distinctions between these afferent connections, optogenetic stimulations targeted to any of them can reinforce instrumental behaviour. This finding challenges the idea that these inputs encode motivationally- neutral information. Mice will also work to obtain optical manipulations to projections neurons throughout the striatum as well as downstream structures, but, regardless of which basal ganglia nuclei are targeted for self-stimulation, the behaviour is always sensitive to dopamine receptor blockade. This work characterizes some of the fundamental organizing principles of basal ganglia information processing.

Copies of recent publications by Dr. Jonathan Britt are available upon request. Please contact Julie Poupart info@can-acn.org.

Press release- **Embargoed until May 27th 2014, 12:00 PM (noon)**
http://www.eurekalert.org/emb_releases/2014-05/cafn-mtr052114.php

Making the right choices in changing circumstances: Cognitive flexibility in the brain

Multiple brain regions interact and compete to inform decision-making

Choosing what is best is not always simple. Should one choose a small, certain reward, or take risks and try to get a larger reward? New research by Stan Floresco, from the Brain Research Centre at the University of British Columbia sheds light on the brain circuits that interact to help us decide the best strategy to adopt in changing circumstances. These results were presented at the 8th annual Canadian Neuroscience Meeting, taking place May 25-28 2014 in Montreal, Canada.

The studies of Dr. Floresco and his team used rats to show that areas deep inside the brain promote a more visceral bias towards large, but uncertain rewards, while brain regions located in the frontal lobes (which regulate higher order functions such as reasoning and planning), regulate and temper these urges when circumstances show the riskier option may be unlikely to yield reward. "It seems that the more primitive regions of the brain drive impulses to pursue larger rewards, but the frontal lobes take a longer view of the situation and put the brakes on these urges in situations when larger rewards may not be the most profitable ones in the long term", explains Dr. Floresco.

In another study, Dr. Floresco revealed that the activity of dopamine neurons seem to provide the brain with short-term updates of the outcomes of recent decisions that can influence the direction of subsequent ones. "Dopamine neurons show brief increases or decreases in activity when rewards are either received or not. However, we showed that if we turned these neurons off after a rewarded choice, or turned them on after a non-rewarded one, we could, in essence, remote control the decision making of these animals, making them behave as if they did not receive a reward (that they actually did) or vice versa", says Dr. Floresco.

Dr. Floresco also recently published an important paper highlighting the important and until recently underestimated role of another brain region, called the lateral habenula, in decision making. "An emerging view was that this brain region was primarily involved in signalling when something bad occured. Yet, our results show that its function is much more complex. When we shut down neural activity within this region, animals show random patterns of decision making, suggesting that this region plays a key role in promoting decision biases in one direction or another."

Media contact: **Julie Poupart**, info@can-acn.org, 514-912-2405

These results show the dynamic competition that exists between signals coming from different brain regions. The integration of these signals requires cognitive flexibility, which is the ability to react differently, update behavior and make appropriate choices in response to changes in one's environment.

Understanding how these signals are transmitted and act in the normal brain can help explain many neuropsychiatric conditions in which this signalling is defective. Schizophrenia is associated with abnormal activity in many of the same brain regions involved in efficient decision making. Delusions associated with schizophrenia can stem from associating strong emotional response to an innocuous situation. Not associating the right affective importance to pleasurable or aversive stimuli can result in inability to feel positive emotions like pleasure and to feel desire, which is one of the hallmarks of depression. Drug addiction can also be considered as a disease of decision making. «By clarifying the mechanisms through which different brain circuits interact to guide normal decision making, these studies may provide important insight into the brain dysfunction that may occur in these different disorders», concludes Dr. Floresco.

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Scientific Presentation Abstract:

Dopaminergic circuits mediating risk/reward decision biases, by Stan Floresco

Choosing between smaller, assured rewards or larger, uncertain ones requires reconciliation of competing biases towards more certain or riskier options. These conflicting urges reflect an interplay between distributed neural circuits linking the frontal lobes to subcortical regions processing emotional and reward-related information that in turn influence response selection. Each of these regions is interconnected with the dopamine system. Our studies have used a probabilistic discounting task to probe the interactions between these systems in regulating risk/reward decision making. Data will be reviewed showing that subcortical circuitry linking the amygdala and the ventral striatum appears to promote a more visceral bias towards larger, uncertain rewards, whereas prefrontal regions serve to temper these urges when riskier options become less profitable via top-down control over the amygdala. Dopamine D1/D2 transmission within these regions also makes dissociable, yet complementary, contributions to risk/reward judgments, promoting either exploitation of current favorable circumstances or exploration of more profitable ones when conditions change. Dynamic fluctuations in prefrontal and accumbens tonic dopamine transmission appear to encode distinct types of information related to decision making related to changes in reward availability, uncertainty and choice biases. On the other hand, phasic increases and decreases in dopamine activity, regulated in part by the lateral habenula, appear to play a key role in providing short-term information about recent outcomes that bias subsequent choice. These findings provide insight into the dynamic competition between cortical/subcortical circuits that shape decision biases and underlie conflicting urges when evaluating options that vary in terms of potential risks and rewards.

