McMaster University

Centre for Metabolism, Obesity and Diabetes Research

Annual Report 2019

Table of Contents

MESSAGE FROM THE CO-DIRECTORS

Gregory Steinberg, PhD
Medicine
Professor

Katherine Morrison, MD, FRCPC
Pediatrics
Professor

The McMaster Centre for Metabolism, Obesity and Diabetes Research (MODR) was founded in July 2018 with the mandate to develop new strategies for the prevention and treatment of chronic metabolic diseases. In this inaugural annual report, it is our pleasure to highlight some of the team's key accomplishments to date.

The MODR team is comprised of over 40 faculty members, research trainees and research and administrative staff with extensive experience in preclinical and clinical research. The investigators are from multiple faculties, departments and research disciplines at McMaster and collaborate with a network of scientists across Canada and around the world. The close collaborative ties between disciplines promotes rapid bidirectional translation of research findings.

Central to this rich research environment is the development of the next generation of scientists. The interdisciplinary composition of the MODR team provides an ideal training environment for post-doctoral fellows, clinician scientists, undergraduate and graduate students. Trainees are actively engaged with hosting the MODR biweekly seminar, annual symposia and monthly journal club - providing trainees with the opportunity to interact with world leaders in metabolism. MODR also hosts career development workshops, organized by the graduate students.

As you read through this report, you will note the extensive research contributions made by members of the MODR team! We are delighted with the engagement and enthusiasm of the MODR members to collaborate to generate new knowledge, to translate knowledge across traditional silos and to develop scientists comfortable in the translational environment through experiential education. We are looking forward to further growth in the years to come!

MESSAGE FROM THE SENIOR ADVISOR

Hertzel Gerstein, MD, MSc, FRCPC
Medicine
Professor

Research is about being open. It is about being open to new ideas; being open to new paradigms that integrate observations from populations, people, animals, cells and molecules; being open to new ways of interacting with researchers from other disciplines without preconceived ideas; and being open to learning other researchers' vocabularies while sharing your own.

It is this spirit of openness that underlies the Centre for Metabolism, Obesity and Diabetes Research. Unlike most research groups, its founders, Drs. Gregory Steinberg and Katherine Morrison, have created a collaborative research group without walls due to background, research materials or research techniques. It is a research group that leverages its members' keen interest in understanding and solving problems related to metabolism, obesity and diabetes, and their ongoing search for answers to questions posed to them by their patients, animals, cells or molecules on a daily basis. It therefore transcends the old paradigm of clinical and basic research and replaces it with research that is tailored to the question being asked. This approach is not easy. However, it is already yielding benefits that are apparent in list of projects contained herein.

As a colleague and collaborator with many in the Centre, and as someone whose career has focused on transdisciplinary collaboration, it is gratifying to see this model succeeding.

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Kinesiology

Professor
Department Chair

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Associate Member – Biochemistry & Biomedical Sciences
Associate Member - Kinesiology

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Health Research Methods, Evidence, and Impact

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Bernardo Trigatti, PhD

Biochemistry & Biomedical Sciences

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Associate Professor

Gita Wahi, MD, FRCPC
Pediatrics

Associate Professor

Geoff Werstuck, PhD
Medicine

Professor

MEMBER SPOTLIGHTS

New McMaster research centre zeros in on metabolism, obesity and diabetes

MODR Co-Directors Gregory Steinberg & Katherine Morrison

Modified from: Tina Depko - Faculty of Health Sciences, McMaster University
October 1, 2018

McMaster University is growing its commitment to addressing the epidemic of obesity and related health consequences with the establishment of the Centre for Metabolism, Obesity and Diabetes Research (MODR).

Two renowned McMaster researchers, Katherine Morrison and Gregory Steinberg, are serving as its co-directors. "We want to use our multidisciplinary expertise, ranging from cellular systems to patient populations, to develop new preventative and treatment strategies to help people with chronic metabolic diseases," said Morrison, professor in the Department of Pediatrics at McMaster and a pediatric endocrinologist with Hamilton Health Sciences.

A team of researchers and clinicians from McMaster and Hamilton Health Sciences will work under the umbrella of MODR.

"The MODR researchers' expertise, complemented by state-of-the-art equipment, unique genetic models, and platforms for studying metabolism makes them world-leaders in biomedical discovery," said Steinberg, professor in the Department of Medicine at McMaster and the Canada Research Chair in Metabolic Diseases.

A specific focus of the MODR team is discovering the biological drivers leading to energy imbalance and metabolic disturbance, understanding the mechanisms for mediators of energy balance and evaluating and treating associated adverse health outcomes. Paul O'Byrne, dean and vice-president of the Faculty of Health Sciences, said the evolution of the MAC-Obesity research program into an established centre is a testament to the caliber of work being conducted at the university.

"The research program led to date by Dr. Morrison and Dr. Steinberg has produced many important findings, and this research will be further advanced by the formal creation of the centre," said O'Byrne.

"We anticipate more essential work ahead, as the combination of basic scientists and clinicians results in a holistic approach in the hunt for more answers to help address this pressing health issue."

