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Schedule at a Glance

Tuesday, 17th February	Wednesday, 18th February	Thursday, 19th February
GO-Week '26 Opening 9:00 am	Workshops Block I 9:00 am – 12:00 pm	Workshops Block III 9:00 am – 12:00 pm
Symposium Session I 9:10 am – 10:30 am	Morning Tea Available 10:30 am – 11:00 am	Morning Tea Available 10:00 am – 11:00 am
Morning Tea 10:30 am – 11:00 am	Break 12:00 pm – 1:00 pm	Break 12:00 pm – 1:00 pm
Symposium Session II 11:00 am – 12:30 pm	Poster Judging Session II 12:30 pm – 1:00 pm	Ira Rangahau 1:00 pm – 4:00 pm
Lunch 12:30 pm – 1:30 pm	Workshops Block II 1:00 am – 4:00 pm	Awards and Closing 4:00 pm – 4:15 pm
Poster Judging Session I 1:00 pm – 1:30 pm	Afternoon Tea Available 2:00 pm – 3:00 pm	Social Event 4:30 pm – 6:00 pm
Symposium Session III 1:30 am – 3:00 pm		
Afternoon Tea 3:00 pm – 3:30 pm		
Symposium Session IV 3:30 am – 5:00 pm		

Symposium

Date: Tuesday, 17th February
Venue: Department of Biochemistry, Seminar Room G13
Time: *9:00 am – 5:00 pm*

Zoom Link: *https://otago.zoom.us/j/8323949924?pwd=NitZS0VkYXRaN0RiL0Z4N2lDTGdHZz09*
Meeting ID: 832 394 9924, Password: 750757

Symposium		
9:00 am - 9:10 am	Mihi Whakatau Welcome and Opening	Professor Phillip Wilcox *Kaikōkiri Māori, Genetics* Professor Louise Bicknell *GO Co-Director*
Session One		Chair: Logan Walker
9:10 am – 9:55 am	Genetics Otago: A template for transdisciplinary research and education at the University of Otago	Distinguished Professor Greg Cook *DVC Research and Innovation*
9:55 am – 10:15 am	Genetic vulnerabilities in rifampicin resistant strains of *Mycobacterium tuberculosis*	Dr Matthew McNeil *Department of Biochemistry*
10:15 am – 10:30 am	Towards One Biosecurity: CRISPR-based environmental biosurveillance and artificial intelligence	Benjamín Durán Vinet *Department of Biochemistry*
10:30 am – 11:00 am Break		

Session Two		Chair: Hamish Salvesen
11:00 am – 11:15 pm	Where's wallaby? Using environmental DNA to detect mobile, elusive terrestrial pests	Gracie Kroos *Department of Anatomy*
11:15 pm – 11:30 pm	SULT1A1 Inhibition and the Prevention of *BRCA1*-Associated Breast Cancer (Via Zoom)	Emily Young *Department of Pathology and Molecular Medicine, Christchurch*
11:30 pm – 12:30 pm	**Keynote:** Adaptation in a fast-changing world: new insights from Aotearoa	Professor Jon Waters *Department of Zoology*
12:30 pm – 1:30 pm Lunch (poster judging)		

Session Three		Chair: Sankalita Ray Das
1:30 pm – 2:05 pm	Building Gene Drive Capacity in Haplodiploid Insects How to Rear your Parasitoid: Optimising *in vitro* rearing for the genetic model *Nasonia vitripennis* Functional Characterisation of Polynesian-Enriched Non-Coding Variants Utilizing a Novel Method of Zebrafish Transgenesis	Hamish Salvesen *Department of Biochemistry* Jacob Grupp *Department of Biochemistry* Caleb Calhoun *Department of Physiology*

	Subspecies or remnants of ancient diversity: functional genomic variation between NI and SI kākā	Charlotte Koenig *Department of Anatomy*
2:05 pm – 2:25 pm	From Ice Age to isolation: Historical demography and inbreeding depression in New Zealand's endemic Hector's and Māui dolphins	Dr Alana Alexander *Department of Anatomy*
2:25 pm – 2:40 pm	From genomes to microbiomes: predictors of infectious disease vulnerability in Māui and Hector's dolphins	Sebastian Alvarez Costes *Department of Anatomy*
2:40 pm – 3:00 pm	Whaia te mātauranga: Education Resources and Initiatives to Enhance Māori Participation in Modern Genetics and Genomics	Professor Phillip Wilcox *Department of Mathematics and Statistics*
3:00 pm – 3:30 pm Break		

Session Four		Chair: Hannah Darroch
3:30 pm – 3:50 pm	Can we use genetics to improve cardiovascular risk prediction?	Associate Professor Anna Pilbrow *Department of Medicine, Christchurch*
3:50 pm – 4:05 pm	The tumour-resident microbiome and methylation in colorectal tumours (Via Zoom)	Dr Arielle Sulit *Department of Surgery and Critical Care, Christchurch*
4:05 pm – 4:20 pm	Pathogenic biallelic variants in VPS52, encoding an intracellular trafficking protein, cause impaired brain development	Dr Sankalita Ray Das *Department of Biochemistry*
4:20 pm – 4:35 pm	Genome Assembly and Annotation of the Invasive Bryozoan *Bugulina flabellata*: A Hybrid Sequencing Approach	Ismael Chowdhury *Department of Biochemistry*
4:35 pm Close		

Workshop Descriptions

Workshops Block I

Variant Classification: Making Sense of Genetic Variants

Date: *Wednesday, 18th February*
Time: *9:00 am – 10:30 am*
Venue: *St David Seminar Room E*

This workshop provides an interactive overview of current ACMG guidelines. Through hands-on cases, participants will be guided through variant filtration and curation using current tools and resources.

Participants will need their own laptop with Excel installed.

Single Cell Sequencing Workshop (Part 1)

Date: *Wednesday, 18th February*
Time: *9:00 am – 10:30 am*
Venue: *Biochemistry, G13*

Hosted by the Single Cell Sequencing and CRISPR Hubs in collaboration with Illumina.

Revolutionising scRNA-Seq accessibility: Illumina's instrument free PIPseq solution and CRISPR screening tool
As one of the fastest-growing next-generation sequencing innovations, single-cell approaches empower every lab to push the boundaries of discovery. Illumina's Single-Cell 3' RNA Prep offers a simple, cost-effective solution designed to democratize single-cell sequencing, enabling researchers across diverse fields—from neuroscience and cancer biology to marine and crop science—to unlock deeper insights. Powered by PIPseq™ technology, this innovative system uses emulsion-based Particle-templated Instant Partitions (PIPs) to efficiently partition complex cell mixtures, paving the way for seamless multi-omic studies, fully supported by the Illumina ecosystem.

Epigenetics Workshop

Date: *Wednesday, 18th February*
Time: *11:00 am – 12:30 am*
Venue: *Biochemistry, G13*

Hosted by the Epigenetics User Group Hub in collaboration with Illumina.

Illumina: Unlocking the Power of Illumina 5-Base for Genomic & Epigenetic Discovery
The Illumina 5-Base Solution represents a breakthrough in multiomic research, enabling simultaneous whole-genome sequencing and methylation profiling in a single, streamlined workflow. By integrating high-accuracy variant detection with comprehensive epigenetic insights, researchers can uncover deeper biological mechanisms, accelerate biomarker discovery, and advance precision medicine. This technology empowers scientists to explore the interplay between genetic code and epigenetic regulation with unprecedented efficiency and scale. Join our upcoming workshop to explore how to harness the Illumina 5-Base platform for your next research breakthrough.

Workshops Block II

ELSC Workshop

Date: *Wednesday, 18th February*
Time: *1:00 pm – 2:30 pm*
Venue: *St David Seminar Room E*

Considering epistemic justice in informed choice for genomic testing

Presented by Sara Filoche

Epistemic justice is when people's positions as a knower and producer of knowledge are upheld. Epistemic justice considers people's epistemological norms (e.g., people's different and varied worldviews and ways of acquiring knowledge), social justice (e.g., human rights and equity) and ethical practice (e.g., autonomy). There are two ways that this can broadly occur. Hermeneutical justice is upheld when people have, for example, the resources and language to express and make sense of the experience to, and for, them. Testimonial justice occurs when people's testimonies of their experience are believed as being credible. The converse occurs in instances of hermeneutical and testimonial injustices. Epistemic injustices are inextricable from injustices associated with race, gender, disability, sexuality and socioeconomic status.

