9th Annual Canadian Neuroscience Meeting

Presented by:

Canadian Association for Neuroscience
Association canadienne des neurosciences

Press releases and Information for the Media

Annual Meeting: **May 24 - 27, 2015**

Westin Bayshore Hotel, 1601 Bayshore Dr, Vancouver, BC V6G 2V4

http://can-acn.org/meeting2015

Public Lectures: **May 23rd 2015, 4PM - 6 PM**

Science World, 1455 Quebec St, Vancouver, BC V6A 3Z7

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9th Annual Canadian Neuroscience Meeting - General Information

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: **Douglas Munoz**, PhD, Queen's University

Vice-President (President-Elect): **Freda Miller**, PhD, University of Toronto

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

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

Chair of the nominations committee: **Sam David**, PhD, McGill University

2015 Meeting Organisation

Chair of the 2015 Scientific Program Committee: **Kurt Haas**, PhD, University of British Columbia

Co-Chair of the 2015 Scientific Program Committee: **Kathleen Cullen**, PhD, McGill University

CAN-ACN Administration

Association Secretariat & Conference Management:

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

Marischal De Armond and **Caitlin Mooney**

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.

2015 Public Lectures

Two public lectures, presented in English and in French have been organised on May 23rd 2015. They are open to all people interested in neuroscience. Admittance is free but limited.

2015 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 releases are available in English and French.

Information about: 2015 CAN-ACN Public lectures May 23rd, 4 PM

Brain function in health and disease: Canadian Association for Neuroscience public lectures to highlight latest research findings

The Canadian Association for Neuroscience is bringing together two experts to deliver public lectures that will highlight how the brain works in health and disease. Dr. Janet Werker, Professor at the Department of Psychology of the University of British Columbia, Canada Research Chair in Psychology, and Director of the Infant Study Centre at UBC, will provide insight on how the brains of babies work, with a lecture titled: "Understanding the foundations of language development by studying the infant brain". Dr. Jon Stoessl, Professor & Head of Neurology at the University of British Columbia, Co-Director of the Djavad Mowafaghian Centre for Brain Health, Canada Research Chair in Parkinson will help us understand how diseases like Parkinson's disease affect brain function, in lecture titled "The Clinic as Laboratory: Lessons from Parkinson's".

Dr. Stoessl's studies provide insight on the normal functioning of the adult brain. His work has increased our understanding of the changes that occur in the brain even before clinical symptoms of Parkinson can be detected. An earlier detection of this disease provides clinicians with the opportunity to begin treatments and interventions to prevent some of the damage. Clinical studies have also helped Dr. Stoessl understand the mechanisms underlying the placebo effect and the overlap of symptoms that result in clinical dementia.

Dr. Janet Werker, studies how infants acquire language skills. Her studies have revealed the critical periods where different steps of language acquisition occur. By studying infants from birth up to two years of age she has uncovered some of the perceptual biases humans have at the beginning of life, and how those are sculpted through maturation, experience, and development to yield the perceptual categories the child uses in language acquisition.

Both public lectures will take place at Science World in Vancouver, British Columbia, on Saturday, May 23, starting at 4PM. These lectures are free and open to the general public, but space is limited.

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

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Press release:

Michael Douglas Gordon is the 2015 CAN Young Investigator awardee

Michael Gordon, from the University of British Columbia, will be receive the 2015 CAN Young Investigator Award at the upcoming 9th Annual Canadian Neuroscience Meeting in Vancouver on May 24, 2015

The Canadian Association for Neuroscience (CAN) is proud to announce that Michael Gordon, from the University of British Columbia, will be awarded the 2015 CAN Young Investigator Award at the upcoming 9th Annual Canadian Neuroscience Meeting in Vancouver, British Columbia on May 24th 2015.

Dr. Michael Gordon's research provides insight into two of the most critical decisions we, and other animals, have to make: what to eat, and how much. He studies this important and complex question in the fruit fly, *Drosophila melanogaster*, which has a relatively simple nervous system, with one million times fewer neurons than ours, yet displays a complex array of behaviours in response to food cues. He has significantly contributed to our understanding of the neural circuits that drive taste responses and feeding preferences.