Myth busted: Researchers show that a high-protein diet does not affect kidney function

Stuart Phillips

Modified from: brighterworld.mcmaster.ca
November 7, 2018

A widely held and controversial myth that high-protein diets may cause kidney damage in healthy adults has been debunked by scientists at McMaster University, who examined more than two dozen studies involving hundreds of participants.

The meta-analysis, published in *The Journal of Nutrition*, challenges the perceived dangers of a protein-rich diet, a notion first introduced in the 1980s which suggested processing large amounts of protein leads to a progressive decline in kidney function over time.

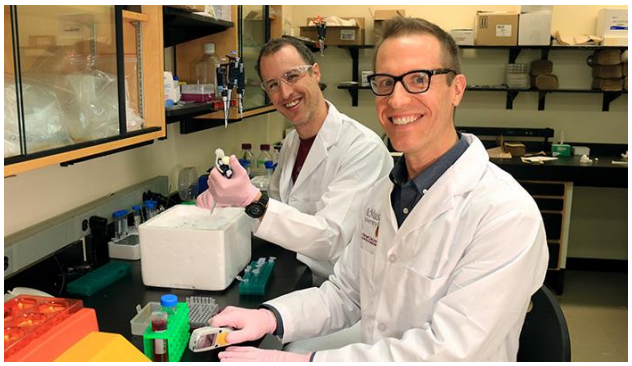
"It's a concept that's been around for at least 50 years and you hear it all the time: higher protein diets *cause* kidney disease," says Stuart Phillips, a professor of kinesiology at McMaster who oversaw the study.

"The fact is, however, that there's just no evidence to support this hypothesis in fact, the evidence shows the contrary is true: higher protein increases, not decreases, kidney function," he says.

"While there is a breadth of evidence showing the benefits of higher protein consumption, some people are still afraid it could cause kidney damage," says Michaela Devries-Aboud, lead author of the study and assistant professor of kinesiology at the University of Waterloo, who conducted the analysis as a postdoctoral fellow at McMaster. "With these findings, we have shown that a higher protein diet is safe. In fact, it should be viewed as an important tool for muscle health across an entire lifespan."

"There is simply no evidence linking a high-protein diet to kidney disease in healthy individuals or those who are at risk of kidney disease due to conditions such as obesity, hypertension or even type 2 diabetes," says Devries-Aboud. According to Phillips, "Protein *causing* kidney damage just lacks any support. I think we can put this concept to rest."

Gut bacteria that are changed by diet play key role in increased risk of diabetes: McMaster researchers



Kevin Foley (first author) & Jonathan Schertzer (senior author)

Modified from: Tina Depko - Faculty of Health Sciences, McMaster University
November 7, 2018

McMaster University researchers have found that gut bacteria, modified by diet, plays a role in elevated blood glucose, which is the primary indicator of Type 2 diabetes.

Based on research in mice, the data showed that while an obesity-causing diet altered gut bacteria within days, it took more than a month for these new gut bacteria to change blood glucose. The findings were published today in the journal *Nature Communications*.

"We were interested in how the microbiota, which is the bacteria that mostly lives in the gut, changes blood glucose because it is a key factor in health," said Kevin Foley, first author and a postdoctoral fellow in the Schertzer lab at McMaster.

"Our results in mice are starting to unravel how food and bacteria work together to increase the risk of Type 2 diabetes."

There are 11 million Canadians living with diabetes or prediabetes, according to Diabetes Canada. Approximately 90 per cent of people with diabetes have Type 2 diabetes. While it more often develops in adults, children can also be affected.

"The findings make sense to us, as people can develop obesity or have weight problems for a very long time before diabetes onset," said Jonathan Schertzer, senior author of the paper, associate professor of biochemistry and biomedical sciences at McMaster, and the Canada Research Chair in Metabolic Inflammation.

"The indication is that diet rapidly changes gut bacteria, but these bacteria play a role later on in the process for elevated blood glucose. This information provides us with a better idea on when to target gut bacteria to help control blood glucose."

The research was led by McMaster in collaboration with Soumaya Zlitni from Stanford University. McMaster researchers were Foley, Schertzer, Emmanuel Denou, Brittany Duggan, Rebecca Chan and Jennifer Stearns. The Schertzer lab is affiliated with McMaster's Farncombe Family Digestive Health Research Institute, and the Centre for Metabolism, Obesity and Diabetes Research (MODR) at McMaster. Funding for the research came from the Canadian Institutes of Health Research (CIHR).

Large international study finds diabetes drug cuts cardiovascular and kidney problems

Hertzel Gerstein

Modified from: Faculty of Health Sciences, McMaster University
June 10, 2019

A clinical trial that followed more than 9,900 people in 24 countries has found that the drug dulaglutide reduced cardiovascular events and kidney problems in middle-aged and older people with Type 2 diabetes.

During more than five years of follow-up, cardiovascular events like heart attacks and strokes were reduced by 12% in people taking dulaglutide compared to people taking a placebo. This effect was seen in both men and women with or without previous cardiovascular disease.