In a US-based project, people were offered prenatal screening for a range of genetic conditions participants shared experiences such as *'I was left in a position where I was like, I don't even know what any of this means. Then I turned to Dr Google, which I do not recommend'*. Another person from the same project shared, *'Maybe if someone would've went through with me, and really explained it, maybe that would've made it a little bit easier to process'*. These examples highlight the importance of considering the hermeneutics of informed choice.

Who gets to decide what information is important for people to know? And how do we make report decisions as informed ones? Informed choice as an epistemic justice consideration has not been widely explored, but it promises new avenues for clinical communication approaches and policy implications.

Single Cell Sequencing Workshop (Part 2)

Date: *Wednesday, 18th February*
Time: *1:00 pm – 2:30 pm*
Venue: *Biochemistry, G13*

Hosted by Millennium Science.

Enabling Discovery at Scale with 10x Genomics Single Cell and Spatial Technologies
Advances in single-cell and spatial genomics are rapidly transforming our ability to study biological systems with unprecedented resolution and scale. In this presentation, we will provide a high-level overview of 10x Genomics technologies, highlighting how these platforms enable researchers to interrogate cellular diversity, cell states, and spatial organisation across a wide range of biological questions.

This session is designed for researchers who are new to single-cell or spatial approaches, as well as those seeking a broader understanding of how these tools fit together within a unified experimental ecosystem. The aim is to provide clarity on the capabilities, flexibility, and advantages of the 10x Genomics platforms.

Workshops Block III

Beyond the Bench: Decoding Science Communication Workshop

Date: *Thursday, 19th February*
Time: *10:00 am – 12:00 pm*
Venue: *Biochemistry, G13*

A career in genetics extends beyond laboratory results and literature reviews—it also involves communicating research clearly and effectively. Whether you are preparing for a presentation, developing a grant idea, or seeking to share your work with broader audiences, this workshop is designed to support you.

Join colleagues from Genetics Otago for a morning focused on science communication. The session will bring together experienced communicators to provide practical guidance on refining your message and engaging diverse audiences.

The programme will begin with an in-depth session led by A/Prof Nic Rawlence, followed by a practical briefing from Ellie Rowley from the University of Otago Communications Division. The workshop will conclude with an open Q&A panel featuring Dr Claire Concannon(Radio New Zealand) and social media expert communicator Taylor Davies-Colley (@TayTalksTrees).

Decode Science CRISPR Workshop

Date: *Thursday, 19th February*
Time: *9:00 am – 12:00 pm*
Venue: *St David Seminar Room D*

Novel CRISPy perspectives

Join an engaging CRISPR seminar series showcasing the breadth of modern applications across molecular biology. This session brings together leading researchers to explore advances ranging from epigenetic methylation, synthetic biology, to airborne environmental DNA surveillance. Presentations will highlight innovations in CRISPR-Cas technologies, and emerging research frontiers applicable to CRISPR-Cas technologies. Our aim is to provide a space within Otago that fosters discussion, novel frontiers and collaboration while connecting foundational science with the rapidly expanding potential of CRISPR-based tools.

Ira Rangahau

Date: Thursday, 19th February
Venue: *Department of Biochemistry, Seminar Room G13.*
Time: *1:00 pm – 4:00 pm*

Zoom Link: *https://otago.zoom.us/j/8323949924?pwd=NitZS0VkYXRaN0RiL0Z4N2lDTGdHZz09*
Meeting ID: 832 394 9924, Password: 750757

	Ira Rangahau – Biochemistry Seminar Room G13	
1:00 pm – 1:05 pm	Welcome and Introduction	Dr Megan Leask *GO Deputy Co-Director, Department of Physiology*
1:05 pm – 1:35 pm	Epigenetic Signatures of Aging and Disease Risk	Dr Victoria Sugrue (via Zoom) *Department of Pathology and Molecular Medicine, Christchurch* *Postdoctoral Fellow*
1:35 pm – 1:55 pm	Pioneering precision medicine for Māori and Pacific people: Estimating metabolic disease-specific (gout) polygenic risk scores (PRS) to find 'biological hubs'	Ben Rangihuna *Department of Physiology* *Assistant Research Fellow*
1:55 pm – 2:15 pm	Antenatal Depression and the Gut Microbiome	Thalia Heiwari (via Zoom) *Department of Molecular Medicine, Wellington* *PhD student*
2:15 pm – 2:30 pm	Niches and Networks: Building community-led genomic surveillance for resilience to evolving viral threats in Aotearoa	Lia Heremia *Department of Microbiology and Immunology* *PhD student*
2:30 pm – 2:45 pm	Genomic Insights into Wing Loss and Melanism in New Zealand Stoneflies	Kahu Hema *Department of Zoology* *PhD student*
2:45 pm – 3:05 pm	Afternoon Tea Break	
3:05 pm – 3:15 pm	The Management of Breast Cancer risk in HDGC Whānau - Te Mana o Wahine; He Puawaitanga Tuatahi	Gemella Reynolds-Hatem (via Zoom) *Department of Biochemistry* *Masters Student*
3:15 pm – 3:25 pm	Identifying epigenetic changes underpinning drug resistance in lung cancer	Safia Farry *Department of Pathology and Molecular Medicine* *PhD student*
3:25 pm – 3:35 pm	Characterising the CYP2D6*71 Allele in Aotearoa: Pharmacogenomic Insights for Māori and Pacific Peoples	Bree Holloway *Department of Physiology* *Masters student*
3:35 pm – 4:00 pm	**Quick Fire Talks**	
	Bioscaffolds as platforms for localised gene therapy	Oaklea Bowden-Morris *Honours student 2025 Department of Physiology, currently UoO Dentistry Student*

	Zebrafish tikanga: assigning function to a cardiac- relevant Polynesian specific frameshift variant in NEB Investigating disrupted reward behaviour in a preclinical mouse model of perimenopause Investigating the function of miRNA499 in cardiovascular disease using zebrafish TBC	Eilish Dalley *Department of Physiology* *Masters student* Ohaia Gillman *Department of Physiology* *Summer Research Project, UoO Medical Student* Nina Turner *Department of Physiology* *Putahi Manawa Summer Research Student* Patrick Gibbons *Department of Physiology* *UoO Medical Student*
4:00 pm	Closing	Dr Megan Leask *GO Deputy Co-Director, Department of Physiology*

Attendees are welcome to stay and attend the Genetics Otago Annual Awards Ceremony and official closing of GO-Week '26. This will then be followed by a social and networking event at Ombrellos from 4:30 pm.

Other Information

Catering and Sponsor Displays

Catering will be located on the 1st floor of the St David Lecture Theatre above the café. This will include morning tea, lunch and afternoon tea on the 17th of February, as well as light morning and afternoon tea on the 18th and 19th. Please note that catering is not supplied on the 18th and 19th of February and attendees will be required to source their own lunch during the break times. Sponsor displays will also be set up in this area. Please take some time during the breaks to engage with our sponsors; without their support, we are unable to host these events.

Posters

Posters will be on display on the 1st floor of the St David Lecture Theatre above the café. for the duration of the event. Posters can be hung from 8:00 am on the 17th of February. If you are presenting a poster, you will be notified as to which of the three judging sessions you will be participating in. Please check carefully that you attend on the correct day, and are at your poster for the duration of the session. Posters will need to be removed by 1:00 pm on Thursday 19th of February.

Poster Judging Session I
Time: 1:00 pm – 1:30 pm, Tuesday 17th February
This judging session will include even-numbered posters

Poster Judging Session II
Time: 12:30 pm – 1:00 pm, Wednesday 18th February
This judging session will include odd-numbered posters

Poster prizes, sponsored by Illumina, will be awarded to the top two posters as determined by the judges. These awards will be presented at the close of the Ira Rangahau session on the 18th of February at 4:00 pm. Please try to attend this.

