# MetaboNews

## This month in metabolomics

August, 2026
Vol 16, Issue 7

MetaboNews is a monthly newsletter published in a partnership between The Metabolomics Innovation Centre (TMIC) and The Metabolomics Society

## In This Issue



**This Months Features:**
MetaboInterview
Simona Indzhova
(McMaster University)

*|MC:SUBJECT|*

# Metabolomics Society News

The Metabolomics Society is an independent, non-profit organization dedicated to promoting the growth, use, and understanding of metabolomics in the life sciences.

General Enquiries

info@metabolomicssociety.org

## Members Corner

**Board of Directors**

**Message from Warwick (Rick) Dunn, President**

Dear Metabolomics Society Members and metabolomics friends,

Although it is the time of year when summer or winter holidays are underway, the society operations still continue (though at a slower pace).

Firstly, our annual election for Directors is in operation. The Society is led through the voluntary efforts of the Board of Directors. We received 22 nominations and thank you all for nominating or being happy to be nominated. The election process is currently underway, all members can vote for up to seven of the nominees and you have until August 20th to place your votes.

Leslie from Snap, Roy and I will then count the votes and inform the successful nominees and hopefully we can announce these in the September issue of MetaboNews. The deadline for applications to sit on the Early-career Members Network has also just closed and a new Chair is currently being voted for to replace Breanna Dixon.

Secondly, planning for the Metabolomics 2027 conference in South Korea has already started and I know many are looking forward to the conference and opportunity to visit South Korea. It will be the first time the conference has been located in South Korea.

Thirdly, the officers and Aurelia have edited the conference application documents and these will be available in the next few weeks for the community to apply to host the Metabolomics 2028 conference in either Africa or Europe. We have already had enquires from both Europe and Africa about how to apply and we look forward to receiving and reviewing the applications. The conference location will officially be announced at the Metabolomics 2027 conference (though always seems to leak to the community before).

I am in the last two months of my role as President and Director and a number of the board will step down at the end of September including the Secretary (Maria) as well as Lynn, Tomas and Breanna. More to follow on this in the next MetaboNews.

All the very best,
Warwick (Rick) Dunn, University of Liverpool, UK
President, Metabolomics Society

## Early-career Members Network (EMN)

### EMN Webinars

**July EMN Webinar — Thank you!**
The EMN Committee extends its gratitude to **Dr Justin Carrard**, Senior Physician in Sport and Exercise Medicine at Lausanne University Hospital (CHUV) and Head of the Sport and Youth Health Unit at the Swiss Olympic Medical Centre for the July webinar entitled 'Metabolomics and Lipidomics in Sport and Exercise Medicine: a physician-scientist perspective'. The webinar recording is available on the MetSoc website: https://metabolomicssociety.org/resources/multimedia/emn-webinars-2026/ .

**August EMN Webinar - REGISTER NOW!**
The EMN Committee is pleased to welcome **Prof. Osbaldo Resendis-Antonio**, Full Professor at the Universidad Nacional Autónoma de México (UNAM) and the National Institute of Genomic Medicine (INMEGEN), Mexico, and **Samaria Johana Gutiérrez Félix,** a Ph.D. student in Biochemical

Sciences at UNAM. Together, they will present the August webinar entitled 'Digital microbiota: from associations to mechanisms in the era of AI and Genome-scale metabolic modeling'. The webinar will take place on Friday, 28 August at 9:00 CST/15:00 UTC/17:00 CEST.

**Register here**:
https://zoom.us/webinar/register/WN_netBjVL2Rpyy8OVTh4Zvgg#/registration

---

## International Affiliates Corner

**Réseau Français de Métabolomique et Fluxomique (RFMF)**
Visit http://www.rfmf.fr/


**RFMF second thematic school on metabolomics in human health, deadline in September**
There are still places available for the 2nd RFMF Thematic School on metabolomics in human health! Registration remains open until September 2, 2026, but places are limited, so don't miss the opportunity to join us.

This thematic school, jointly organized by the RFMF and the French Society of Clinical Biology, will be supported by the CNRS.

The school will focus on metabolomics in human health.

Dates: October 5–9, 2026
Schedule: From Monday, October 5, 2026, at 12:00 p.m. to Friday, October 9, 2026, at 12:00 p.m.
Venue:
Centre de Vacances Paul Langevin
24 rue du Coin
73500 Aussois, France

All information is available on the website: RFMF Thematic School #2

Registration fees:

- Academic participants: €1,300
- Industry participants: €2,000
- FREE for CNRS staff, subject to availability

Places are limited. Registration deadline: September 2, 2026

.

To register, please visit: Registration – RFMF Thematic School #2

We look forward to welcoming many of you for a full week of discussion around the numerous challenges facing metabolomics in health, identifying key barriers that need to be addressed, and helping to build and strengthen a community around this important field.

**MetaboHUB training in metabolomics**

**TRAINING "Hands-On Metabolomics: From Bench to Data" – Places Still Available!**

**There are still places available for the upcoming MetaboHUB training!** If you are interested in learning how to integrate metabolomics into your research or analytical projects, this is a great opportunity to gain both theoretical knowledge and practical, hands-on experience.

**Why this training?**

Want to integrate metabolomics into your research or analytical projects, but don’t know where to start? Whether your focus is on **agrosciences, nutrition, human health, environmental science, biotechnology, or animal health**, this training designed by **MetaboHUB** is for you!

**Information**

**When?** November 23–27, 2026
**Where?** Toulouse, France – INSA Campus
**Who is it for?** Researchers, engineers, technicians, doctoral and postdoctoral students, academics and industry professionals, biologists and analysts

**What will you learn?**

Expert scientists from the MetaboHUB infrastructure will introduce you to the fundamental principles and cutting-edge techniques of metabolomics through a dynamic combination of **theoretical and hands-on sessions**.