In addition, during the same period, the drug reduced the development of kidney disease by 15%.

The trial was led by the Population Health Research Institute (PHRI) of McMaster University and Hamilton Health Sciences. Two papers describing the cardiovascular and kidney results of the trial were published today in the journal *The Lancet* from the study called the Researching Cardiovascular Events with a Weekly Incretin in Diabetes (REWIND) trial.

"Compared to others, people with diabetes have twice the rate of cardiovascular events like heart attacks and strokes, and up to 40% of people with diabetes develop kidney disease," said Hertzel C. Gerstein, principal investigator for the study, professor of medicine at McMaster and deputy director of the PHRI.
"The REWIND trial shows that dulaglutide can safely reduce these events while improving diabetes control and modestly lowering weight and blood pressure in middle-aged people with Type 2 diabetes."

The REWIND trial was funded by Eli Lilly and Company, the makers of the dulaglutide drug Trulicity. The study was designed and led by a team of scientists that included representatives of the funder. The data were analyzed by scientists of the PHRI and the principal investigator had final responsibility for the papers.

New study may have the reason why heart medication gives muscle pain

Thomas Hawke (senior author) & Irena Rebalka (first author)

Modified from: Faculty of Health Sciences, McMaster University
October 16, 2019

A study from McMaster has found a potential mechanism explaining why some people who take drugs to lower their cholesterol develop sore, aching muscles.

The use of statin drugs to significantly lower cholesterol, and ultimately reduce the risk of cardiovascular disease, has become widespread and large-scale studies suggest that nearly half of Americans and a quarter of Canadians are receiving or are eligible for statin treatment.

Understanding why statins cause muscle pain and how this could be treated could remove a significant obstacle for health-care professionals to effectively manage a patient's cholesterol and lower their cardiovascular disease risk.

The McMaster research team found muscle cells treated with statins released glutamate, an amino acid, at much higher levels than muscle cells that were untreated. As glutamate is a potent activator of muscle pain receptors, this release was proposed to trigger the sensation of muscle pain.

Thomas Hawke, senior author of the study and a professor in pathology and molecular medicine at McMaster University, said, "We found that statins were able to enter the muscle cells and cause oxidative stress. This resulted in the muscle trying to increase its production of antioxidants to combat this stress. The side-effect of this antioxidant production was the release of glutamate out of the muscle cells."

Irena Rebalka, first author of the study and a research associate in the Hawke Lab, added, "We found that administering some well-known antioxidants, such as vitamin E, was successful in helping reduce glutamate release. We are now expanding our studies to determine further compounds which could be used in conjunction with a person's statin prescription to reduce the burden of muscle pain resulting from this drug."

More electronic device use tied to more sugar and caffeine in teens

Katherine Morrison

Modified from: brighterworld.mcmaster.ca
October 22, 2019

Do young teens who spend more time with TV and electronic devices drink more sugared or caffeinated drinks than others? Yes, they do, says a study of U.S. teens led by McMaster University researchers.

"It is a concern because many exceed recommended levels of both sugar and caffeine", says pediatrician Katherine Morrison, who led the research together with colleagues at McMaster and California State University - Fullerton.

"Greater electronic device use, particularly TV, is linked to more consumption of added sugar and caffeine among adolescents," she said.

The study, published today in PLOS, found that more than 27 per cent of teens exceed recommended sugar intake and 21 per cent exceed recommended caffeine from soda and energy drinks. Males consumed more sodas and energy drinks than females, and youth in Grade 8 consumed more than those in Grade 10.

The researchers were surprised to find video game use was only weakly linked to more caffeine consumption.

"Given the marketing campaigns that target video gamers, we expected a particularly strong association between caffeine intake from energy drinks or sodas with video game use, but TV was linked more strongly," said Morrison. However, using a computer for school was linked to a lower likelihood of exceeding sugar consumption cut-offs.

Secret behind diabetes drug's benefits revealed

From left to right: Hertzel Gerstein, Emily Day, Gregory Steinberg

Modified from: Tina Depko - Faculty of Health Sciences, McMaster University
December 9, 2019

Researchers at McMaster University have unlocked one of the secrets behind the many benefits of metformin.

One of the most widely used medications in the world; metformin is commonly prescribed for Type 2 diabetes. However, in addition to its effects on lowering blood sugar, in preclinical models, metformin shows benefits on aging and a number of diverse diseases such as cognitive disorders, cancer and cardiovascular disease.

One question researchers have been asking is how this is being achieved. A multi-year study led by a collaboration of McMaster's basic science and clinical researchers has found that metformin induces the expression and secretion of a protein called growth differentiating factor 15, or GDF15.

The results were published today in *Nature Metabolism.*

"Studies over the past two decades have shown that metformin does more than lower glucose, but we haven't understood why," said Gregory Steinberg, senior author and professor of medicine at McMaster. He is also co-director of the Centre for Metabolism, Obesity and Diabetes Research at McMaster.