Using the fly brain as a model, the Gordon lab combines molecular genetics with optical techniques and electrophysiology to map taste circuits, probe how these circuits encode information, and unravel their impact on feeding. These studies contribute to our understanding of how the brain translates sensory information into behaviour.

Dr. Gordon's work has shown that food preference can be viewed as a changing metric, based initially on taste, but evolving with experience, and the animal's physiological condition. These studies support the concept that in addition to sensing the palatability of food, like the sweetness or bitterness, flies also have a mechanism for sensing its caloric content, and that this could drive longer-term food preferences.

More recently, Dr. Gordon's team has uncovered a neural mechanism used by the fly brain to integrate the opposing effects of sweet and bitter tastes. Information from multiple sensory cues, the physiological state and experience of the animals thus all contribute to guiding feeding decisions.

Dr. Gordon's publication track record demonstrates the importance of his research contributions, and it is particularly impressive to note his productivity in the short four years since he has established himself as an independent researcher at the University of British Columbia. His recent research has been published in prestigious journals such as *Nature Communications* (2015), *Current Biology* (2014), *Neuron* (2014, 2013), and *The Journal of Neuroscience* (2012). Dr. Gordon's contributions, which include earlier publications in

Neuron in 2009 and Nature in 2005, have had significant impact and are highly cited, further demonstrating his position as a leader in the field of feeding regulation.

In addition, Dr. Gordon is a much-appreciated Faculty member at the department of Zoology at the University of British Columbia. His extensive teaching duties and successful mentorship of both undergraduate and graduate students attests to his importance in the Department.

The Canadian Association for Neuroscience wishes to thank Dr. Vanessa Auld, Professor at the University of British Columbia, and Dr. Tim O'Connor, Professor and Chair of the Graduate Program in Neurosciences at University of British Columbia, for nominating him for this award.

Within a very short time period as an independent researcher, Dr. Michael Gordon has established himself as an exceptional young scientist and a rising star in Canadian Neuroscience. We are very proud to present him with the 2015 CAN Young Investigator Award.

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Selected publications

LeDue EE, Chen YC, Jung AY, Dahanukar A, Gordon MD. Pharyngeal sense organs drive robust sugar consumption in *Drosophila*. Nat Commun. 2015 Mar 25;6:6667. doi: 10.1038/ncomms7667.

Chu B, Chui V, Mann K, Gordon MD. Presynaptic gain control drives sweet and bitter taste integration in *Drosophila*. Curr Biol. 2014 Sep 8;24(17):1978-84. doi: 10.1016/j.cub.2014.07.020.

Pool AH, Kvello P, Mann K, Cheung SK, Gordon MD, Wang L, Scott K. Four GABAergic interneurons impose feeding restraint in *Drosophila*. Neuron. 2014 Jul 2;83(1):164-77. doi: 10.1016/j.neuron.2014.05.006.

Mann K, Gordon MD, Scott K. A pair of interneurons influences the choice between feeding and locomotion in *Drosophila*. Neuron. 2013 Aug 21;79(4):754-65. doi: 10.1016/j.neuron.2013.06.018.

Stafford JW, Lynd KM, Jung AY, Gordon MD. Integration of taste and calorie sensing in*Drosophila*. J Neurosci. 2012 Oct 17;32(42):14767-74. doi: 10.1523/JNEUROSCI.1887-12.2012.

Gordon MD, Scott K. Motor control in a *Drosophila* taste circuit. Neuron. 2009 Feb 12;61(3):373-84. doi: 10.1016/j.neuron.2008.12.033.

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

Yang CH, Rumpf S, Xiang Y, Gordon MD, Song W, Jan LY, Jan YN. Control of the postmating behavioral switch in *Drosophila* females by internal sensory neurons. Neuron. 2009 Feb 26;61(4):519-26. doi: 10.1016/j.neuron.2008.12.021.