**Interactive format:**

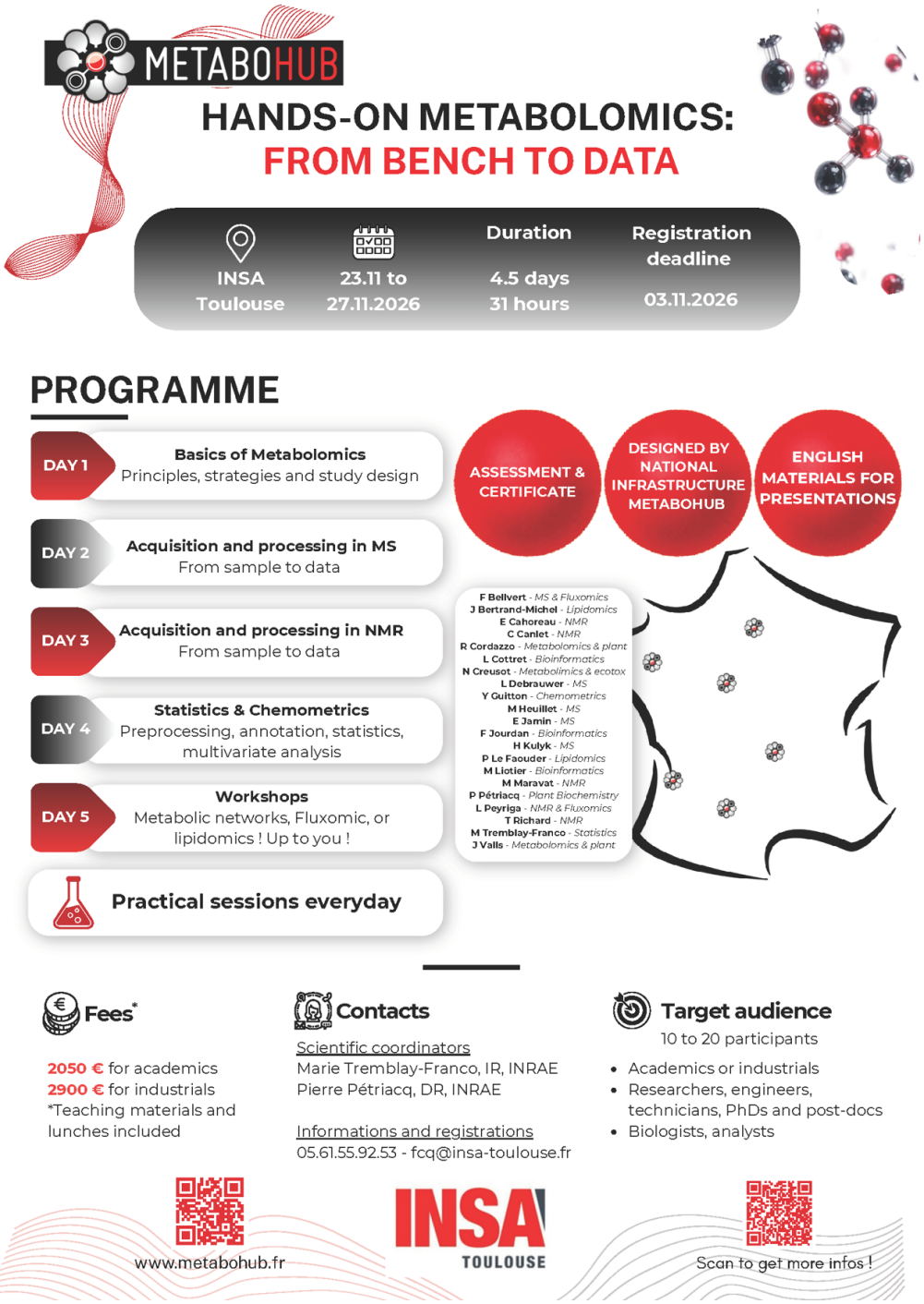
- Practical workshops
- Discussions and case studies
- Keynotes and workshops
- **Training certificate provided**

**Registration & contact:** fcq@insa-toulouse.fr

**Limited places are still available – register soon to secure your spot!**

ℹ All the information is available in the picture below.

*|MC:SUBJECT|*




Back to top

*|MC:SUBJECT|*


## MetaboTalks

*|MC:SUBJECT|*


## Introducing MetaboTalks: TMIC's New Speaker Series

Introducing MetaboTalks — a new online speaker series connecting researchers, trainees, and collaborators across the metabolomics and multi-omics community. Each session features an in-depth conversation with two leading voices in the field, covering their work, their methods, and where the discipline is headed next. New sessions will run monthly, and the series is free and open to everyone.

Join us on **September 16, 2026, 10:00–11:00AM MDT** for the 1st episode, which brings together two talks that, together, span the field's next decade and its newest frontier:

**Dr. David Wishart** (University of Alberta), a founding figure in metabolomics and Tier

1 Canada Research Chair in Metabolomics and Precision Medicine, opens with *What's Next for Metabolomics?* — a three-horizon vision for the field, from solving its long-standing "dark matter" identification problem within two years, to push-button automated workflows within five, to metabolomics becoming so deeply embedded in routine multi-omics science that it disappears as a standalone discipline within a decade.

**Dr. Adriana Zardini Buzatto** (University of Calgary), Tier II Canada Research Chair in Lipidomics, follows with *Tracing Environmental Exposures Through Lipidomics* — showing how ion mobility-enabled lipidomics can detect the biological effects of exposures like cadmium and PFAS in human liver cells, often before conventional toxicology testing would catch them.