Awards

Awards will be presented by Genetics Otago Co-Directors, at the conclusion of Ira Rangahau (4:00 pm on the 18th of February). Awards to be presented are:

- The 2026 Genetics Otago Award
- Mentor Award
- Student Supervisor Award
- ***Decode Science*** *and* ***Millennium Science*** Publication Awards
- ***Illumina*** Poster Awards
- Science Communication Award

Abstracts
Keynote Speakers

Adaptation in a fast-changing world: new insights from Aotearoa
Jon Waters
Department of Zoology, University of Otago, Dunedin

How are organisms adapting to the planet's fast-changing ecosystems? Our recent research highlights dramatic evolutionary shifts in native species facing human-driven environmental change. While most recent studies have focused on traits under simple genetic control, the mechanisms driving the evolution of more complex phenotypes ('ecotypes') in response to environmental change remain unresolved. Traditional evolutionary theory suggests that ecotype evolution, controlled by multiple genes, should require recombination over thousands of years (transporter hypothesis). However, emerging data from our lab call this model into question, with evidence that key adaptive alleles may be co-located and carried in-tact, with potential for 'jackpot' individuals to fuel rapid evolution of complex ecotypes.

Invited Speakers

From Ice Age to Isolation: Historical Demography and Inbreeding Depression in New Zealand's Endemic Hector's and Māui Dolphins
Alvarez-Costes, S.[1], Baker, C.S.[2], Constantine, R.[3], Carroll, E.L.[3], Reeves, I.M.[4], Dutoit, L.[5], Ferreira, S.[1], Heimeier, D.[3], Gemmell, N.J.[1], Gillum, J.[1], Hamner, R.M.[2], Rayment, W.[6], Roe, W.[7], Te Aikā, B.[8], Alexander, A.[1]

[1]*Department of Anatomy, School of Biomedical Sciences, University of Otago, Dunedin, Aotearoa New Zealand,*
[2]*Marine Mammal Institute and Department of Fisheries, Wildlife, and Conservation Sciences, Oregon State University, Newport OR, USA,*
[3]*School of Biological Sciences, University of Auckland, Auckland, Aotearoa New Zealand,*
[4]*College of Science and Engineering, Flinders University, Bedford Park, South Australia, Australia,*
[5]*Department of Zoology, University of Otago, Dunedin, Aotearoa New Zealand,*
[6]*Department of Marine Science, University of Otago, Dunedin, Aotearoa New Zealand,*
[7]*School of Veterinary Science, Massey University, Palmerston North, Aotearoa New Zealand,*
[8]*Research and Enterprise, University of Otago, Dunedin, Aotearoa New Zealand.*

Hector's and Māui dolphins, endemic to Aotearoa, New Zealand, are small coastal dolphins facing significant anthropogenic threats. The IUCN lists the ~15,000 Hector's dolphins (Te Waipounamu/South Island) as endangered, while the Māui dolphin (Te-Ika-a-Māui/North Island), with only ~54 individuals, is critically endangered. We assessed the demographic history and population structure of both subspecies using whole genome data from 48 individuals. Palaeoceanographic trends have shaped contemporary admixture patterns and population structure, with the closure of Te Moana-o-Raukawa/the Cook Strait separating the Māui and Hector's dolphins during the LGM, and with productive regions (East/West Coast) acting as sources for less favourable habitats (South Coast). Māui dolphins diverged from Hector's dolphins ~12–16 kya and exhibit reduced genetic diversity, inbreeding depression, and higher genetic load, confirming the previously reported genetic decline in the Māui dolphin, which could severely impact its survival given their critically low population size. Similarly, the South Coast Hector's population shows elevated inbreeding compared to the larger, more diverse East and West Coast populations. Admixed individuals at population edges display higher genetic diversity, emphasizing the importance of protecting migratory corridors. Conservation strategies must prioritize migratory corridors while assessing adaptive variation and deleterious alleles in local populations to ensure the recovery of these subspecies.

Genetic vulnerabilities in rifampicin resistant strains of *Mycobacterium tuberculosis*

XinYue Wang [1,2], William J Jowsey [1,2] , Chen-Yi Cheung [1] , Nina Dickerhof [3] , Jamie RH Taka [4] , Mark B Hampton [3] , Ghader Bashiri [4] , Paul Gardner [5,6] , Peter C Fineran [1,2,6,7] , Gregory M Cook [1,8] , Simon A Jackson [2,7,9] , Matthew B McNeil [1,2 *]

[1] *Department of Microbiology and Immunology, University of Otago, Dunedin, New Zealand*
[2] *Maurice Wilkins Centre for Molecular Biodiscovery, University of Auckland, Auckland, New Zealand.*
[3] *Mātai Hāora – Centre for Redox Biology and Medicine, Department of Pathology and Biomedical Science, University of Otago Christchurch, Christchurch, New Zealand*
[4] *School of Biological Sciences, The University of Auckland, Auckland, New Zealand.*
[5] *Department of Biochemistry, University of Otago, Dunedin, New Zealand*
[6] *Genetics Otago, University of Otago, Dunedin, New Zealand*
[7] *Bio-Protection Research Centre, University of Otago, Dunedin, New Zealand*
[8] *School of Biomedical Sciences, Queensland University of Technology, Translational Research Institute, Woolloongabba, Queensland 4000, Australia*
[9] *School of Pharmacy and Biomedical Sciences, University of Waikato, Hamilton, New Zealand*

The global health burden caused by *Mycobacterium tuberculosis* is aggravated by the emergence and spread of drug resistance. Mutations that cause drug resistance can have collateral effects that increase the vulnerability of downstream pathways to inhibition. Here, using genome scale CRISPR interference we identified collateral effects associated with rifampicin resistance in *M. tuberculosis*. We demonstrate that the rifampicin-resistant mutant RpoB(S450L) had increased sensitivity to the dysregulation of sulphur metabolism due to transcriptional dysregulation, although this vulnerability did not translate to all *rpoB* genotypes. These collateral vulnerabilities are affecting the evolution of mycobacteria in clinical settings by influencing the distribution of non-synonymous potentially adaptive mutations in genes involved in sulphur metabolism. Finally, we demonstrate that these metabolic vulnerabilities in rifampicin-resistant mutants can be extended to the metabolism of host relevant carbon sources. Collectively, this work highlights how drug resistance impacts the physiology of *M. tuberculosis*, the ability of *M. tuberculosis* to adapt to host-like environments and the importance of considering how physiological variation in *M. tuberculosis* could influence outcomes of new treatment regimens.

Can we use genetics to improve cardiovascular risk prediction?

Anna Pilbrow

Department of Medicine, University of Otago Christchurch

Heart failure is a leading cause of avoidable, premature death worldwide. Any condition that damages the heart (e.g. coronary artery disease) can lead to heart failure. However, the rate of progression from coronary artery disease to heart failure varies considerably between people and is difficult to predict. Genetics may offer a new approach, identifying those at risk early in the disease course. We aimed to test whether adding genetic information to established clinical risk factors could improve accuracy of predicting incident non-fatal or fatal heart failure within 5 years among people with diagnosed coronary artery disease. Utilising large international cohorts, we found that common genetic variants associated with risk of HF can improve prediction of HF within 5 years among people living with CAD, beyond established clinical risk factors. Our data highlight the potential for genetic risk scores to add prognostic value in low risk groups for whom traditional risk factor profiling may be less accurate (e.g. women). Our data also suggest that genetic risk scores may have limited utility in those recently incurring acute coronary events and should not be used to downgrade risk in those with traditional risk factors. In summary, genetic risk scores in combination with clinical risk factors may help identify people at impending risk of HF who would benefit from more proactive management and monitoring.