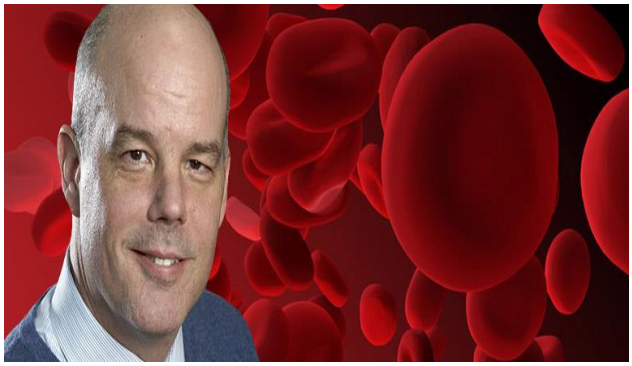
"We went into this study with the idea that metformin might communicate with other tissues in the body by causing the secretion of a protein from the liver. We were totally surprised when we found out that metformin caused the secretion of GDF15, a protein which is known to suppress appetite."

The study team took that knowledge and applied it to mice to better understand the science behind the outcome. Scientists deleted the gene that makes GDF15 in mice, then treated them with metformin. The results showed that mice without GDF15 did not eat less or lose weight, despite being administered metformin, establishing GDF15 as the connection between metformin and weight loss.

"The possibility that GDF15 has a role in multiple beneficial effects of metformin treatment on aging or diseases like cancer needs to be studied," Steinberg said.

The study was funded by the Canadian Institutes of Health Research (CIHR) and Diabetes Canada.

Researchers discover autoantibody in blood that accelerates underlying cause of cardiovascular disease



Rick Austin

December 20, 2019
Modified from: Veronica McGuire - Faculty of Health Sciences, McMaster University

A new study published in the *Journal of Clinical Investigation Insight* has identified a unique autoantibody directed against a cellular protein named GRP78 which accelerates atherosclerosis, the underlying cause of cardiovascular disease.

The research team, led by McMaster vascular biologist Richard Austin, has now demonstrated that anti-GRP78 autoantibodies can bind to GRP78 on the surface of lesion-resident endothelial cells and speed up atherosclerosis.

The study also demonstrated that mouse models of atherosclerosis as well as patients with established cardiovascular disease have significantly elevated blood levels of these autoantibodies that both correlate and contribute to disease progression.

"Normally, GRP78 is embedded in cells where it acts as a molecular chaperone to assist in protein folding. However, we made the surprising discovery that GRP78 moves to the surface of lesion-resident endothelial cells and activates the immune system to generate anti-GRP78 autoantibodies," said Austin, the senior author and professor in the Department of Medicine at McMaster.

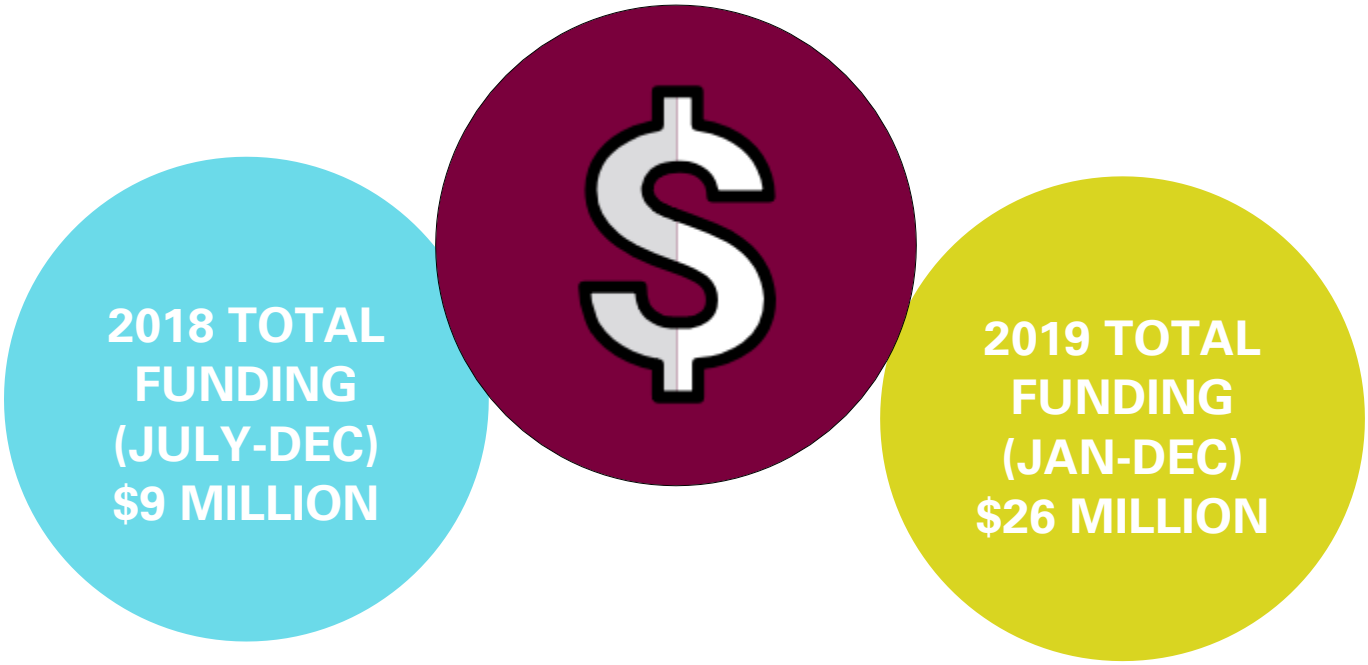
"This increase in the levels of anti-GRP78 autoantibodies have the unique ability to bind to GRP78 on the surface of endothelial cells which drives several critical stages of atherosclerosis," added Ali Al-Hashimi, co-first author and Austin's postdoctoral fellow. "This is what accelerates atherogenesis and hence the increase in cardiovascular disease."