September 16, 2026 · 10:00–11:00AM MDT · Online

Register Here for Free: https://ualberta-ca.zoom.us/webinar/register/WN_uj0AdsR-TDKvPW0zUi9ifw#/registration

For Additional Details Visit: https://metabolomicscentre.ca/science/metabotalks/

## MetaboInterview

### Simona Indzhova


Simona Indzhova earned her Honours Bachelor of Science in Life Sciences, with a minor in French, from McMaster University. She began her Master's in the Biochemistry & Biomedical Sciences program at McMaster University under the supervision of Dr Marie Pigeyre at the Population Health Research Institute, after which she transferred to her PhD. Her project to date has focused on the use of retinal images as a non-invasive approach for predicting cardiovascular disease and all-cause mortality. Combined with metabolomic profiling, this work has provided insights into the biological pathways linking retinal microvasculature with CVD and mortality. She is currently working on integrating multi-omics approaches to improve risk prediction. Once this phase of the project is complete, she plans to extend these methods to brain health and cognition. Simona hopes her research will help identify individuals at risk of cardiometabolic disease, cognitive decline, and mortality, while also investigating the biological pathways underlying retinal vascular alterations and their links to these outcomes.

**What inspired you to pursue this area of research within Metabolomics?**

The first part of my project focused on identifying plasma metabolites that link both retinal microvascular complexity with cardiovascular risk. Through this work, I found that nicotine-related metabolites were the strongest biomarkers of this relationship. However, I came to realize that one of the challenges in metabolomics is that the number of metabolomic features often exceeds the number of study participants, and that this might reduce the informativeness of examining individual metabolites in isolation. This motivated a shift toward the development of a metabolomic risk score, which summarizes an individual's metabolomic profile into one simple score. After all, diseases do not arise from any one metabolite acting alone, but rather from complex interactions among multiple metabolites. Metabolomic risk scores are uniquely positioned to capture these collective effects, yet despite their potential, they remain a relatively underexplored concept in the study of disease progression, which inspired me to pursue this area of research. I wanted to first develop a metabolomic risk score for all-cause mortality to capture the global health of an individual, after which I will zoom in on developing metabolomic risk scores for more specific diseases.

**Has working in Metabolomics changed the way you think about biology, health, or disease?**

Absolutely. Metabolomics has highlighted how dynamic human biology is and while traditional risk factors often provide a snapshot of health status, metabolomics offers a continuous and up-to-date picture of the state of a biological system. I believe that metabolomics has the best potential for applications in biomarker discovery as studies have demonstrated that metabolomics plays a role in areas of biology, ecology, aging, cancer and even neurodegenerative disorders. Working in Metabolomics has reinforced the idea that diseases develop gradually through interconnected metabolic pathways long before clinical symptoms appear.

**Was there a specific finding or result from your research that surprised you the most?**

One of the more striking observations to emerge from this work has been the extent to which aggregating multiple metabolites into a composite risk score substantially improves predictive performance relative to examining individual metabolites in isolation. Many metabolites showed only modest associations with all-cause mortality when considered independently, yet when integrated into a metabolomic risk score, the collective signal became stronger. This was not entirely unexpected in theory, but seeing it reflected in the data was a meaningful moment in the project. On the same note, I was surprised to see the difference in the strength of the association between the metabolomic risk score and traditional risk factors with all-cause mortality. This reinforced to me the potential of metabolomic profiling and the need to treat the metabolome as an integrated system rather than focusing on single biomarker approaches.

---

**What is one discovery or advancement in Metabolomics that you think deserves more attention?**

I believe the potential of large-scale, population-based metabolomics studies embedded within longitudinal cohorts remains underappreciated. Resources such as the Canadian Longitudinal Study on Aging combine clinical phenotyping, long-term follow-up data, and metabolomics measurements in large, well-characterized populations. These datasets allow researchers to examine how metabolic profiles evolve over time in relation to aging and health outcomes in ways that cross-sectional studies simply cannot support. As analytical methods continue to improve and the cost of metabolomic profiling continues to decline, population-based longitudinal metabolomics will only become more powerful, and I hope that greater awareness of what these resources can offer will attract more researchers.

---

**How do you see your work contributing to collaboration across disciplines or research communities?**

Research on metabolomic risk scores is multidisciplinary, spanning epidemiology, biochemistry, biostatistics, and clinical medicine. I see this work as naturally positioned to serve as a bridge across these groups. For epidemiologists, a validated metabolomic risk score offers a novel tool for population-level risk stratification. For biochemists, the metabolites that contribute most strongly to the score point toward specific biological pathways worth investigating further. For clinicians, there is an important longer-term

translational question about whether metabolic profiling could be integrated into routine patient assessment to enhance preventive care. My hope is that presenting this work at conferences like CanMetCon will open dialogue with researchers from adjacent fields whose expertise could extend and strengthen the findings in directions I have not yet considered!

---

**What is one thing you wish more people understood about researchers or scientific work?**

One of the most persistent misconceptions about science is that it proceeds in a clean, linear fashion. People think that researchers formulate a hypothesis, conduct their experiments, and arrive at a clear answer in an orderly sequence. In practice, research is far more iterative and uncertain. It involves revisiting assumptions, troubleshooting unexpected results, refining analytical approaches, and sometimes abandoning directions that initially seemed promising. The polished version presented in a published paper rarely reflects the complexity of the process that produced it. I think a broader public understanding of this reality would foster more realistic expectations around the pace of scientific progress and a deeper appreciation for the persistence and rigour that research demands.