Gordon MD, Dionne MS, Schneider DS, Nusse R. WntD is a feedback inhibitor of Dorsal/NF-kappaB in *Drosophila* development and immunity. Nature. 2005 Sep 29;437(7059):746-9. Epub 2005 Aug 17.

More information

For more information about Dr. Gordon and his research, please visit his laboratory website:http://www.zoology.ubc.ca/~gordon/Gordon_Lab_-_Home.html

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Press release about public events organized around the meeting

Title:

Canadian Neuroscience Meeting, Public Talks and Café Scientifique in Vancouver May 23-27

Subtitle:

Canadian Association for Neuroscience, Djavad Mowafaghian Centre for Brain Health and Science World team up to present latest neuroscience research to the public

Approximately 700 leading neuroscientists from across Canada and around the world will be in Vancouver from May 24 to 27 for the 9th Annual Meeting of the Canadian Association for Neuroscience (CAN). The Djavad Mowafaghian Centre for Brain Health (DMCBH) has partnered with CAN and Science World British Columbia to host a series of events on May 23 to engage the public with the newest in brain health research.

“This year’s Annual Meeting is an exciting one for us,” says Dr. Kurt Haas, a researcher at DMCBH and Chair of the CAN 2015 Scientific Program Committee. “We’re doing more public outreach, and really trying to capitalize on the public’s interest in neuroscience.”

Public events will take place on Saturday, May 23 at TELUS World of Science in Vancouver.

CAN Public Talks

May 23, 4:00 – 6:00 p.m.

Free, open to the general public. Space is limited, please RSVP:

https://eventbrite.ca/event/17047247777

The Canadian Association for Neuroscience is bringing together two experts to deliver public lectures that will highlight how our brain works in health and disease.

Dr. Janet Werker, Professor at the Department of Psychology of the University of British Columbia, Canada Research Chair in Psychology, and Director of the Infant Study Centre at UBC, will provide insight on how the brains of babies work, with a lecture titled: "Understanding the foundations of language development by studying the infant brain."

Learn more about Dr. Werker here:
http://can-acn.org/janet-werker-can2015-public-lecture-speaker

Dr. Jon Stoessl, Professor & Head of Neurology at the University of British Columbia, Co-Director of the Djavad Mowafaghian Centre for Brain Health, Canada Research Chair in Parkinson will help us understand how diseases like Parkinson's affect brain function, in lecture titled "The Clinic as Laboratory: Lessons from Parkinson's."

Learn more about Dr. Stoessl here:
http://can-acn.org/jon-stoessl-can2015-public-lecture-speaker

Café Scientifique

May 23, 7:00 – 9:00 p.m. (doors open at 6:30 p.m.)

Free, open to the general public. Space is limited, please RSVP:
swcafescientifiqueparkinsons.eventbrite.ca

Is the latest research shaking things up?

Parkinson's disease is the second most common neurodegenerative disorder, after Alzheimer's disease. It affects both the motor and non-motor functions of over 100,000 Canadians and 6.3 million people worldwide. Join us for discussion, debate and dessert, as we explore the latest research and developments with professionals working toward improving treatment and finding a cure.

Expert Panelists

Dr. Martin McKeown, Clinical Neurologist at the Djavad Mowafaghian Centre for Brain Health and Director, Pacific Parkinson's Research Centre

Dr. Austen Milnerwood, Researcher at the Djavad Mowafaghian Centre for Brain Health and Head, Translational Neuroscience, Centre for Applied Neurogenetics

Noel MacDonald, Caregiver and co-founder of Porridge for Parkinson's

The event will be moderated by Emily Wight, Communications Manager at DMCBH.

Meet a Scientist

May 23, all day

With admission to TELUS World of Science

Graduate students and postdoctoral fellows from the Graduate Program in Neuroscience at DMCBH and the University of British Columbia will be interacting with guests of TELUS World of Science throughout the Centre. Students have designed family friendly demonstrations and activities based on their neuroscience research. Come ask a question, meet a scientist and learn a little more about the human brain!

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.