Copies of recent publications by Dr. Stan Floresco are available upon request. Please contact Julie Poupart info@can-acn.org.

Press release- **Embargoed until May 28th 2014, 12:00 PM (noon)**

http://www.eurekalert.org/emb_releases/2014-05/cafn-nrs052114.php

New research shows memory is a dynamic and interactive process

Results could be used to help ameliorate memory problems in older adults

Research presented by Morris Moscovitch, from the Rotman Research Institute at the University of Toronto, shows that memory is more dynamic and changeable than previously thought. Dr. Moscovich's results reveal that important interactions between the hippocampus and the neocortex, two regions of the brain, have different yet complementary roles in remembering places and events. These results highlight that different forms of memories exist in the brain, and that these are encoded in different, but interacting parts of the brain. Dr. Moscovitch proposes a novel theory to explain these interactions, that furthers our understanding of what we remember, and could be useful for treatment and management of people with memory disorders. These results were presented at the 8th Annual Meeting of the Canadian Association for Neuroscience held in Montreal, Canada May 25 to 28th 2014.

By studying how humans remember events and places in the short and long term, and how rodents remember and navigate through familiar and unfamiliar environment, Dr. Moscovitch and others have revealed differences between what they call "episodic memory", which is a form of memory rich in contextual details, dependent on a brain region called the hippocampus, and another form of memory, called "semantic memory" which relies primarily on neocortex, and which is a more general memory, recording the gist of the initial episodic memory.

Studies in animals and humans have shown that the hippocampus, a brain region located deep inside the brain, has a central role in recent and remote episodic memory. Patients with hippocampal loss, including the famous Henry Molaison (patient HM) and Kent Cochrane (patient KC), were shown to be unable to make new memories, but they retained the ability to recall earlier events, in a schematic, general fashion. Dr. Moscovitch, investigating how rich, recent memories are often converted to more schematic, remote memories has elaborated a theory he has termed "multiple trace/transformation theory".

According to multiple trace/transformation theory, each time an episodic memory is retrieved, it is automatically re-encoded by the hippocampus along with the new context in which retrieval occurs. Over time, and with every retrieval, multiple memory traces accumulate; the neocortex extracts

similarities from these traces to form a generalized memory, the semantic memory. By this process, the memory is transformed over time, from a mostly hippocampus dependent, context-rich memory, to a more general memory, a recording of the essential elements of the memory, that captures the gist of the initial episodic memory.

Dr. Moscovitch presented results that show that the same processes apply to memory about places and the environment. Initially dependent on the hippocampus, they also are transformed, and become schematic memories that can be retrieved without the involvement of the hippocampus. As it was previously thought that the hippocampus was always involved in remembering places, this discovery sheds new light on the different forms of memory that exist.

"Spatial representations provide the framework in which events unfold, so that they interact with each other to form rich episodic memories that have both spatial and event elements" says Dr. Moscovitch. "Memory for events is facilitated if they occur in familiar rather than unfamiliar places. These findings could be used to help ameliorate memory problems in older adults, and in people with dementia, who have to leave their home and move into new living quarters."

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Scientific Presentation Abstract:

Spatial (and event) memory in humans and rodents - presentation by Dr. Morris Moscovitch

Since the discovery of place cells, it has been believed the hippocampus in both rodents and humans is needed for representing spatial layouts (environments) allocentrically. That representation, in turn, is needed for navigation, regardless of whether the environment was encountered and learned recently or long ago. Evidence from studies on spatial memory and navigation in humans and rats will be presented to examine this proposal. The results will show that the hippocampus is needed to retain and retrieve detailed memories of spatial layouts (scenes) for as long as the memories exist. With time, however, some of these memories are transformed, shedding contextual details, but retaining schematic cognitive maps that can represent space allocentrically. In the process, these transformed memories lose their hippocampal signature, and are represented in extra-hippocampal structures where they can be retained and from where they can be retrieved without hippocampal involvement. A multiple trace theory of hippocampal-neocortical interaction, and a transformation hypothesis, are proposed to account for the data.

Copies of recent publications by Dr. Morris Moscovitch are available upon request. Please contact Julie Poupart info@can-acn.org.