---

**How do you think your research could translate into real-world applications?**

In the longer term, a well-validated metabolomic risk score for all-cause mortality has meaningful potential as a complementary clinical tool, one capable of providing information about biological aging and systemic health risk that conventional markers may not fully capture. This could be particularly valuable in identifying individuals who appear low-risk by traditional risk factors but whose metabolic profile suggests an elevated underlying vulnerability, enabling earlier and more targeted preventive intervention. While translating research findings into clinical practice requires many additional steps, including replication across diverse populations and assessment of clinical utility, I believe this work contributes meaningfully to that long term goal and helps lay an important scientific foundation for future applications in precision medicine and preventive health care.

---

**What impact do you hope your research presented at CanMetCon 2026 will have in the next few years?**

My primary hope is that this work encourages broader scientific interest in the development and validation of metabolomic risk scores within large, population-based cohorts. If it prompts other research groups to examine whether similar approaches replicate across different populations, analytical platforms, or outcome definitions, that would represent a meaningful contribution to advancing the field. I also hope the work draws attention to the Canadian Longitudinal Study on Aging as a valuable resource for aging-related metabolomics research and invites potential collaborators who might extend these findings in complementary directions. More broadly, I hope presenting at CanMetCon 2026 positions this research within the wider Canadian metabolomics community and contributes to a growing conversation about how metabolomic risk scores can meaningfully be applied to questions of population health and aging.

---

**What trends or emerging areas in metabolomics are you most excited to follow?**

I am particularly excited by the growing integration of metabolomics with other omics layers within multi-omics analytical frameworks, which is something I am actively exploring in my own work. The ability to integrate findings across multiple biological levels holds tremendous potential for strengthening causal inference and highlighting the specific mechanisms through which metabolic dysregulation contributes to disease. I am following advances in untargeted metabolomics and improvements in metabolite annotation, which remain among the most significant bottlenecks limiting the field. I also find the application of machine learning very promising and I look forward to seeing how that area can advance in the coming years.

---

**If you could expand your project without any limitations, what would you explore next?**

If I could expand my project without any limitations, I would prioritize replicating and validating my metabolomic risk score across multiple independent cohorts spanning different ancestries, geographic regions, and age groups. Generalizability is one of the most important and often underexamined

questions with these scores, and being able to derive the same associations in the UK Biobank or the Prospective Urban Rural Epidemiology cohort would help strengthen my findings. Beyond that, I would be very interested in examining how the metabolomic risk score changes longitudinally through repeated metabolite measurements over the course of 10 years. It would be good to explore whether shifts in an individual's score over time are predictive of outcomes, or whether the score is modifiable through lifestyle or clinical intervention. This would move the work away from a purely predictive tool towards something with genuine actionable potential in a preventive medicine context. Finally, an area I would love to pursue further is the integration of metabolomics with other omics layers. This is actually something I am currently exploring, specifically in the context of genomics and epigenomics. However, if there weren't any limitations, I would extend this further to include proteomics. Ultimately, I believe that no single omics layer tells the complete biological story, and that the most powerful insights will come from studies that are able to integrate multiple layers simultaneously.

---

**Outside of research, what helps keep you motivated, creative, or balanced during demanding projects?**

Outside of research, what helps keep me motivated and balanced during demanding projects is taking time to rest both mentally and physically, as research is a marathon and not a race. Stepping away from my work, through fitness classes, spending time with friends and family, and travelling, consistently helps me return to problems with greater clarity. I also try to set clear daily goals so that I can advance my project by completing smaller tasks every day, celebrating progress by appreciating small achievements too, not just waiting for big wins, and reminding myself how my work adds value to the bigger picture. In addition, I create a routine and stick to it since that helps me reduce decision fatigue and builds momentum. Every few weeks, I check in with my progress and make any adjustments that I need to make in order to keep moving towards my goal.

---

**Do you have any other comments that you wish to share about metabolomics?**

I would simply say that metabolomics is at an exciting inflection point. The combination of larger datasets, improved analytical platforms and

computational methods means that the questions we can now ask were largely out of reach even a decade ago. For those considering entering the field or just starting out, I would encourage them to embrace its interdisciplinary nature. It is a field that rewards curiosity and I feel fortunate to be working in it right now!

---



Back to Top

---

## MetaboReads

### Measurement, Missingness, and the Limits of Annotation

Two papers this month take aim at a problem most laboratories handle by convention rather than by evidence: what happens to a feature that fails to appear in the data. A missing value in an untargeted run is usually imputed, dropped, or ignored, and a spurious feature is usually filtered on criteria inherited from someone else's workflow. Treating either as a purely technical nuisance assumes that absence and presence carry no biological information, an assumption almost nobody has tested at scale. The work below tests it from two directions, one genetic and population-scale, the other analytical and bench-scale, and both arrive at the same uncomfortable conclusion: the detection step is not neutral. As untargeted platforms move further into etiological and predictive research, decisions buried in data processing

propagate directly into biological claims.

---

**Genome-wide association studies of missing metabolite measures from two population-based studies**

**Faquih and colleagues** in *Genome Biology* **reported** that missingness in untargeted metabolomics is partly heritable. Working across the Netherlands Epidemiology of Obesity Study and the Rhineland Study, they ran a logistic GWAS on the probability of a metabolite being absent for 224 features missing in 10 to 90 percent of participants. Meta-analysis returned 55 metabolome-wide significant associations involving 28 metabolites and 41 lead SNPs, 42 of the associations novel. The implicated biology is not random: beta-oxidation, bile acid handling, steroid metabolism, and xenobiotic processing account for much of the signal, which is what one would expect if genotype places some individuals' concentrations below the limit of detection. Imputing those values discards a real signal and replaces it with a fabricated one.