The research was a collaborative effort with Wadih Arap and Renata Pasqualini from Rutgers Cancer Institute of New Jersey and Katey Rayner from University of Ottawa.

"The observation that the FDA-approved and commercially available drug enoxaparin (Lovenox) may protect against atherosclerotic lesion growth and may stabilize the plaque by specifically disrupting the binding of anti-GRP78 autoantibodies to cell surface-associated GRP78 is mechanistically interesting and could well open up new therapeutic strategies," added Dr. Arap, director of Rutgers Cancer Institute of New Jersey in Newark and chief of the Division of Hematology/Oncology at Rutgers New Jersey Medical School.

The study was funded by the Canadian Institutes of Health Research and the Heart and Stroke Foundation of Canada.

GRANT FUNDING



Aboca Company

AFP Innovation Fund

AMGEN Inc -Investigator initiated grant

Canada Foundation for Innovation (CFI) John R. Evans Leaders Fund

Canadian Institutes of Health Research (CIHR)*53

CISCO Systems Canada

Clinical Research Institute of Montreal (IRCM)

Collaborative Health Research Projects (CHRP) Grant

Crohn's and Colitis Canada Grant-in-Aid

Crohn's and Colitis Foundation, USA Research Award

Cystic Fibrosis Canada (CFC) Research Operating Grant

David Braley Center for Antibiotic Discovery

Department of Anesthesia Academic Fund - Hamilton Health Sciences

Department of National Defense (DnD): Innovation for Defense Excellence and Security

Diabetes Canada *2

EJ Moran Campbell Early Career Research Award Funding sources: McMaster University

Eli Lilly & Co
ERA: Early Researcher Award

Genome Canada Bioinformatics and Computational Biology Competition

Genomic Applications Partnership Program (GAPP)

Hamilton Health Science RFA

HAHSO
Hamilton Community Foundation, Abacus, Enhancement, Education Research

Hamilton Health Science Foundation *2

Hamilton Health Sciences Foundation New Investigator Fund *2

Hamilton Health Sciences Foundation Research Program support

Hamilton Health Sciences Research Strategic Initiative Program

Hamilton Health Sciences RFP: Digital Health

Health Canada Operating Grant

Heart and Stroke Foundation of Canada *6

Kidney Foundation of Canada (KFOC)

Labarge Centre for Mobility on Aging: Targeted Call for Proposals on Nutrition, Exercise and Metabolism
Labatt Heart Centre Innovation Fund

McMaster Institute for Research in Aging | Collaborative for Health & Aging.
Ontario SPOR Support Unit
McMaster University Department of Orthopedics

McMaster University Faculty of Science Research Infrastructure Renewal Fund *2

McMaster University Student Life Enhancement Fund
McMaster University, Strategic Alignment Fund, Education Research

Ministry of Research, Innovation and Science, Early Researcher Awards

Mitacs Accelerate Grant

Mitacs McMaster University- Espervita Therapeutics Fellowship Support

Myos Technology

National Cattlemen's Beef Association

National Health and Medical Research Council of Australia *3

National Institutes of Health (NIH)

Natural Sciences and Engineering Research Council of Canada (NSERC) *11

Northern Ontario School of Medicine (NOSM) Internal funding

Novo Nordisk Canada Inc (Mississauga, ON) Investigator Initiated Grant
Ontario Ministry of Health and Long-Term Care Academic Health Sciences Centre Innovation Fund *2

Ontario Ministry of Research, Innovation, and Science Early Researcher Award*2
Ontario Research Fund *2

Population Health Research Institute *3

Roquette Frères

Sanofi-Aventis (Canada) * 2

Sidewalk Toronto Small Research Grants
Social Sciences and Humanities Research Council of Canada (SSHRC) *3

The Research Institute of St. Joe's Hamilton, Teresa Cascioli Charitable Foundation Research Award in Women's Health

Together in Pink Association Against Cancer in Women

Ventech Solutions

Weston Family Microbiome Initiative. Operating Grant

MODR SEMINARS & EVENTS

The Centre runs bi-weekly seminars featuring graduate students, faculty members of McMaster University and collaborating universities and clinicians. These events are accredited by the Royal College of Physicians and Surgeons of Canada and adhere to their accreditation standards.

2018 Events

Dr. David Carling, MRC London Institute of Medical Sciences, Imperial College
Investigating the regulation of energy metabolism by AMPK in vivo

Dr. Geoff Werstuck, McMaster University
Why do people with diabetes die from heart attacks and strokes?