Canadian Neuroscience Meeting

The **Canadian Association for Neuroscience** is holding its 9th Annual Meeting in Vancouver, May 24 to 27 2015. Held yearly since 2007, it brings together researchers working in all fields of neuroscience research. Organized by neuroscientists and for neuroscientists, it highlights the best and most novel neuroscience research in Canada every year. Only registered neuroscientists can participate, but credentialed journalists can attend and report on presentations. Learn more about this meeting at:

http://can-acn.org/meeting2015

Djavad Mowafaghian Centre for Brain Health

The **Djavad Mowafaghian Centre for Brain Health** comprises more than 250 investigators with multidisciplinary expertise, bridging basic science and clinical care in a state-of-the-art facility on UBC's Point Grey campus. The Centre provides opportunities for education, collaboration, and interaction with patients from across BC. The Centre is the largest and most comprehensive brain care and research centre in Canada, and is a partnership of the UBC Faculty of Medicine, Vancouver Coastal Health, and Vancouver Coastal Health Research Institute.

Science World British Columbia

Science World British Columbia is a not-for-profit organization that engages British Columbians in science and inspires future science and technology leadership throughout our province.

Press release for Mel Goodale's presentation at the Canadian Association for Neuroscience meeting, May 24, 2015 in Vancouver.

Press release embargoed until May 24th 2015, 7:00 PM PDT | 10PM EDT

Title:

Research news:
Can you see what I hear? Blind human echolocators use visual areas of the brain.

Canadian expert Mel Goodale determines echolocators use echoes to detect multiple properties of objects through areas of the brain associated with vision.

Summary:

Certain blind individuals have the ability to use echoes from tongue or finger clicks to recognize objects in the distance, and use echolocation as a replacement for vision. Research done by Dr. Mel Goodale, from the University of Western Ontario, in Canada, shows echolocation in blind individuals is a full form of sensory substitution, and that blind echolocation experts recruit regions of the brain normally associated with visual perception when making echo-based assessments of objects.

Text:

Certain blind individuals have the ability to use echoes from tongue or finger clicks to recognize objects in the distance, and some use echolocation as a replacement for vision. Research done by Dr. Mel Goodale, from the University of Western Ontario, in Canada, and colleagues around the world, is showing that echolocation in blind individuals is a full form of sensory substitution, and that blind echolocation experts recruit regions of the brain normally associated with visual perception when making echo-based assessments of objects. Dr. Goodale's latest results were presented at the 9th Annual Canadian Neuroscience Meeting, on May 24th 2015 in Vancouver British Columbia.

"Our experiments show that echolocation is not just a tool to help visually-impaired individuals navigate their environment, but can act as an effective sensory replacement for vision, allowing them to recognize the shape, size, and material properties of objects" says Mel Goodale.

Just like multiple properties (size, expected weight, texture, composition) of an object assessed by visual cues are encoded in different brain regions, recent research done in the Goodale laboratory shows that the same is true of information obtained through the auditory cues provided by echolocation. Indeed,

many of the same regions in the sighted brain that are used for the visual assessment of objects are recruited in the blind brain when objects are explored using echolocation.

To understand what an object is, and to know how to interact with this object, knowing what an object is made of, its "stuff", is equally important as knowing its structure or shape. While his initial studies have investigated how echolocators detect the shape and distance of objects, Dr. Goodale's most recent studies have investigated how they perceive the material or "stuff" that different objects are made of.

“Remarkably, expert blind echolocators can tell whether something is hard or soft, dense or not, just by listening to the echoes bouncing back from that material” notes Dr. Goodale.

While sighted individuals use visual cues to get information about the composition of objects, such as the sheen of metal, or the fuzziness of fur, echolocators must rely on the auditory cues that result from the echoes of the clicks they emit. To determine how the brains of echolocators process these cues, researchers have recorded the echoes produced by echolocator's clicks on different materials (a blanket, fake foliage and a whiteboard) and looked at the response these sounds produced in the brains of sighted people, of blind non-echolocators and of blind echolocators. To view which brain regions were activated in these individuals, an advanced brain imaging technique called functional magnetic resonance imaging (fMRI) was used.