**A systematically optimized two-dose differential strategy integrating stable isotope tracing and mass shift defect filtering for reducing false positives in high-resolution mass spectrometry-based drug metabolite profiling**

**Lin and colleagues** in *Talanta* **described** that a data processing workflow that couples a two-dose differential design with stable isotope tracing and mass shift defect filtering to suppress false positives in drug metabolite profiling. Using D0 and D3 labeled sildenafil, they recovered 56 putative metabolite features confirmed by fragmentation behavior and characteristic isotopic shifts, sidestepping the usual dependence on authentic standards. The comparison of incubation formats is the practically useful part: separate incubation of labeled and unlabeled compound outperformed co-incubation in a single tube, which markedly suppressed feature detection. Systematic tuning placed the optimum at an MSDF window under 0.12 Da, a retention time tolerance of 0.2 min, and three paired samples. For anyone attempting xenobiotic metabolite identification without reference standards, these are settings worth borrowing rather than rediscovering.

---

## Metabolic Signatures of Disease, Aging, and Environmental Exposure

The appeal of a metabolite signature is that it reports on physiology rather than genotype, but the distance between a reproducible signature and a usable clinical test remains the field's most persistent embarrassment. The papers gathered here occupy different points along that distance, from subcellular compartments through circulating profiles in defined patient groups to prospective cohorts assembled specifically to test whether a signature precedes the disease it marks. What emerges

is a consistent asymmetry: finding a discriminating profile is now routine, while demonstrating that it carries information the clinic does not already have is not. Two of these studies also make the case that where you sample matters as much as what you measure, since organelle-level and tissue-level extractions can average away signals that prove large and linear once isolated. The environmental exposure work adds a further complication by showing that dose can change mechanism rather than simply scale it. Signature discovery in this area is maturing into signature qualification, a slower and less quotable business.

---

**Atlas of lysosomal aging reveals a metabolite signature shared with lysosomal storage disorders**

**Puszynska and colleagues** in *Science* **reported** that lysosomes accumulate a defined metabolite signature with age, and that the signature is one already familiar from pediatric disease. Applying rapid lysosomal isolation across brain, heart, muscle, and white adipose tissue, they built a multitissue atlas of the aging lysosomal metabolome and found glycerophosphodiesters and cystine rising linearly with age, well before organismal decline became apparent. Those same metabolites are causally implicated in Batten disease and cystinosis, which places juvenile lysosomal storage disorders and normal aging on a shared biochemical axis rather than in separate literatures. Caloric restriction blunted the accumulation in heart and muscle but left the brain unaffected, a tissue specificity that will need explaining. The organelle-resolved approach is the enabling piece, and it raises the obvious question of what other compartment-specific accumulations whole-tissue metabolomics has been diluting into invisibility.

**Integrative serum proteomic and metabolomic profiling in ovarian endometrioma: an exploratory multi-omics study**

**Chen and colleagues** in *Frontiers in Medicine* **found** that ovarian endometrioma leaves a coordinated immune and metabolic imprint in the circulation. Their exploratory analysis paired untargeted metabolomics with proteomics in affected women and healthy controls, with proteomic changes concentrated in immune and extracellular processes while metabolite shifts fell in lipid metabolism, amino acid metabolism, and redox pathways. Cross-omics integration pointed to pathway-level correspondence in carbohydrate and nucleotide metabolism, lipid remodeling, and antioxidant defense, suggesting the two layers report on a single underlying process rather than on parallel ones. Among the platelet-associated proteins pursued further, pro-platelet basic protein held up in an independent ELISA cohort, while MMRN1 and PDGFA moved in the same direction without reaching significance. The validation cohort is small and the authors are appropriately restrained about it, though a platelet-linked circulating signal in a condition usually framed as local lesion pathology is worth following.

**Microplastics induce osteoporosis via dose-divergent mechanisms: Multi-omics reveal metabolic compensation and inflammatory senescence**

**Zhang and colleagues** in *Journal of Hazardous Materials Advances* **showed** that polystyrene microplastics damage bone through mechanisms that change with dose rather than intensify with it. Rats received 0.13 or 1.3 mg/kg for four weeks, with particles visualized in femoral heads and integrated metabolomic and proteomic profiling backed by RT-qPCR validation. The low dose produced little measurable effect on bone metabolism, while the high dose caused frank bone loss, suppressed osteoblast function, disturbed osteoclast-mediated resorption, and switched on apoptotic and inflammation-driven senescence pathways absent at the lower exposure. Lipid, amino acid, and energy metabolism were disrupted differently at the two doses, and sustained elevation of Pg 34:1 and quinic acid tracked with skeletal damage. Dose-divergent mechanism is a genuine problem for risk assessment, since extrapolating downward from high-dose experiments assumes a continuity these data do not support.

**Towards early detection of pancreatic cancer: The current status of cohort studies by the Diabetes-Pancreatic Ductal AdenoCarcinoma Working Group**

**Maitra and colleagues** in *Pancreatology* **described** that the current state of the Diabetes-Pancreatic Ductal Adenocarcinoma Working Group, whose premise is that new-onset diabetes offers a window into occult pancreatic cancer. The group identified more than 20,000 subjects with glycemically defined new-onset diabetes and banked serial blood samples from 2,270 of them, with PDAC risk and the lead time from glycemic onset to clinical diagnosis now defined in a cohort of 18,838. That lead time is the entire argument for early detection in this population, and having it quantified prospectively changes what a candidate biomarker must demonstrate. Metabolomic and biomarker studies running on the biobank will report against Phase 3 validation criteria rather than against case-control convenience samples. The resulting repository, which incidentally captures a large number of type 2 diabetes subjects with serial sampling, will outlast the specific hypotheses it was built to test.