Dr. Sandeep Raha, McMaster University
Exploring the role of mitochondrial signaling in placental function

Dr. Graham Holloway, University of Guelph
Mitochondrial bioenergetics during exercise and disuse situations

Dr. Laura Anderson, McMaster University
Issues in Childhood Obesity: From measurement to evaluation of a population health intervention

2019 Events

Dr. Chris McGlory, McMaster University
Strategies to counteract muscle loss during period of physical activity

Dr. Jason Dyck, University of Alberta
Connecting Diabetes & Heart Failure

Tamana Yousuf, PhD Student, McMaster University
Investigating the role of hepatic T-cell death associated gene 51 protein in obesity and insulin resistance

Khrystyna Platko, PhD Candidate, McMaster University
Investigating the role of hepatic T-cell death associated gene 51 protein in obesity and insulin resistance

Dr. Maureen McDonald, McMaster University
Can you "train" your arteries? Assessing the impact of exercise training on blood vessel structure and function in health and disease

Kelly Bradbury, MSc Candidate, McMaster University
Investigating the beverage patterns of children and adolescents at the time of enrollment into Canadian pediatric weight management programs

Jodi Rabeneck, MSc Candidate, McMaster University
Development and utilization of an enhanced in-vitro model of skeletal muscle

Donna Fitzpatrick-Lewis, McMaster University
Systematic Review Evidence: Let's get the questions right

Emily Day, PhD Candidate, McMaster University
GDF15 is a metformin stimulated hepatokine that is important for promoting weight loss

Katherine Kennedy, PhD Candidate, McMaster University
Impact of pre-pregnancy BMI on maternal gut microbiota over the course of pregnancy

Dr. Paola Muti, McMaster University
Could melatonin be beneficial for keeping breast cancer away?

Dr. Michaela Devries-Aboud, University of Waterloo
How exercise, sex and IMCL influence muscle metabolism and glycemic control

Shuman Zhang, MSc Candidate, McMaster University
The environmental toxin, chlorpyrifos disrupts mitochondrial function in brown adipose tissue and promotes the development of obesity and metabolic dysfunction in mice

Caroline Seiler, MSc Candidate, McMaster University
Dietary triggers of gastrointestinal symptoms in IBS

Dr. Cameron McAlpine, Harvard Medical School
Sleep modulates hematopoiesis and protects against atherosclerosis

Dr. Nabil Seidah, University of Montreal
The proprotein convertases: building bridges between fundamental research and clinical applications

Dr. Laura Anderson, McMaster University
Measurement of obesity in population-based studies across the lifecourse

Julian Yabut, PhD Candidate, McMaster University
Harnessing Transferable Skills Learned in Graduate Training: Meeting the Needs of Non-Academia

Dr. Mueez U-Din, Postdoctoral Fellow, McMaster University
The application of medical imaging method to study human adipose tissue metabolism

Dr. Evangelia Tsiani, Brock University
Muscle cell glucose update: Use of novel polyphenols against insulin resistance

Dr. Arthur Cheng, York University
Calcium and its role in regulating skeletal muscle force and fatigue in chronic diseases

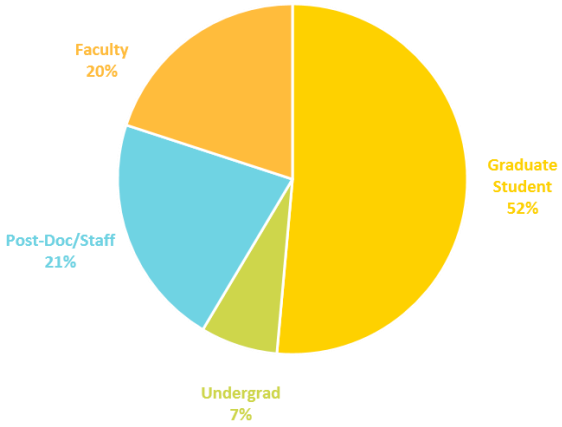
Michael Wong, PhD Candidate, McMaster University
The design and fabrication of microphysiological in vitro systems to model the human placenta

Dr. Basma Ahmed, PhD Candidate, McMaster University
Investigating the relationship of brown adipose tissue, non-alcoholic fatty liver disease and gut microbiota in human adults

SYMPOSIUM SUMMARY

2018
6th Annual Symposium
Metabolism, Obesity & Diabetes

- Official launch of the Centre
- Total attendance = 70



2019
7th Annual Symposium

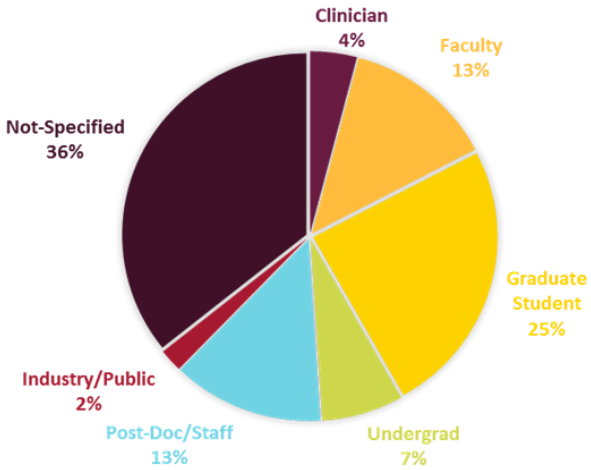
Non-Alcoholic Fatty Liver Disease (NAFLD): A Global Epidemic with Implications for Diabetes, CVD and Cancer

