QGCS 18th Annual Symposium Keynote Lecture

DNA Nanostructures: From Design to Targeted Cancer Therapy

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11:00 am
October 23rd, 2025
Chernoff 202

DNA is universally recognized as the molecule of life, encoding the genetic instructions that define us. But the very attributes that make it such an effective information carrier also make it an exceptional material for constructing nanoscale objects.

In our work, we use DNA to create three-dimensional structures, such as cages, nanotubes, and DNA-polymer nanostructures, that hold great promise for precise drug delivery. These assemblies can be finely tuned in size, shape and molecule presentation. They can encapsulate drug cargo and only release it in response to disease-related biological signals. They resist enzymatic breakdown, block the production of disease-causing proteins, and show favourable biodistribution in vivo, pointing to new opportunities in targeted cancer treatment. We will also describe how small molecules can redirect DNA assembly into completely new structures, extending the possibilities of this biomaterial beyond classical base-pairing.

Hosted by QGCS and C2MCI

QGCS 18th Annual Symposium Professor Highlight Lecture

Genome mining for "extremozymes" – Extremophiles as sources of novel biocatalysts

Dr. Graeme Howe
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10:00 am
October 24th, 2025
Chernoff 202

Directed evolution has enabled the development of extremely useful biocatalysts that have, in some cases, supplanted traditional organic chemistry in the industrial production of value-added commodity chemicals. Generally, mutations introduced into a protein scaffold to increase catalytic efficiency are accompanied by a compensatory destabilization of the enzyme. To circumvent this issue and allow a more thorough exploration of sequence space around naturally occurring enzymes, we have turned to sequence similarity networks (SSNs) to mine the genomes of thermophilic microorganisms to identify novel thermostable variants of enzymes with potential industrial utility. These networks allow for the exploration of whole protein families and the interrelatedness of every member sequence through an 'all-by-all' BLAST. Through the iterative construction of SSNs with varying sequence identity cutoffs, we have produced networks of putative isofunctional clusters that allow a single sequence with known catalytic function to reveal the role of thousands of unannotated PETase-like genes.

Our initial efforts in this arena have focused on the search for new thermostable plastic-degrading enzymes as a part of the OpenPlastic consortium's efforts to develop a circular plastics economy. Following the initial discovery of a cutinase-like enzyme from Ideonella sakaiensis that degrades polyethylene terephthalate (PET), there has been an explosion in research revolving around the biocatalytic degradation of PET and other plastics. While several engineered PETases have emerged that are sufficiently stable and catalytically efficient for industrial PET degradation, we opted to direct our initial efforts to exploit the wealth of PETase sequence-function relationships to mine the genomes of extremophiles for new PETases as starting points for further engineering efforts. Using the I. sakaiensis enzyme as a seed sequence, SSNs were constructed that led to the identification of 10 putative PETases from bacteria with optimal growth temperatures ranging from 50 °C to 80 °C. This presentation will detail our efforts to characterize these enzymes and their potential utility in the degradation of PET plastics. Similar bioinformatics-driven approaches to identify and characterize thermostable enzymes that degrade polyamides and polyurethanes will also be presented.

Hosted by QGCS

QGCS 18th Annual Symposium Seminar

Understanding Alzheimer's Disease at the Molecular Level

***Dr. Donald F. Weaver*[1,2]**
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[1]Krembil Research Institute, UHN, Toronto

11:30 am
October 24th, 2025
Chernoff 117

Alzheimer's disease (AD) is the most common type of dementia affecting 52 million people worldwide. It is a lethal neurodegenerative disorder that chronically affects memory and cognition. There are no curative or disease modifying agents available for the treatment of AD. A major hurdle in the discovery of such agents is the fact that the underlying mechanisms that cause AD are not understood and have not been elucidated. The time-honoured notion that AD is caused by misfolding and aggregation of a protein called β-amyloid (Aβ) has failed to deliver any useful small molecule therapeutics. AD is in need of new therapeutic directions. Based upon an extensive series of molecular modelling simulations (at the QM and MM levels), we have devised a new molecular-level mechanistic model of AD. In this model Aβ is reconceptualised as an antimicrobial immunopeptide which mistakes the molecular topography of neuronal membrane surfaces as bacterial membrane surfaces, subsequently mistakenly attacking host neurons rather than bacteria. This error in innate immunity processing culminates in neuronal death and the clinical presentation of AD. Exploiting this novel model for purposes of rational drug design and development via medicinal chemistry will be discussed.

Hosted by Dr. Diane Beauchemin