---

## Gut Microbial Metabolites as Therapeutic Levers

Microbiome studies earn their keep when they name a molecule. The three interventions below span an anthraquinone from traditional herbal medicine and two defined bacterial strains, and each traces its effect to a specific metabolite rather than stopping at a shift in community composition. Two work by subtraction, lowering a metabolite that drives host pathology, while the third works by addition, enriching a compound that acts on host cells directly. All three support the causal claim with something beyond correlation, whether fecal transplant, antibiotic depletion, or exogenous administration of the candidate metabolite, which is a standard this subfield has taken an unreasonably long time to adopt. The nominated mediators are chemically unremarkable small molecules, an oxidized amine, a hydroxy fatty acid,

an unusual amino acid, rather than exotic secondary metabolites. Therapeutic reasoning here runs through metabolism rather than around it.

---

**Rhein alleviates acute pancreatitis by inhibiting TMAO-mediated inflammatory signaling pathways and reducing acinar cell injury**

**Zhang and colleagues** in *Journal of Advanced Research* **demonstrated** that rhein alleviates acute pancreatitis by lowering microbial TMAO production rather than by acting on the pancreas directly. Antibiotic depletion, fecal microbiota transplantation, and in vitro culture established that the effect requires an intact gut community, and rhein itself concentrated in stomach and intestine rather than distributing systemically. Combining 16S and metagenomic sequencing with LC-MS on fecal samples and transcriptomics on acinar tissue, they placed TMAO upstream of TLR4/NF-κB/NLRP3 activation, with downstream oxidative stress, lipid peroxidation, and acinar necrosis. Serum TMAO concentration correlated with disease severity in patients, giving the mouse mechanism a clinical anchor. The result reframes poor systemic bioavailability as an asset rather than a liability, since acting locally on the microbiota is the point.

**Bifidobacterium breve B2798 Attenuates Diet-Induced Obesity Through Gut-Adipose Crosstalk**

**Zhao and colleagues** in *Food Frontiers* **reported** that Bifidobacterium breve B2798 reverses established diet-induced obesity through coordinated changes in gut and adipose tissue. Mice were kept on a high-fat diet for 12 weeks before treatment began, so the design tests reversal rather than prevention, and the strain reduced weight gain, epididymal and inguinal fat mass, and liver weight while improving insulin sensitivity without shifting glucose tolerance. Fecal metabolomics showed elevated hydroxy fatty acids including 12-hydroxystearic acid alongside partial correction of dysbiosis, with Faecalibaculum rodentium enriched and Coriobacteriia and Deferribacteres reduced. In epididymal fat, Slc2a4 and Acsm3 expression returned toward baseline and gene set enrichment pointed to increased fatty acid oxidation, thermogenesis, and oxidative phosphorylation. The dissociation between insulin sensitivity and glucose tolerance deserves attention, since it suggests the strain acts on adipose handling of lipid rather than on systemic glucose disposal.

**Lactobacillus johnsonii Suppresses Colorectal Cancer in Association With Intestinal Statine Enrichment and Tumor Cell Apoptosis**

**Chen and colleagues** in *Food Frontiers* **found** that Lactobacillus johnsonii suppresses colorectal tumors in association with intestinal enrichment of statine. Using AOM/DSS and MC38 syngeneic models, they combined microbiome and metabolomic profiling with tumor transcriptomics, then tested the candidate mediator in microbiota-depleted mice, normal human intestinal epithelial cells, and CRC lines.

Treatment activated TNF-related signaling and apoptosis-associated gene sets in tumor tissue, and exogenous statine reproduced key effects by upregulating CCL5, inducing tumor cell apoptosis, and slowing progression. The authors frame statine as a candidate rather than a proven mediator, and the microbiota-depletion arm is what makes that framing defensible. Whether the same axis operates in tumors arising outside inflammatory models is the obvious next question.

---

## Dietary and Feed Bioactives with Defined Physiological Targets

Functional ingredient research has a credibility problem rooted in endpoints chosen after the fact, and the way out is mechanism. Each study in this section starts from a food or feed component with a claimed physiological benefit and asks which pathway carries it, using metabolomics to nominate the intermediate and then testing that nomination with pharmacology, a dose series, or targeted assays. The results are usefully unflattering in places. One intervention shows a clear benefit only at an intermediate dose, with higher amounts performing worse than lower ones, while another produces measurable metabolic change with no effect on the growth endpoint it was presumably purchased to improve. A third reaches a receptor and a transcription factor, which is roughly where a nutritional bioactive stops being a supplement and starts being a lead compound. Reporting the null performance outcomes alongside the mechanistic findings is what makes this body of work usable.

---

**Oral bovine osteopontin alters intestinal immune marker profiles in juvenile rats: concurrent changes in the aryl hydrocarbon receptor/signal transducer and activator of transcription 3/interleukin-6 axis**

**Li and colleagues** in *Food Bioscience* **showed** that oral bovine osteopontin shapes intestinal immune markers in juvenile rats along a dose-specific rather than dose-proportional curve. Medium-dose supplementation raised serum antibody and complement levels, lowered pro-inflammatory cytokines, and improved jejunal barrier integrity, while both lower and higher doses did comparatively little. Molecular profiling placed intestinal osteopontin expression in epithelial cells co-localized with macrophages, alongside a raised T-bet/GATA3 transcript ratio and upregulation of the AHR/STAT3/IL-6 axis at both protein and mRNA level. Serum metabolomics identified accompanying shifts in immune-associated metabolites, though the directional relationships among these changes remain correlational and the authors say so. For early-life nutrition formulation, the non-monotonic dose response is the finding that matters most.