These studies show that material-related signals activate a region of the brain called the parahippocampal cortex (PHC) in blind expert echolocators, but not in sighted people or blind non-echolocators. PHC activation is associated with scene perception in sighted individuals. Just as in sighted individuals using vision, the brain regions that play a critical role in processing the structure and geometry of objects are distinct from the brain regions that process the cues that signal the material properties of objects in blind echolocators.

Interestingly, other studies in the Goodale lab have shown that blind expert echolocators are also subject to illusions, for example the size-weight illusion in which the perception of mass is influenced by the size of an object. If two objects of equal weight are presented to both a sighted and a blind echolocator, both will find the smaller object feels heavier when they lift it using a string attached to a pulley. This illusion, thought to be based on the lifter's cognitive expectations, and the fact that it is also present in blind echolocators, but not in blind non-echolocators, shows that echolocation is an effective form of sensory substitution for vision.

Because echolocation allows blind individuals to perceive objects from a distance, it can be used as an alternative to vision, allowing the perception of distant objects that would be impossible through touch. In fact, some echolocators are proficient enough to use this ability to perform complex tasks such as riding a bicycle – or even sinking a basketball!

Dr. Mel Goodale is Director of the Brain and Mind Institute at the University of Western Ontario. He holds the Canada Research Chair in Visual Neuroscience. Dr. Goodale is a member of the Royal Society of Canada and the Royal Society (London, UK). Dr. Mel Goodale is the 2015 Presidential Lecturer at the 9th Annual Canadian Neuroscience Meeting. Learn more about Dr. Goodale here: Melvyn Goodale profile page: http://can-acn.org/melvyn-goodale-can2015-presidential-lecturer

Sources of funding:

Canadian Institutes of Health Research, the Natural Sciences and Engineering Research Council of Canada, Canada Research Chairs Program.

Press release for Christopher Pack's presentation at the Canadian Association for Neuroscience meeting, May 25, 2015 in Vancouver.

Press release embargoed until May 25th 2015, 9:30 AM PDT | 12:30PM EDT

Title:

Research news: Patterns of brain activity reorganize visual perception during eye movements

Scientists measuring brain activity have found that in many regions, such as the sensory or motor cortex, activity sometimes oscillates at different frequencies, forming wave-like patterns. Despite the fact that such oscillations are frequently observed, and present in many brain regions, their functional role remains unclear. Research done by Dr. Christopher Pack, from McGill University, who looked at such waves occurring in a region of the visual cortex of the brain, suggests these oscillations could have a role in resetting the sensitivity of neurons after eye movements. Further results suggest these waves could also have a role in supporting the brain's representation of space. These results were presented at the 9th Annual Canadian Neuroscience Meeting, on May 25th 2015 in Vancouver, British Columbia.

Vision is an extremely dynamic process - Even when we look at a fixed image, our eyes are making rapid movements, called "saccades" to explore the image that is sent to our brain. By recording neuronal activity in monkeys as they performed tasks that caused saccades, Dr. Christopher Pack has shown that there are waves of activity that cross specific vision processing areas of the brain in defined patterns, and that these patterns are reorganized by saccadic eye movements. After a saccade, this wave of activity reorganizes to travel from the fovea, at the center of the visual field, and which is the area of the eye with the best visual acuity, to the periphery. As the wave passes, it is suggested to reset the sensitivity of the area, and its pattern allows visual processing to occur earlier in the fovea relative to the periphery resulting in an early focus on the object that attracted the eyes. This work was recently published in the journal Neuron (Zanos et al., 2015).

More recent work has shown that patterns of oscillations can facilitate the representation of visual space during eye movements. This is important because each saccade shifts the position of visible objects on the retina, and hence the brain needs to know the stable positions of objects in external space. By permitting rapid neural communication across neurons encoding different spatial positions, oscillations could contribute to this important perceptual process.

This research was supported by:

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Canadian Institute of Health Research (MOP-115178) and Natural Sciences and Engineering Research Council of Canada (341534-12).

Press release for Liisa Galea's presentation at the Canadian Association for Neuroscience meeting, May 25, 2015 in Vancouver.