The tumour-resident microbiome and methylation in colorectal tumours
Arielle Sulit, Claudia Rose Kinder, Jessica Permain, Allison Miller, Rachel Purcell

Colorectal cancer (CRC) is a heterogeneous disease manifesting differently between patients, such that they can be grouped into subtypes. One subtype of CRC develops via methylation of CRC-associated genes, leading to hypermutation, and giving rise to its hallmark microsatellite instability (MSI). Methylation patterns have previously been shown to be associated with the microbiome and its components. Our group identified a microbe, *Fusobacterium spp*, that is enriched in MSI+ CRC and showed that lipopolysaccharide (LPS) from this bacterium is associated with a decrease in the mismatch repair (MMR) gene, *MLH1*. *MLH1* defects are a known cause of MSI. We therefore hypothesised that *Fusobacterium spp* affects methylation patterns in colorectal tumours, leading to impaired expression of *MLH1* or similar genes. We then used CRC patient tumour tissue samples with known *Fusobacterium spp* abundance, and analysed their methylation patterns using Oxford Nanopore Technology (ONT)'s reduced representation methylation sequencing (RRMS). We found increased methylation in the promoter region of the *MLH1* gene in samples with higher abundances of the microbe, and corresponding decrease of *MLH1* gene expression in these samples. Results from this study will set the groundwork for future research into epigenetic effects of the tumour-resident microbiome.

Towards One Biosecurity: CRISPR-based environmental biosurveillance and artificial intelligence
Benjamín Durán-Vinet[1,2,3],Donna Huber[1,4], Sebastián Alvarez-Costes[1], Madison Salyer[5], Sara Ferreira[1], Jackson Treece[1], Antoinette Piaggio[5], Stacey Buckelew[6], Alana Alexander[1], Nathan Kenny[2], Michael Knapp[1], Htin Lin Aung[3], Neil Gemmell[1]

[1] Department of Anatomy, Faculty of Biomedical Sciences, University of Otago, Dunedin, New Zealand.
[2] Department of Biochemistry, Faculty of Biomedical Sciences, University of Otago, Dunedin, New Zealand.
[3] Department of Immunology and Microbiology, Faculty of Biomedical Sciences, University of Otago, Dunedin, New Zealand.
[4] Van Hall Larenstein University of Applied Sciences, Leeuwarden, Netherlands.
[5]United States Department of Agriculture, Animal and Plant Health Inspection Service, Wildlife Services, National Wildlife Research Center, Fort Collins, Colorado, United States.
[6]United States Fish and Wildlife Service, Invasive Species Program, Homer, Arkansas, United States.

One Biosecurity safeguards human, animal, plant, and ecosystem health by integrating science, technology, and policy to confront biological threats. CRISPR-based environmental biosurveillance platforms can enable this vision by delivering rapid, sensitive, field-deployable detection, providing real-time pre-border intelligence and coordinated post-border response through connected systems that unify fragmented biosecurity efforts using environmental nucleic acids. Biological invasions, such as *Corbicula fluminea* in the Waikato River, cost the global economy over US$423 billion annually. Introduced species often reproduce rapidly and adapt quickly, outcompeting endemic taxa and disrupting ecosystem function. Early detection therefore remains a critical challenge for effective biosecurity management. Molecular approaches based on detecting environmental DNA (eDNA) allow species biomonitoring from trace genetic material; however, PCR-based methods can be limited by inhibitors and sensitivity constraints in complex environmental samples. CRISPR-based environmental biosurveillance (CRISPR-eBx) has emerged as a powerful alternative, but assay performance depends on optimal guide RNA (gRNA) design, which remains challenging due to complex sequence–activity relationships. To address this, we have leveraged artificial intelligence (AI) to predict and rank candidate guides *in silico* under user-defined parameters. Our SENTINEL (Smart Environmental Nucleic-acid Tracking using Inference from Neural-networks for Early-warning Localisation) platform streamlines assay development for known and emerging threats. We

demonstrate sensitive and specific detection at ultra-low concentrations (10 aM, ~5 copies μL^{-1}), highlighting the integration of AI and CRISPR-eBx as a scalable framework for rapid, tailored biosecurity surveillance.

Functional Characterisation of Polynesian-Enriched Non-Coding Variants Utilising a Novel Method of Zebrafish Transgenesis

Caleb Calhoun[1], Hannah Darroch[1,2], Raheel Hussain[1], Milly Morice[1], Robert Lalonde[3], Christian Mosimann[3], Julia Horsfield[2], Megan Leask[1]

[1]*Department of Physiology, University of Otago, Dunedin, NZ,*
[2]*Department of Pathology, University of Otago, Dunedin, NZ,*
[3]*Department of Pediatrics, Section of Developmental Biology, University of Colorado School of Medicine, Anschutz Medical Campus, Auroa, CO, USA*

Chronic inflammation is a shared characteristic of many metabolic diseases, such as gout, type 2 diabetes (T2D), and inflammatory bowel disease. Incidence of these diseases are disproportionately represented across populations specific to Aotearoa, suggesting an underlying genetic contribution to development of these conditions. Utilising Polynesian-specific genomic wide association study (GWAS) data as a proxy, we have been able to identify and prioritise Polynesian-specific single nucleotide polymorphisms (SNPs) putatively associated with chronic inflammation. These SNPs are found within putative enhancer regions, non-coding elements of the genome that influence of transcription of downstream protein-coding regions. Unpublished data from our lab indicates that the putative enhancer region containing non-coding *JAZF1* variant *rs150587514* drives reporter expression (GFP) in the kidney, cerebellum, and pineal gland. Behavioural monitoring of *jazf1-/-* zebrafish larvae demonstrates a significant disruption of diurnal rhythm patterns, corroborating the expression observed in the pineal gland. Utilising a newly developed method of targeted transgene integration in zebrafish (pIGLET), we have begun efforts to characterise SNPs within *JAZF1*, *ELF1*, and *OPRL1* loci. Functional knockout populations of *elf1* and *oprl1* have also been generated via CRISPR/Cas9 genome editing, with future assays waiting to be informed from tissue expression findings via pIGLET fluorescent analyses.

Subspecies or remnants of ancient diversity: functional genomic variation between NI and SI kākā.

Charlotte Koenig, Michael Knapp

New Zealand's endemic forest parrot, the kākā (*Nestor meridionalis*), has been classified as two separate subspecies based on size and colour variation. Both the South Island kākā (*N. m. meridionalis*) and the North Island kākā (*N. m. septentrionalis*) have suffered from severe population decline and now inhabit disconnected pockets of suitable habitat. Previous reduced representation studies found no genetic basis to support the separation into subspecies. It is not known if the morphological variation in the kākā has a genetic basis, or if it is regulated by other mechanisms such as epigenetics. I will build on the reduced representation studies using our dataset of 10 high quality whole genomes per subspecies. Using Illumina short-read and ONT long-read sequencing I have created chromosome level reference genomes for each subspecies to which I have mapped the rest of my short-read data. Using comparative genomic analyses, I will explore the molecular differences between the subspecies, going from large scale inversions down to single nucleotide polymorphisms. These results will illuminate if the basis of morphological variation is genetic and if the variation between subspecies is natural or an artifact of population decline. This will inform conservation management and the validity of the subspecies classification.

SULT1A1 Inhibition and the Prevention of *BRCA1*-Associated Breast Cancer.

Emily Young[1], Devon Bull[1], Mark Hampton[2], Logan Walker[1], George Wiggins[1]

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Preventative therapy for women who are genetically predisposed to developing breast cancer is critical for improving clinical management and reducing the overall burden of cancer. Currently, the most effective risk reduction strategy is a bilateral mastectomy, where the lifetime risk of breast cancer can be reduced from ~65% to <10%. However, this is an invasive, irreversible procedure that can be significantly distressing, especially to younger women, limiting its uptake. We previously conducted the largest genome-wide copy number variant (CNV) analysis of *BRCA1* and *BRCA2* pathogenic variant carriers. This analysis revealed that deletions overlapping *SULT1A1* were associated with reduced breast cancer risk in *BRCA1* pathogenic variant carriers. SULT1A1 is a sulfotransferase enzyme important for metabolism, bioactivation and inactivation of steroid hormones, including oestrogen. To investigate the potential protective mechanism of a *SULT1A1* deletion in high-risk carriers, we analysed the expression profile of 1084 breast tumours with either a *SULT1A1* copy number deletion or diploid status. Additionally, to assess cellular impact we have tested candidate SULT1A1 inhibitors on the proliferation and metabolism of MCF-7 cells with and without *BRCA1* variants. Gene expression analysis revealed altered expression of genes associated with the oestrogen response pathway and cell cycle checkpoint regulation. Three known inhibitors of SULT1A1; ethinylestradiol, curcumin and quercetin, were tested for their effects on cell proliferation and metabolism; however, no significant findings were observed. To determine if SULT1A1 expression and candidate inhibitors are associated with enzyme activity, we are developing an *in vitro* colourimetric assay in breast cancer cell lines. Once established, these assays will enable accurate testing of SULT1A1 inhibition and provide a method for assessing inhibitor activity in *BRCA1* carriers compared to wild-type cells. These results present insight into whether SULT1A1 inhibition provides a protective effect and the potential as a preventive strategy for individuals with *BRCA1*-associated hereditary breast cancer.