**Peptide IRW upregulates ACE2 in spontaneously hypertensive rats via dopamine/D1R signaling pathway**

**Wang and colleagues** in *Journal of Advanced Research* **demonstrated** that the bioactive tripeptide IRW raises ACE2 through dopamine signaling at the D1 receptor. Transcriptomics and metabolomics on mesenteric arteries from treated spontaneously hypertensive rats identified 651 genes co-expressed with ACE2 and flagged Nr4a1 among 17 predicted transcription factors, while metabolomics showed dopamine rising in proportion to ACE2 expression. In endothelial cells, dopamine upregulated both ACE2 and Nr4a1, an effect abolished by the D1R antagonist SCH23390 and reduced by Nr4a1 knockdown. D1R blockade in vivo removed both the ACE2 induction and the antihypertensive effect, closing the loop between the correlative omics and the pharmacology. Dopamine as the intermediate between a dietary peptide and renin-angiotensin regulation was not an obvious hypothesis, and it would not have surfaced without the metabolomic arm.

### Phytogenic Modulation of Rumen Fermentation Reshapes Amino Acid Metabolism in Weaned Dairy Calves: A Mechanistic Insight

**Fouad and colleagues** in *Animals* **reported** that a phytogenic blend of cinnamaldehyde, capsicum, curcumin, and saponin reshapes amino acid metabolism in weaned calves without touching growth performance. Sixteen Holstein calves received either the blend at 0.5 g per kg dry matter or no supplement for 60 days, with rumen fermentation, serum amino acids, and performance measured. Ruminal pH fell while acetate, propionate, and valerate rose, and serum leucine dropped as aspartate increased, with enrichment analysis converging on alanine, aspartate, and glutamate metabolism. Growth was unchanged, a useful negative result given how these products are marketed. The value of the study lies in showing that a fermentation shift can propagate into host amino acid pools while leaving the production endpoint alone, a dissociation that argues against average daily gain as the sole readout of phytogenic efficacy.

---

## Microbial Control of Food Quality: Fermentation and Postharvest

Food fermentation research has quietly become one of the more demanding applications of untargeted metabolomics, because the question is never simply what changed but when, driven by which organism, and whether the process can be steered. The studies collected here treat microbial metabolism as something to be scheduled rather than merely observed, whether by inoculating a defined starter, staging temperature, tracking community succession across a fermentation, or applying a volatile to shut fungal growth down. Time-resolved sampling is the common methodological commitment, and it pays off, since several of the metabolic transitions reported here would be invisible in an endpoint comparison. The reach extends past flavor into lignocellulosic valorization and postharvest shelf life, putting the same analytical toolkit to work on waste streams and on losses after harvest. Pairing metabolomics with proteomics or transcriptomics is now standard in this literature, and the better studies use the second layer to assign enzymatic

responsibility rather than to decorate the first. Process control is the destination, and a few of these are close enough to write specifications from.

---

**Insights into the flavor formation mechanism of reduced-salt shallow-fermented sausages under oscillatory temperature fermentation with starter culture inoculation**

**Chen and colleagues** in *Food Research International* **reported** that a two-stage oscillatory temperature fermentation with a Staphylococcus SM-75 starter can carry the flavor of reduced-salt Sichuan air-dried sausage. Esters, aldehydes, and alcohols dominated the key aroma compounds, with ethyl caprylate, ethyl caprate, ethyl palmitate, ethyl caproate, 3-methyl-1-butanol, phenylacetaldehyde, and phenethyl alcohol carrying most of the distinctive profile. Untargeted metabolomics assigned amino acid and lipid metabolism as the principal routes to those compounds and separated their contributions across the process: the hot-air stage drove proteolysis and the transamination and decarboxylation steps yielding compounds such as phenethyl alcohol, while the cold-air stage favored enzymatic fatty acid degradation and esters including ethyl palmitate. Reconstructing the flavor network stage by stage turns temperature staging into a tunable variable rather than a traditional practice. Salt reduction without flavor loss is a persistent formulation problem, and this is a plausible route to it.

**Metabolomic and proteomic analysis reveals the metabolic changes of alkaloids in Ganoderma lucidum fermented tea**

**Dai and colleagues** in *Food Research International* **found** that alkaloid profiles shift substantially during fermentation of Ganoderma lucidum tea, and identified the enzymes plausibly responsible. Paired metabolomics and proteomics returned 832 differential metabolites enriched in alkaloid and amino acid pathways, with 11 alkaloids and related compounds changing across the process and 24 differentially expressed proteins linked to alkaloid metabolism. Targeted quantification pointed to two serine hydroxymethyltransferase isoforms acting indirectly through glycine, serine, and threonine metabolism rather than on alkaloid pathways themselves. That indirect route is the interesting part, since it suggests one-carbon metabolism sets the precursor supply on which alkaloid accumulation depends. For product development it also identifies a control point that fermentation conditions could plausibly manipulate.

**Multi-Omics Characterization of Temporal Microbial and Metabolic Dynamics During Solid-State Fermentation of Mulberry Branch Residue**

**Qian and colleagues** in *Agronomy* **reported** that solid-state fermentation of mulberry branch residue proceeds through a clear microbial succession with matching metabolic turnover. A defined consortium of lactic acid bacteria, Bacillus

subtilis, and Saccharomyces cerevisiae was tracked for 12 days at 35 degrees C using 16S sequencing and untargeted LC-MS, with Bacillus at 19.5 percent of the bacterial community early on before more diverse assemblages took over. Metabolomics annotated 2,782 features dominated by lipids, organic acids, and phenylpropanoids, and OPLS-DA singled out pseudouridine and 3-methylxanthine as stage-discriminating. Correlation analysis tied particular genera to metabolite classes, including Geobacillus with alkaloids and glycerophospholipids, while FAPROTAX predictions of persistent chemoheterotrophy are inference rather than measured function and the authors flag them as such. The study is explicitly descriptive, the right posture for a first pass at a waste stream nobody has characterized in this detail.