Press release embargoed until May 25th 2015, 3:00 PM PDT | 6:00 PM EDT

Title:

Research news:
Motherhood permanently alters the brain and its response to hormone therapy later in life

Liisa Galea from the University of British Columbia presents her latest findings on hormone therapy and brain function at 9th Annual Canadian Neuroscience Meeting

Summary:

Research by Liisa Galea, University of British Columbia, suggests the form of estrogens used in Hormone Therapy (HT) and previous motherhood are critical to explain why HT has variable effects on cognitive functions. Estradiol had beneficial effects while estrone did not. Furthermore, the effects of estrone also depended on the experience of motherhood: estrone-based HT impaired learning in middle-aged rats that were mothers, while it improved learning in rats that were not.

Text:

Hormone therapy (HT) is prescribed to alleviate some of the symptoms of menopause in women. Menopausal women are more likely to be diagnosed with Alzheimer's disease but not other forms of dementia, and HT has been prescribed to treat cognitive decline in post-menopausal women with variable degrees of effectiveness. New research by Dr. Liisa Galea, at the University of British Columbia, suggests the form of estrogens used in HT and previous motherhood could be critical to explain why HT has variable effects. Research in women, and Dr. Galea's research in animals, shows that one form of estrogens, called estradiol, which is the predominant form of estrogens in young women, had beneficial effects, while estrone, which is the predominant form of estrogens in older women, did not. Furthermore, the effects of estrone also depended on whether the rats had experienced motherhood: estrone-based HT impaired learning in middle-aged rats that were mothers, while it improved learning in rats that were not. Dr. Galea's latest results were presented at the 9th Annual Canadian Neuroscience Meeting, on May 25th 2015 in Vancouver British Columbia.

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"Our most recent research shows that previous motherhood alters cognition and neuroplasticity in response to hormone therapy, demonstrating that motherhood permanently alters the brain" says Dr. Liisa Galea.

Dr. Liisa Galea is interested in how hormones affect brain and behaviour. Hormone therapy (HT) has been shown to have variable effects on brain function and Dr. Galea noted that one factor that had not received much attention was the form of estrogens used in HT. There are three forms of estrogens: estradiol, estrone and estriol. Estradiol is the most potent of estrogens, and it is the predominant form in young women, while estrone is a weaker estrogen and is the predominant form in post-menopausal women. A systematic review of the published scientific literature indicates that estradiol-based HT may have more beneficial effects, while estrone-based HTs may have more detrimental effect on cognition and dementia risk in women.

Dr. Galea studied how two forms of estrogens, estradiol and estrone, affect neuroplasticity, which is how neural pathways in the brain change in response to various factors. Her studies focused on a specific brain region, called the hippocampus, which has important roles in memory and spatial ability, such as navigational skills. Both forms of estrogens increased the production of new cells in a part of the hippocampus called the dentate gyrus in young females. However, only chronic estradiol, but not chronic estrone, significantly increased the survival of these new neurons, and increased the expression of zif268, a protein involved in neuroplasticity.

Chronic estradiol, but not chronic estrone, also improved performance of young female rats in a behavioural test called the water maze. The water maze is a test of memory and orientation in which rats must find a submerged platform in water that they cannot see; they must instead rely on cues located around them to orient themselves and swim to the platform. Rats receiving estradiol based HT found the platform significantly better that rats receiving estrone-based HT.

Finally, Dr. Galea's previous research had shown that motherhood causes changes in the architecture of connections in the hippocampus, so her team investigated whether the different forms of estrogens could have different effects on rats that had experienced motherhood once (primiparous rats) and on those who had not (nulliparous rats). They found that estrone-based HT improved learning in middle-aged nulliparous rats, but impaired learning in primiparous rats of the same age. These primiparous rats also showed a reduction in neurogenesis and zif268, a protein involved in neuroplasticity in the hippocampus.

As estrone is a component of the most common form of HT prescribed for women in the US, these findings could have implications for the treatment of age-related neurodegenerative disorders in women.