Where's wallaby? Using environmental DNA to detect mobile, elusive terrestrial pests

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Wallabies, introduced to New Zealand from Australia in the late 1800s, strongly exemplify the detection challenges posed by invasive terrestrial species that are rare, cryptic, or highly mobile. Over much of their range, wallabies occur at low densities across large landscapes, making their surveillance and management challenging using standard detection tools. Recent research has demonstrated that airborne and water environmental DNA (eDNA), which refers to the genetic material constantly being shed by organisms into their environment and captured from the air or water, can rapidly identify terrestrial vertebrate diversity in an area without any visualization of the target(s). Leveraging these findings, we investigated the utility of air and water as sources of eDNA for the targeted monitoring of wallaby pest species *Notamacropus rufogriseus* in New Zealand, using a species-specific, highly sensitive probe-based quantitative PCR assay. We explore the parameters influencing detection in natural environments containing high densities of wallaby (Hakataramea Valley, South

Canterbury) and low densities of wallaby (Naseby, Central Otago) including radio-collared individuals with known locations and movements. Our results showed that airborne eDNA, as opposed to water eDNA, provides significantly higher detection rates of *N. rufogriseus* when the density of wallabies is high (> 2 wallabies per hectare). However, when the population density is low (< 0.1 wallabies per hectare), neither method could infer a positive detection, which has important implications for the application of these tools in a management setting.

Building Gene Drive Capacity in Haplodiploid Insects
Hamish Salvesen

Invasive species represent a significant threat to Aotearoa's ecosystems and the productivity of our primary sector. Gene drives offer promise as tool for non-chemical intervention for suppression of a target population. Synthetic alleles encoding the CRISPR/Cas system can be designed to copy-and-paste into the target wildtype loci in the germline of heterozygotes, resulting in skewed allele inheritance. Homing gene drives have been developed in several dipteran insect species and mice. However, it remains unclear whether gene drives will be effective in Hymenoptera, given their predisposition towards eusociality and haplodiploid genetic architecture. We are designing and developing a modular gene drive system in a model Hymenopteran, *Nasonia vitripennis*. Gene drives require both a target gene, and for Cas9 expression to be present in meiotically active cells where an allele can be 'driven'. We have identified target genes, assayed gene expression for relevant endogenous promoter activity, and are establishing pipelines for the generation of gene-edited and transgenic individuals. These initial steps towards developing a Hymeopterna gene drive are critical in supporting our capacity to test gene drives in invasive wasp and ant species that threaten Aotearoa's conservation estate.

Genome Assembly and Annotation of the Invasive Bryozoan Bugulina flabellata: A Hybrid Sequencing Approach
Ismael A Chowdhury

The bryozoan *Bugulina flabellata* is a globally invasive marine invertebrate and has historically been used as a model for developmental morphology, yet its genomic architecture has remained uncharacterized. Here, we present the first high-quality genome assembly and annotation for *B. flabellata* using a hybrid sequencing approach. After conducting phylogenetic and morphological investigations to confirm species identity, we used Oxford Nanopore Technology (ONT) to generate long-read gDNA data, alongside short-read Illumina data, which was used for polishing. This data was assembled with Flye and scaffolded with ragtag using ultra-long reads (ONT) to produce a 286.3 Mb assembly with a scaffold N50 of 21.5 Mb, high contiguity (L50=5), and exceptional completeness (BUSCO: 91.9%). We complemented the genome with a full-length transcriptome derived from direct RNA sequencing on the Nanopore GridION platform. This was fed into Braker3, alongside protein sequences from well-annotated sister species, to generate comprehensive gene predictions and annotations, identifying 20,826 gene loci. This work establishes a critical genomic resource for the genus Bugulina, enabling future studies on the evolution, development, and invasion biology of bryozoans. The integrated approach demonstrates the power of hybrid sequencing for assembling complex invertebrate genomes and linking genomic variation to morphological traits.

How to Rear your Parasitoid: Optimising *in vitro* rearing for the genetic model *Nasonia vitripennis*
M. Jacob Grupp, Hamish A. Salvesen, Kimberley R. Dainty, Peter K. Dearden

Invasive *Vespula* and *Polistes* wasps pose a significant threat to Aotearoa New Zealand's ecosystems. We use the Hymenopteran parasitoid *N. vitripennis*, a tractable model organism with short generation times, to undertake research into genetic control strategies against invasive wasp species. Ultimately,

we aim to develop transgenic tools to manipulate *Nasonia* genomes to better understand their unique evolutionary history and genetic features such as haplodiploidy. Female *Nasonia* oviposit embryos into blowfly pupa hosts, where they develop into adult wasps over 14 days. Previously, genetic manipulation of *Nasonia* relied upon the removal and return of embryos to their hosts. To support visual observation of developing wasps and embryonic screening for genetic modification, we are developing a pipeline for rearing *Nasonia* embryos through to adult eclosion. We have adapted an *in vitro* rearing technique using protein extract from pressed blowfly pupae to allow visualisation of injected larvae. Injected embryos were placed onto a mesh in 24 well plates and provided with protein extract for 8 days, followed by a 6 day dry period. To optimise embryo to adult survival we varied amounts of protein extract, addition of supplements, and medium sterilisation techniques. Successful *in vitro* rearing of edited *N. vitripennis* will transform future genetic and developmental work on this species. An *in vitro* rearing system will allow observations of development, the ability to tag endogenous gene expression, and an opening for techniques such as oral transfection to achieve transgenesis.

Whaia te mātauranga: Education Resources and Initiatives to Enhance Māori Participation in Modern Genetics and Genomics

Phillip Wilcox

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Over the last decade various education-based initiatives have been initiated that seek to enhance Māori participation in genetics and genomics research. From virtually nothing and with limited funding, these initiatives have targeted multiple demographics ranging from pre-NCEA level 1 taurira Māori, to university undergraduate and graduate students, to 'flax roots' pākeke living and working in Māori communities. In parallel, Māori-specific content has been introduced to genetics (and other) courses at both undergraduate and graduate level at the University of Otago. Beginning in 2016 with the Summer Internship of iNdigenous peoples in Genomics Aotearoa (SING-A, www.singaotearoa.nz), subsequent initiatives included the University of Otago's genetics module in its 2018 Science Wananga series that targets young high school level tauira Māori. More recently, Te Roopu Kōkiri (TRK) has been established, which is a collective of Māori biomedical researchers across the Maurice Wilkins Centre partner organisations, including Māori health organisations. There is also the Ira Hāpai collective – of SING-A alumni and faculty, consisting of over 100 tāngata Māori. In addition, the Ruatau project specifically targets genomics researchers who are Māori by descent but raised in te ao pakeha. At the more senior pakeke/kaumātua-level, a Māorispecific charitable trust, Ira Tātai Whakaeke, has been established which involves multiple senior health and genomics researchers, most of whom have represented their hapori in other matters, and two of whom were recently acknowledged among the 100 Māori leaders (https://100maorileaders.com/leaders/irene-kereama-royal and https://100maorileaders.com/leaders/dr-kimiora-henare). However, despite the embedding of these initiatives there are still many challenges. These include a lack of committed long-term financial resourcing for these e/orts despite demonstration of considerable benefits arising from them. Other challenges stem from ongoing structural bias, systemic racism, and false allyship. Nonetheless, benefits from these e/orts are beginning to flow back to communities, and have situated Aotearoa-New Zealand as a world leader in incorporation of indigenous knowledge and peoples in modern genetics education.