Press release- **Embargoed until May 28th 2014, 12:00 PM (noon)**

http://www.eurekalert.org/emb_releases/2014-05/cafn-cpn052114.php

Cocktail party neuroscience: Making sense of voices in a crowd

The role of prediction and attention in understanding of speech

Listening to a conversation in the context of a cocktail party presents a great challenge for the auditory system. Without realizing it, one must extract, from a complex mixture of sound, the sound of a single voice to understand and track it. Researchers at Queen's University, lead by Dr. Ingrid Johnsrude, are studying how our brains meet that challenge, and allow us to distinguish specific voices in crowded, noisy and distracting environments. Her studies have revealed that the brain does not simply rely on the incoming sounds that reach the ear to understand and retain speech, but rather also relies on information from other senses and prior knowledge to facilitate comprehension. These results were presented at the 8th Annual Meeting of the Canadian Association for Neuroscience held in Montreal, Canada May 25 to 28th 2014.

Dr. Johnrude's studies exposed test subjects to degraded or clear speech in the presence or absence of distraction. By looking at activation of different brain regions while test subjects were exposed to different listening conditions, Dr. Johnsrude's research has revealed that the early processing of sound, which occurs in a brain region called the primary auditory cortex, depends on higher-level linguistic knowledge encoded in other regions of the brain.

Following a conversation in a noisy environment also requires one to disregard surrounding noises and distractions and specifically focus on a conversational partner. While clear speech was understood and remembered whether subjects were distracted or not by other tasks, attention was shown to be critically important to understand degraded speech.

What you hear and understand of a conversation is influenced by what you are used to hearing, so it will be easier to understand a familiar voice than that of a stranger. This was shown to be especially true for older adults, who were shown to have more difficulty understanding new voices in a cocktail party situation as they age, but did not show a decline in the ability to understand familiar voices in the same situation.

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"We're all familiar with the glass half empty view of aging – that, as you get older, everything gets worse" says Dr. Johnsrude. "You need glasses, your memory goes, and it's harder to hear when you're conversing in a busy place like a restaurant or a party, where many people are talking at once. We wanted to investigate the glass-half full side of aging. One thing that older people have more of than younger people is experience. I study how the experience of older people, like their familiarity with the voice of their significant other, helps them compensate for age-related declines in other abilities."

Furthermore, Dr. Johnsrude was able to show that activation of certain brain regions, the higher-order speech sensitive cortex, could be viewed as a neural signature of effortful listening. Measuring the effort required to understand speech, using the techniques developed by Dr. Johnsrude, may provide a novel way to assess the efficacy and comfort of hearing protheses, and help researchers optimize the benefits obtained from these devices.

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About the Canadian Association for Neuroscience:
The Canadian Association for Neuroscience is the largest association dedicated to the promotion of all fields of neuroscience research in Canada. The association has been organizing a yearly annual meeting since 2007. Learn more about our meeting at: http://www.can-acn.org/meeting2014

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For news media only:

Please contact Julie Poupart, Communications Director for the Canadian Association for Neuroscience, for further information, to receive a press pack, or to request an interview with a neuroscientist: info@can-acn.org. Press passes for the Canadian Neuroscience Meeting, May 25-28 2014, in Montreal, are available for accredited journalists.

Scientific Presentation Abstract:

The role of prediction and attention in speech perception, by Ingrid Johnsrude

When speech is heard in the presence of background sound, or when hearing is impaired, the sensory information at the ear is often too ambiguous to support speech recognition by itself. In order to disambiguate and interpret the incoming sounds, the brain must integrate the auditory information with other sensory information and with prior knowledge to facilitate understanding. Prior information can enhance speech perception in different ways. For example, intelligibility and segregability of familiar voices is greater than for unfamiliar voices in the presence of competing speech; and coherent, predictive, semantic context appears to reduce processing load. A recent series of experiments exploit behavioural and imaging methods to explore the mechanisms underlying the integrative processes that

permit knowledge and experience to enhance understanding of degraded speech, and to examine how recruitment of such mechanisms is gated by attentional state. The field of visual perception has long recognized the important role played by feedback connections to early visual cortices in shaping perception; an emerging literature in the auditory domain is consistent with the idea that early auditory processing (in primary auditory cortex) is modulated by higher-level (linguistic) knowledge. This work adds to a growing literature indicating that primarily feedforward accounts of perceptual processing are incomplete, and that frontally mediated control processes are essential to accurate speech comprehension in the noisy and variable listening conditions that are characteristic of everyday life.

Copies of recent publications by Dr. Ingrid Johnsrude are available upon request. Please contact Julie Poupart info@can-acn.org.