"Hormones have a profound impact on our mind. Pregnancy and motherhood are life-changing events resulting in marked alterations in the psychology and physiology of a woman. Our results argue that these factors should be taken into account when treating brain disorders in women" concludes Dr. Liisa Galea.

Funding:

This research was funded by the Alzheimer's Society of Canada and Canadian Institutes for Health Research (MOP102568).

Press release for Min Zhuo's presentation at the Canadian Association for Neuroscience meeting, May 27, 2015 in Vancouver.

Press release embargoed until May 27th 2015, 3:00 PM PDT | 6:00 PM EDT

Research news:
A better understanding of links between pain and anxiety reveals treatment opportunities

Dr. Min Zhuo and his team at the University of Toronto have found the biological basis for the link between pain and anxiety

Summary:

Anxiety is common in people suffering from chronic pain, and people with anxiety are more likely to suffer from chronic pain. Dr. Min Zhuo and his team at the University of Toronto have found the biological basis for this link in the connections between neurons in a brain region known as the anterior cingulate cortex (ACC). Better yet, they have identified a molecule that can reduce chronic pain-related anxiety.

Full Text:

Pain has both physical and emotional components. Anxiety is common in people suffering from chronic pain, and people with anxiety are more likely to suffer from chronic pain. Dr. Min Zhuo and his team at the University of Toronto have found the biological basis for this link in the connections between neurons in a brain region known as the anterior cingulate cortex (ACC). Better yet, they have identified a molecule that can reduce chronic pain-related anxiety. Dr. Zhuo's latest results were presented at the 9th Annual Canadian Neuroscience Meeting, on May 27th 2015 in Vancouver British Columbia.

"This study provides the first synaptic mechanisms to explain the multiple functions of ACC neurons in pathological conditions such as chronic pain" says Dr. Zhuo.

Chronic pain can be viewed as a learned memory: In the way that repetition of a piano piece enables you to learn it by facilitating transmission of the appropriate signals through your neurons, pain that persists can become chronic because your neurons become more efficient at transmitting pain signals. This strengthening of connections between neurons through repeated use is called Long Term

Potentiation, or LTP. Previous work in the Zhuo lab has shown that in animal models of chronic pain, LTP occurs in a part of the brain called the Anterior Cingulate Cortex, or ACC, and that inhibiting LTP reduces chronic pain.

Interestingly, increased activity in the ACC is also seen in humans suffering from anxiety disorders and in animal models of anxiety. However how LTP in the ACC differs in chronic pain and anxiety was not known, nor why the two would interact to result in more pain in anxiety sufferers, and more anxiety in pain sufferers.

The most common form of LTP is an increase in the number of receptors in neurons that are downstream of the synapse, which is the structure through which neurons communicate with each other. This is called post-LTP, because it is present after the synapse. Another less studied form of LTP occurs before the synapse, and involves the release of a larger amount of the signal from the neuron upstream. This is called pre-LTP. By using molecules that specifically block pre-LTP or post-LTP, Dr. Zhuo found a new form of pre-LTP that occurs in the ACC. Pre-LTP had previously only been seen in other brain regions.

"It is novel to demonstrate that both pre- and post-LTP can take place at the same cortical synapse!" says Dr. Zhuo. "As compared with post-LTP, pre-LTP employs a different set of molecules to induce and express the injury-related potentiation; it provides new opportunities for us to discover new drugs that may selectively control anxiety vs pain in future."

Dr. Zhuo's team also showed that pre and post-LTP were present in conditions of chronic pain, but that pre-LTP was only present when the pain became chronic, and not in cases of acute pain. They found that blocking pre-LTP reduced anxiety, and also that conditions that produced anxiety in animals (without pain) resulted in pre-LTP. These experiments led them to conclude that pre-LTP in neurons of the ACC mediates anxiety.

The discovery that two forms of LTP exist in the ACC, with pre-LTP associated with anxiety and post-LTP associated with pain, explains why these two conditions are linked, as both conditions result in an increase in transmission of the glutamate signal between neurons in the ACC.