Pathogenic biallelic variants in VPS52, encoding an intracellular trafficking protein, cause impaired brain development

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Vacuolar protein sorting-associated protein 52 (VPS52) is a core subunit of the Golgi-associated retrograde protein (GARP) and the endosome-associated recycling protein (EARP) complexes, which mediate retrograde and recycling vesicle trafficking. Here, we characterise *VPS52* as a novel disease gene. Through exome sequencing of a New Zealand family and subsequent international collaborations, we have identified 15 cases from ten families with biallelic *VPS52* variants. While some cases are lethal in utero or early postnatally, for those surviving, clinical features include microcephaly, seizures, developmental delay, and non-neurological abnormalities such as cholestasis and arthrogryposis. We hypothesised that these variants are pathogenic, acting through a hypomorphic loss-of-function mechanism. Patient-derived fibroblasts and reporter cell models confirmed that variants reduce VPS52 transcript or protein levels, impairing GARP/EARP complex stability and lysosomal homeostasis. Furthermore, vps52 genome editing in a zebrafish model altered normal development, recapitulating developmental defects observed in patients, including seizures. Our findings demonstrate that biallelic, hypomorphic loss-of-function variants in *VPS52* disrupt intracellular trafficking, establishing it as a critical gene for human development. This study provides vital insight into the requirement for robust vesicle transport during brain morphogenesis and systemic proteostasis.

From genomes to microbiomes: predictors of infectious disease vulnerability in Māui and Hector's dolphins

Sebastian Alvarez-Costes, Ludovic Dutoit, William Rayment, William Pearman and Alana Alexander

Host–microbiome associations are complex ecosystems that have implications for host health, physiology, and adaptation to changing environments. Hector's and Māui dolphins are two recently diverged subspecies that inhabit distinct coastal habitats in Aotearoa, New Zealand, both heavily impacted by anthropogenic pressures, including exposure to novel pathogens. Here, we characterised the skin microbiomes of 30 individuals, including Māui dolphins from the North Island and Hector's dolphins from three South Island populations, and evaluated how microbiome composition varies with cause of death (biopsy, trauma-related, and confirmed infectious disease). We then integrated microbiome profiles with whole-genome resequencing data in a multi-omics framework to test how host genetic variables interact with microbiome features to shape infectious disease susceptibility. Māui dolphins showed lower alpha diversity and fewer shared taxa with Hector's dolphin populations, consistent with reduced microbiome richness in this critically endangered subspecies. Individuals that died from infectious disease showed the highest number of skin-associated taxa, whereas biopsied individuals exhibited the least diverse microbiomes. Multi-omics modelling revealed that the probability of pathogen-related death increased with higher inbreeding (high FROH), lower genome-wide heterozygosity (low GWH), reduced alpha diversity, and elevated phylogenetic diversity of the skin microbiome, with a higher risk in females. Together, these results demonstrate that interactions between host genomic health and skin microbiome structure shape infectious disease risk in small,

endangered cetacean populations, and highlight the potential of explainable multi-omics models to develop non-invasive biomarkers for conservation-focused health monitoring.

Posters

1. Decoding Regeneration: Uncovering the genetic regulation driving whole-body regeneration in *Botrylloides diegensis*

Devon A. Gamble, Berivan Temiz, Megan J. Wilson.

Wilson Laboratory, Department of Anatomy, University of Otago.

Whole-body regeneration (WBR) is the process by which a fully functional organism regenerates from a tissue fragment. The colonial ascidian *Botrylloides diegensis* exhibits this extraordinary ability, regenerating an entire organism from a small fragment of its shared circulatory system in just ten days. WBR involves widespread changes in gene expression; however, the regulatory mechanisms driving these changes remain poorly understood. Previous studies show that histone deacetylase inhibition disrupts WBR and leads to early colony death, highlighting the critical role of chromatin remodelling in this process. This study aims to map and characterise key gene regulatory elements and analyse their dynamic changes during early WBR. CUT&RUN profiling of the histone modifications H3K4me3, H3K27ac, and H3K4me2, reveals enrichment at transcriptional start sites and across gene bodies, and will enable the identification of active promoters and enhancers. Integration with ATAC-seq data across regenerative stages highlights changes in chromatin accessibility and will reveal changes in regulatory element activity. Complementary RNA-seq of histone deacetylase-inhibited and control tissues identifies differentially expressed genes potentially regulated by these epigenetic changes. By constructing a genome-wide landscape of epigenetic changes during early WBR, this study will elucidate the regulatory mechanisms that enable *B. diegensis* to undergo this extraordinary regenerative process.

2. Investigating the role of *SCNN1D* in breast cancer progression, metastasis and therapy response

Krittika Zutshi

Department of Physiology

Epithelial sodium channels (ENaCs) are non-voltage gated ion channels composed of α, β and γ subunits, encoded by *SCNN1A*, *SCNN1B*, and *SCNN1G* respectively. A fourth δ subunit, encoded by *SCNN1D*, can substitute for α to form a δβγ-ENaC complex with distinct gating and regulatory features. While ENaC has been implicated in key oncogenic processes such as proliferation, apoptosis, migration and invasion, the role of *SCNN1D* in breast cancer remains poorly understood. This study investigated *SCNN1D* expression level and its potential prognostic significance in breast cancer. Bioinformatic analysis of publicly available datasets revealed that *SCNN1D* mRNA levels progressively decline from normal to tumour and metastatic breast tissues. High *SCNN1D* expression was significantly associated with improved recurrence-free survival particularly in Luminal A breast cancer subtype, suggesting a tumour suppressive related function. To validate these findings, we assessed *SCNN1D* transcript levels in four breast cancer cell lines (MDA-MB-231, MDA-MB-231 overexpressing αENaC, MCF-7 and SK-BR-3) using RT-qPCR. Expression levels varied across cell lines, with MDA-MB-231 showing the highest *SCNN1D* transcript expression and SK-BR-3 the lowest. In contrast, western blot analysis revealed elevated *SCNN1D* protein levels in MCF-7 (Luminal A subtype). This discrepancy could reflect enhanced translational efficiency or increased protein stability in MCF-7 cells. These preliminary findings suggest that *SCNN1D* may play an important role in breast cancer. They provide the foundation for future research to better understand how this gene affects tumour growth, relapse, and response to treatment. Overall, these results provide a basis for further mechanistic studies to clarify its function and potential utility as a prognostic biomarker.

3. Investigating Genetic Variations in Myalgic Encephalomyelitis/Chronic Fatigue Syndrome as Potential Diagnostic Biomarkers

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Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) is a debilitating, lifelong condition without a molecular diagnostic test. Genetic biomarkers, enabling diagnosis of ME/ CFS soon after onset, would ensure a better quality of life for patients. Having tests that assess risk for developing ME/CFS within a family already affected means guidance can be provided to at risk individuals, particularly when exposed to potential triggers such as viral infections. Decode ME, a Genome Wide Association Study (GWAS) identified eight single nucleotide polymorphisms (SNPs) genetically linked to ME/CFS. A Precision Life combinatorial analysis identified 15 SNP clusters, each with a core SNP, that could identify >90% of the ME/CFS samples in the UK biobank. Candidate SNPs were selected and primers designed to PCR amplify the SNP sites in genomic DNA of four members of a ME/CFS-affected family. Amplicons were analysed, purified and sequenced to determine whether the SNPs were present. There was marked genetic heterogeneity for some core cluster SNPs, but none of the GWAS variants were identified. Future work in larger families, such as a Māori whānau, is needed to test if an identified risk variant identifies all those members affected, and could translate to a family risk signature for ME/CFS.