- Total attendance = 98

NEW THIS YEAR

- Trainee abstract competition
  - Obtained outside unrestricted funding from Lilly

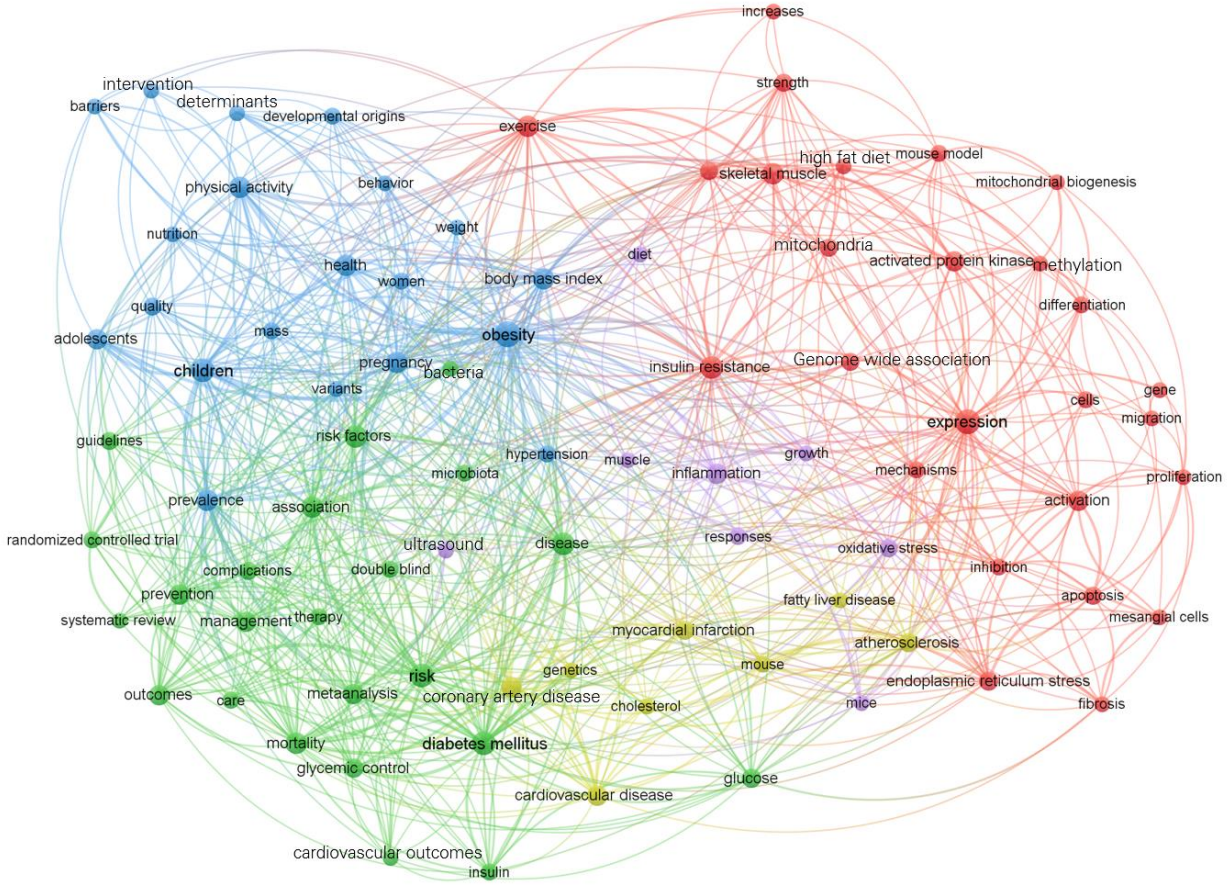
Lilly CANADA



PUBLICATIONS

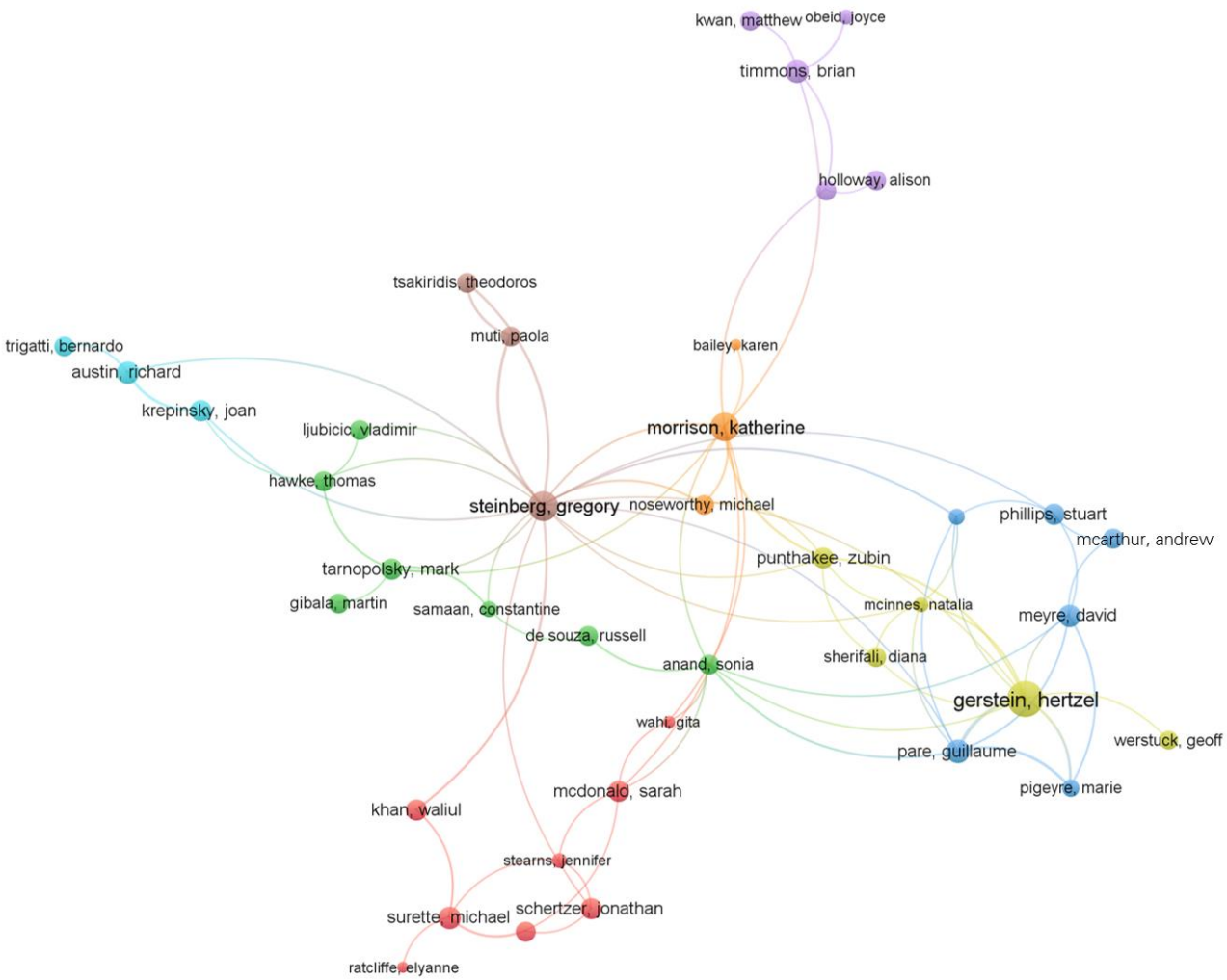
MODR Research Scope

The scope of the research work that our members are involved in is highlighted in the figure below. This figure represents a map of the subject keywords found in members publication citations from 2018-2019. This figure was generated using VOSviewer; keywords that occur at least five times in titles and abstracts of the publications are represented by an individual node on the map. The size of the subject category (node) indicates publication activity in that subject (i.e. a larger node indicates that more articles contain the keyword) and the smaller the space between the nodes, the more often those keywords co-occur.



Co-authorship between MODR Members is illustrated in the figure below. Web of Science was utilized to collect author citations (between 2018-2019) and from those citations, a network visualization of co-authorship was generated using VOSviewer. A thicker line indicates an increased number of co-authorship publications; a larger circle indicates the document number.

Co-Authorship between MODR Members



JULY-DEC 2018 PUBLICATIONS

Altmetric

Who is talking about our research?

Average Altmetric Score

Mentioned by:

- 4280 tweeters
- 2207 Facebook posts
- 5230 readers on Mendeley
- 1499 citations

169 papers published

Abderrahmani A, Yengo L, Caiazzo R, Canouil M, Cauchi S, Raverdy V, Plaisance V, Pawlowski V, Lobbens S, Maillet J, Rolland L, Boutry R, Queniat G, Kwapich M, Tenenbaum M, Bricambert J, Saussenthaler S, Anthony E, Jha P, Derop J, Sand C, Rabearivelo I, Leloire A, **Pigeyre M**, Daujat-Chavanieu M, Gerbal-Chaloin S, Dayeh T, Lassailly G, Mathurin P, Staels B, Auwerx J, Schürmann A, Postic C, Schafmayer C, Hampe J, Bonnefond A, Pattou F, Froguel P. Increased Hepatic PDGF-AA Signaling Mediates Liver Insulin Resistance in Obesity-Associated Type 2 Diabetes. Diabetes. 2018 Jul; 67 (7):1310-1321. Epub 2018 May 4. doi.org/10.2337/db17-1539

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