Dr. Zhuo's team also found a novel molecule, called NB001, which can specifically block neuronal pre-LTP, and has powerful analgesic, or pain-reducing, effects in animal models of chronic pain. Further investigation of the signaling pathways of pre- and post-LTP should reveal new drug targets for treating pain and anxiety.

"This work is a result of a global collaboration with three different countries, each contributing valuable and novel research. This model of global collaboration is, is the future of excellence in scientific research" concludes Dr. Zhuo.

Source of funding:

the EJLB-CIHR Michael Smith Chair in Neurosciences and Mental Health, Canada Research Chair, NeuroCanada, and Canadian Institute for Health Research operating grants (CIHR66975 and CIHR84256); also funding from the National Honor Scientist Program of the National Research Foundation funded by the Korea government (MSIP); the MRC (UK) as well as Chinese national science foundation (China). Experiments were performed in multiple sites including Toronto, Seoul and Xian.

Press release for Graham Collingridge's presentation at the Canadian Association for Neuroscience meeting, May 27th, 2015 in Vancouver.

Press release embargoed until May 27th 2015, 3:00 PM PDT, 6 PM EDT

Research news: Molecules involved in Alzheimer's have a role in weakening of connections between neurons

Graham Collingridge finds molecules strongly associated with Alzheimer's disease are important players in a process called long-term depression (LTD).

Summary:

Dr. Graham Collingridge, from the University of Toronto, has found that molecules that are strongly associated with Alzheimer's disease are important players in a process called long-term depression (LTD). LTD is a process through which the strength of synapses, the connections between neurons, is selectively reduced. Dr. Collingridge's recent research suggests improperly regulated LTD could cause the degeneration of the connections between neurons that is a core feature of Alzheimer's and other neurodegenerative diseases.

Full Text:

Alzheimer's disease is the most common form of dementia, affecting over 44 million people worldwide. Inside the brain, Alzheimer's disease is characterized by loss of neurons, and presence of abnormal tangles and plaques in the brain. Dr. Graham Collingridge, recently recruited from Bristol (U.K.) to the University of Toronto, has found that molecules that are strongly associated with Alzheimer's disease are important players in a process called long-term depression (LTD). LTD is a process through which the strength of synapses, the connections between neurons, is selectively reduced. Dr. Collingridge's recent research suggests improperly regulated LTD could cause the degeneration of the connections between neurons that is a core feature of Alzheimer's and other neurodegenerative diseases.

"Recently, we have found that tau has a key physiological role in the process of LTD", explains Dr. Collingdridge. "Tau aggregates to form the hall-mark tangles of Alzheimer's disease and serves as the best marker of disease progression. Our finding that tau has a normal function at synapses adds considerable weight to the argument that Alzheimer's disease is triggered by a mis-regulation of a normal synaptic mechanism. We have also recently identified a novel and very rapid synaptic action of

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Abeta, a protein fragment that forms the senile plaques and is strongly implicated in the aetiology of Alzheimer's disease."

Learning and memory involve modifications in the distribution and strength of synapses, which are the points of connection between neurons. The two most studied forms of such modification are long-term potentiation, which is a strengthening of a synapse, by an increase in the transmission of signals through a synapse, and long-term depression, which is a weakening of the synapse. These modifications, also called synaptic plasticity, are a major process used for information storage in the brain and spinal cord.

Over the past decade, researchers have realized that aberrant synaptic plasticity may lie at the heart of many brain disorders. Dr. Collingridge's research, which focuses on a brain region called the hippocampus, which is important for memory storage, helps identify they key players in LTD. Once identified, these become potential therapeutic targets, as new drugs could be designed specifically to activate or inhibit them.

"Over the last few years we, and others, have identified many of the molecules involved in LTD. Potentially targeting these could provide novel approaches for the treatment of Alzheimer's disease and other neurodegenerative conditions" says Collingdridge.

Alzheimer is an incurable and chronic disease, and current treatments bring only modest improvements to symptoms, and do not work for all patients. A better understanding of the process affected in the brain will lead to identification of new drug targets, and potentially, life-changing preventive therapies or treatments.