4. Investigating genetic drivers of high tumour mutational burden in breast cancer

Liam Young

Department of Biochemistry

Breast cancer incidence is on the rise globally, with an increase by 38% and mortality by 68% expected by 2050. Aotearoa has the highest annual breast cancer incidence rate of 3,660, this estimate would increase cases to over 5,000. To help manage the burden of breast cancer, interventions are required. Breast cancer varies in aggression with tumour mutational burden (TMB) associated with poorer outcomes. Across Pasifika and Māori, breast cancer patients with high TMB have been linked to a 29.5 kb germline deletion within the APOBEC locus. This variant is seen at a ~52% frequency among both populations fusing together APOBEC3A and APOBEC3B creating a chimeric mRNA with higher stability outcompeting wildtype. APOBEC canonically acts as a viral defence element acting as a cytidine deaminase with dysregulation seen to cause host mutagenesis. Studies have linked the variant to increased TMB, however, the fusion protein's implications have remained unexplored. This research project aims to generate this variation within the SSM3 mouse cell line via CRISPR to analyse the APOBEC mutational signature on DNA and expression to identify the role of fusion protein. Understanding of the function of this variant is critical for carriers, allowing pre-emptive action and informing future preventative strategies.

5. Studying the developmental effects of reducing Orc3 levels in the Zebrafish model

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The origin recognition complex (ORC) binds DNA to initiate DNA replication. Genetic studies indicate that ORC may have functions beyond DNA replication. We have identified three families with biallelic *ORC3* variants who had hypomyelinating leukodystrophy, a phenotype not previously associated with ORC, while monoallelic variants in seven families may cause a range of clinical features. Studies in *Drosophila* and mice have also shown that ORC3 is expressed in the non -

proliferating brain, and abnormalities occur when it is knocked down. We aim to create an ORC3 CRISPR knockout line in *Danio rerio* (zebrafish) as a model organism, investigate the consequences of reduced ORC3 expression, and test patients' variants for their effects in vivo. CRISPR design tools were used to design sgRNA targeting early exons of ORC3. The gRNA was synthesized successfully, and ORC3 knockout line generated by microinjecting the gRNA and Cas9 mRNA into embryos, followed by live imaging and genotyping. RT-qPCR was used to measure *ORC3* mRNA levels to select the most effective gRNA, with a future breeding of edited embryos to examine its impact on development. Understanding the ORC3 brain-specific role in vertebrates might offer molecular insight into ORC3-associated neurodevelopmental disorders and the disease pathophysiology caused by the patients' variants.

6. DeCOde: Understanding CONSTANS (CO) variation for flowering control in perennial ryegrass

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Perennial ryegrass (*Lolium perenne* L.) is New Zealand's primary forage and the foundation of its pasture systems. Each spring, ryegrass undergoes a seasonal shift to flowering that reduces pasture quality and livestock performance. To mitigate this, we aim to develop ryegrasses that remain vegetative under field conditions by increasing their daylength requirements for flowering. Previous research identified a variant of the flowering gene *CONSTANS* (*CO*) as a target for increasing ryegrass daylength requirements. CO is a transcription factor that promotes expression of the key floral inducer *VRN3* during the day; at night, it is degraded by the COP1/SPA complex. The balance of these two activities determines a plant's daylength requirements for flowering. Targeted sequencing of *CO* uncovered a SNP encoding a serine-to-arginine substitution in a putative transactivation domain. We hypothesise this change may reduce the transactivation ability of CO, and aim to test this using yeast- and tobacco-based assays. Interestingly, the SNP is also located close to a degradation motif recognised by COP1/SPA, suggesting it may affect CO stability. We aim to test this hypothesis in a tobacco-based expression system. Insights gained from this study will support the development of non-flowering ryegrass to improve spring pasture quality and livestock performance.

7. Investigating the genetics underlying late flowering in perennial ryegrass

Lachlan Taylor[1], Lynette Brownfield[1]

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Perennial ryegrass (*Lolium perenne*) is a critical forage in New Zealand farming systems, utilised due to its high metabolisable energy (ME) content in vegetative tillers. ME availability decreases during the transition from vegetative to reproductive tiller growth, impacting agricultural productivity. Flowering (heading) in perennial ryegrass is induced by long photoperiods after prolonged exposure to cold temperatures and naturally occurs in spring. Genetic variation in the flowering control pathways has been linked to variation in flowering times in other temperate grasses, however this has not been well characterised in ryegrass. This project focuses on a ryegrass population which has been consistently observed to segregate for floral emergence times, from late October through to mid-December. Using extended daylength experiments we have shown that the photoperiod response plays a role in variable flowering times and genetic analyses have identified variation in Chromosome 4 that is associated with late flowering in this population. We are now exploring variants in putative photoperiod genes in this region of Chromosome 4 to investigate the function of these genes in ryegrass and identify alleles that could be used for breeding late-flowering ryegrass.

8. Designing a functional genomics pipeline for immune GWAS variants: early-stage insights

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Genome-Wide Association Studies (GWAS) have identified thousands of genetic variants associated with immune-mediated diseases however, functional follow-up of these signals remains a major challenge. This bottleneck is particularly pronounced for non-coding variants, whose eXects are often only observed under specific cellular and inflammatory contexts. Early-stage validation of an immunologically relevant cellular system has laid the groundwork for a post-GWAS framework to interpret gout risk variants identified in a Polynesian gout GWAS. Key design choices include the selection and validation of a genetically tractable immune cell model (BLaER1 cells), benchmarking inflammatory responses against primary cells and other commercially available models, and assessing suitability for genetic manipulation and reporter-based assays. In parallel, strategies for prioritising both missense and non-coding variants associated with gout risk have identified variants within *PINK1* and *TRIM7*, as well as regulatory regions near *ELF1*, *IRX1*, and *OPRL1*. Functional investigation of these variants will be conducted using CRISPR base editing and luciferase reporter assays in BlaER1 cells, with immune stimulation used to capture context-dependent regulatory eXects. Overall, this work provides a transparent account of how a functional genomics pipeline is constructed, oXering practical insights for navigating the transition from GWAS to experimental immune biology, while guiding this framework within Māori and Polynesian research priorities.

9. The Genomic Repertoire of the Order Hymenoptera in Relation to Eusociality

Phoebe Keddell

Department of Biochemistry

The Hymenoptera, bees, wasps, ants and sawflies, are one of the most speciose and diverse animal orders within nature. The ecological and economic effects that its >150,000 named species cause can be felt internationally, with a significant amount of its impact attributable to their propensity to form co-operative, complex societies. Hymenopteran sociality can range from that of small parasitic wasps that never interact with their offspring, to the specialized eusocial societies of honeybees and ants. These eusocial societies have independently evolved 12-14 times within the Hymenoptera, disproportionate to the rarity of sociality in other taxa. There are over 500 publicly available hymenopteran genomes that may be mined to understand the evolution of eusocial traits. Using consistent methods for each, I reannotated and quality-filtered these genomes for the purpose of accurate orthogroup identification, in which a gene family is collated across all species to summarise the phylogenetic relationship between all orthologues and paralogues across the order. Included species were sorted into consistent categories of sociality, which, when combined with orthogroups, allows the identification of convergent, sociality-associated patterns of orthogroup retraction and expansion. These sociality-associated patterns of orthogroup evolution provides a universal resource to understand the evolution and mechanisms of hymenopteran eusociality.

10. Tracking marine metazoan diversity changes to extreme climate events in Bay of Plenty kelp forest and rocky reef ecosystems: an eDNA approach

Nicolas Restrepo

Department of Anatomy

Kelp forests and rocky reef ecosystems are vital coastal habitats that are facing increasing stress from extreme climate events, such as marine heatwaves and marine darkwaves in the Bay of Plenty. This study employs environmental DNA (eDNA) metabarcoding, targeting both COI and 16S gene regions, to track biodiversity responses and community shifts in these ecosystems. Seven sites representative of kelp bed habitats in the Bay of Plenty, spanning gradients of sedimentation and thermal exposure, are being sampled. Samples have been collected in 2023, 2024, and 2025, supporting multi-year monitoring of biodiversity shifts and community dynamics in response to environmental changes. The results will (1) quantify biodiversity changes linked to extreme events in kelp forest and rocky reef habitats; (2) reveal correlations between biodiversity shifts and environmental stressors using multivariate models; and (3) forecast future species distribution changes under climate scenarios. This research will provide a robust framework for understanding climate impacts on coastal marine biodiversity, validate eDNA for long-term ecosystem monitoring using COI and 16S markers, and guide conservation strategies to protect native biodiversity and Māori taonga species.

11. Probing the function of neural *jazf1* with whole-brain imaging and RNA-seq in the larval zebrafish

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JAZF1 is a transcriptional regulator known for its role in metabolic regulation through the liver and pancreas, but its role in the brain is poorly understood. Genome-wide association studies revealed a strong link to type 2 diabetes – a significant risk factor for Alzheimer's disease. JAZF1 is expressed in microglia and excitatory neurons in association cortices in AD. We created a *jazf1* knock-out zebrafish to probe its role in the brain through whole-brain functional and structural imaging and RNA-seq. Wildtype and *jazf1* KO larval zebrafish at 6 days post fertilisation were immunostained and imaged using ERK (tERK) as a structural readout, and phosphorylated ERK (pERK) as a functional readout. The image stacks were morphed, registered, harmonised and then submitted to pair-wise non-parametric statistical analyses. Size changes in brain regions were quantified by using the Jacobian determinant used for morphing, and a pERK/tERK ratio was used as a surrogate measure for brain activity. We found *jazf1* KO resulted in smaller hindbrains and a more active hypothalamus. Our RNA-seq data on adult fish brains suggest an upregulation of *apoe* – the strongest contributor to sporadic AD. Gene ontology analyses indicated a strong activation of lipid metabolism, immune and cytokine regulation processes in *jazf1*-/- larvae. We have generated *jazf1* KO zebrafish to characterise its function in the brain. Our brain mapping study corroborate with existing data to indicate *jazf1* may have a direct role in regulating insulin actions via the hypothalamus. RNA-seq data provide some support for *jazf1*'s potential multi-faceted role in AD. We are currently using neuroimmune and metabolic challenges as system-wide perturbations to further uncover the functional involvement in *jazf1* in T2D and AD-like states.

12. Understanding the genotype-phenotype relationship of growth disorders through functional isoforms of DONSON.

Pragya Bradu, Sankalita Ray Das, Matthias Fellner, Elizabeth C. Ledgerwood and Louise S. Bicknell

DONSON (Downstream neighbour of *SON*) is involved in the initiation and progression of DNA replication. Patient variants identified in *DONSON* are responsible for causing three microcephalic primordial dwarfism disorders – Microcephaly-micromelia syndrome (MIMIS), Microcephaly, short stature and limb abnormality (MISSLA) and Meier-Gorlin syndrome (MGORS), where patients with different variants differ in height and brain growth. My project aims to understand the genotype-phenotype relationship between *DONSON* MGORS and *DONSON* MISSLA by exploring possible protein isoforms of DONSON. Using a unique pipeline, nanopore datasets of different cell lines from the NCBI-Gene Expression Omnibus (GEO) database was used to study for exon skipping events, producing evidence of potential different in-frame isoform reads of *DONSON* that could exist in the cells. Next, two most common possible isoforms of DONSON (exon 5/6del and exon 2del) were analysed for structural properties, predicted using AlphaFold and ChimeraX software. *In-silico* mass spectrometry analysis suggests that these DONSON isoform proteins could exist. RT-PCR analysis further confirmed the presence of *DONSON* isoforms. These bioinformatic analysis and preliminary experimental data suggests that possible DONSON isoforms could exist and therefore play a role in the genotype-phenotype relationships between *DONSON* MGORS and *DONSON* MISSLA.

13. Title TBC

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14. Comparative Epigenomic Insights from Peripheral Blood and Cell-Free DNA for Diagnostic Biomarker Discovery in Myalgic Encephalomyelitis/Chronic Fatigue Syndrome

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Post-viral syndromes such as Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) and Long COVID (LC) share over 95% of reported symptoms, including postexertional malaise, yet their shared and disease-specific molecular mechanisms remain poorly defined. Previous epigenetic studies, including our own, have primarily focused on peripheral blood mononuclear cells (PBMCs) using array-based and sequencing-based platforms. Our recent analysis interrogating ~4 million CpG sites identified distinct yet overlapping methylation landscapes, with 118 differentially methylated fragments (DMFs) shared across both conditions. Building on these findings, we propose a pilot study to assess whether disease-relevant epigenetic signatures can be detected in plasma-derived cell-free DNA (cfDNA). Unlike PBMC DNA, cfDNA provides a minimally invasive, systemic snapshot of DNA released from multiple tissues, offering potential insight into the multi-organ involvement of post-viral syndromes. Although cfDNA methylation profiling has proven diagnostic value in oncology and prenatal medicine, it remains unexplored in ME/CFS and LC. Using a modified RRBS workflow, we are generating paired cfDNA and PBMC methylomes from 5 ME/CFS patients and 5 matched healthy controls. This study will evaluate PBMC–cfDNA concordance, identify systemic signals beyond immune cells, assess cfDNA's biomarker feasibility, and apply tissue-of-origin deconvolution, laying the groundwork for cfDNA-based epigenetic biomarkers in post-viral disease.

15. Molecular Identification and Phylogenetic Affinities of Parasitic Nematodes in Freshwater Fish from Northern Colombia

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Fish-parasitic nematodes represent an important yet underexplored component of tropical freshwater biodiversity, with implications for fish health, ecosystem dynamics, and potential zoonotic transmission. In the La Mojana wetland region (San Jorge River basin, Sucre, Colombia), parasite diversity remains poorly characterized. This study aimed to identify nematodes infecting freshwater fish using an integrative morphological and molecular approach. Ten freshwater fish species were collected with the assistance of local artisanal fishermen. Fish were dissected and all recovered nematodes were preserved inethanol for morphological and molecular analyses. Larval anisakids were preliminarily classified as *Contracaecum* sp. based on morphological features such as ventriculus structure and the presence of a posterior mucron. Camallanid nematodes were tentatively identified as *Spirocamallus* sp. based on buccal capsule morphology and body characteristics. Genomic DNA was extracted from individual specimens. The ITS1–ITS2 region of nuclear rDNA (*Contracaecum*) and partial 18S and 28S (LSU) rRNA genes (other taxa) were amplified by PCR, sequenced, and compared with GenBank references using BLAST. Phylogenetic relationships were inferred using Bayesian Inference in MrBayes implemented on the CIPRES Science Gateway. Molecular analysis confirmed the presence of *Contracaecum* sp. and *Spirocamallus* sp. in several host species. Genetic variation among *Contracaecum* isolates suggests unsuspected lineage diversity and non-random patterns of host use within the freshwater fish community, while the detection of adult *Spirocamallus* highlights the coexistence of parasites with distinct life-cycle strategies. This study provides the first integrative molecular characterization of nematodes in La Mojana and underscores the value of combining morphological and molecular approaches to uncover parasite diversity in understudied tropical freshwater ecosystems.

16. Swipe left on self-pollen: Uncovering the molecular basis of self-incompatibility in clover

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Clover is the most important pastoral legume to agriculture in New Zealand. Many clover species, including White clover (*Trifolium repens*), are self-incompatible, meaning for fertilisation to occur pollen must come from a genetically distinct plant. Self-incompatibility has slowed genetic gain and limited breeding in white clover as it prevents inbreeding which is needed to purge deleterious alleles and fix beneficial alleles. Self-incompatibly has evolved in a range of plants resulting in distinct mechanisms across families. Self-incompatibility often originates from a single locus, the S-locus, that contains one gene expressed in the male tissue (pollen/anther) and another in the female tissue (stigma/pistil). These genes often encode a complementary set of interacting proteins, a receptor and ligand pair, that underpin the recognition of self or non-self pollen. We have identified the putative S-locus genes in clover and predict that they encode a receptor and ligand. In this research, we aim to validate and characterise the predicted proteins, including their expression patterns, protein-protein interactions and cellular locations. This will uncover the molecular mechanism responsible for self-incompatibility in clover, and ultimately advance clover breeding.