**Opposite effects of garlic essential oil and protein fertilizer on aerial hyphae outbreak of wine-cap mushrooms: Mechanisms involving the redox imbalance and membrane destabilization**

**Yin and colleagues** in *Postharvest Biology and Technology* **showed** that garlic essential oil suppresses aerial hyphal outbreak in stored wine-cap mushrooms by destabilizing membranes rather than by generally slowing metabolism. Treatment disrupted intracellular ROS homeostasis and compromised membrane integrity, while a protein fertilizer comparator preserved both redox balance and hyphal structure and promoted the growth the oil suppressed. Integrated transcriptomics and metabolomics located the effect in coordinated perturbation of glycerolipid and sphingolipid metabolism, with suppressed glutathione biosynthesis leaving antioxidant capacity depleted. Running an inhibitory and a stimulatory treatment side by side is what licenses the mechanistic claim, since the contrast separates targeted lipid disruption from nonspecific metabolic slowdown. Aerial hyphae are a real commercial loss in edible fungi, and a natural volatile with an identified mode of action is a more defensible preservative than one selected empirically.

---

## Environmental Cues and Regulatory Control of Metabolic Flux

Whether the organism is a maize seedling, an engineered tobacco line, a grafted medicinal shrub, or a genome-reduced bacterium, the operative question is the same: what decides where carbon goes. The studies here approach it through four different kinds of input, an applied osmolyte, a heterologous transcription factor family, a rootstock, and extracellular pH, and each pairs metabolomics with a second layer to catch the regulation as well as the outcome. Two findings sit awkwardly with the textbook picture. Reprogramming in these systems is frequently achieved by redistributing flux among existing pathways rather than by switching new ones on, and in the bacterial case it happens without canonical transcriptional regulators at all, through selection among prewired modules and reorganization of RNA modifications. That second result matters beyond microbiology, since it points to a regulatory layer that metabolomics studies routinely omit. The practical thread is that modest, well-

chosen perturbations at the regulatory level produce larger metabolic effects than brute-force pathway engineering usually manages.

---

Glycine Betaine Mitigates Flooding and Drought Damage in Maize by Regulating Respiration, ROS Homeostasis, and Metabolism

Mao and colleagues in Physiologia Plantarum showed that foliar glycine betaine buffers maize against flooding and drought through the same core adjustments, despite the two stresses pulling in opposite directions. Three genotypes differing in water tolerance were stressed at V3 in a factorial design with sampling at day 4, day 8, and after recovery, and both stresses cut root growth, relative water content, and water potential while raising hydrogen peroxide, superoxide, and membrane permeability. Glycine betaine restored root traits and water status, stabilized instantaneous water use efficiency, and moderated fermentative metabolism by raising lactate dehydrogenase activity while restraining pyruvate decarboxylase, limiting ethanol and pyruvate accumulation. Transcriptomic and metabolomic profiling of the tolerant and sensitive lines pointed to upregulated starch, sucrose, and glycolytic gene expression alongside changes in lipid and betaine-related genes consistent with membrane remodeling. Two years of field data confirmed that mitigation is genotype-dependent and largest in the sensitive line, which is the result breeders and agronomists will care about.

Metabolic engineering of tobacco plants using Carica papaya NAC transcription factors: a biotechnology tool to improve plant metabolite production

Aguilar-Santana and colleagues in Biotechnology Letters reported that Carica papaya NAC transcription factors expressed in Nicotiana tabacum reprogram specialized metabolism well beyond any single pathway. GC-MS of methanolic extracts from transformed and non-transformed plants showed increased phytosterols including campesterol, beta-sitosterol, and stigmasterol, while untargeted profiling picked up broader shifts across organic acids, steroids, and lipids. Transformed seedlings also accumulated phenylpropanoids such as coumarin and sinapaldehyde, alkaloids including tropinone and allocryptopine, and aromatic amino acid derivatives such as tryptamine and phenylacetic acid. Rising acetate and acetyl-CoA pools point to flux redirection toward these branches rather than to enhancement of individual enzymatic steps. Transferring a regulator across species is a blunter instrument than pathway-specific engineering, and the breadth of the response here shows both the appeal and the control problem.

The effects of grafting on the stems of Eleutherococcus sessiliflorus were jointly analyzed by transcriptomics and metabolomics

Liang and colleagues in Frontiers in Plant Science found that grafting Eleutherococcus senticosus onto Eleutherococcus sessiliflorus rootstock raises

secondary metabolite accumulation in the scion stem without altering its basic compositional profile. Self-grafted controls showed no significant effect, while cross-root grafting left moisture, total ash, and extractive content unchanged and improved nutrient accumulation and antioxidant capacity. Paired transcriptomics and metabolomics with qRT-PCR validation located the response in unsaturated fatty acid and amino acid biosynthesis, with differential expression of SCD, FAD2, icd, and PK. A clear shift in specialized metabolism with no movement in the standard quality indices should give pause to anyone relying on those indices to grade medicinal plant material. For a species under collection pressure in the wild, rootstock selection is a more tractable lever than breeding.

Integrated multi-omics analysis reveals a pH-driven metabolic and translational switch in Ureaplasma parvum

Hase and colleagues in Microbiology Spectrum reported that Ureaplasma parvum handles pH change by choosing between prewired programs rather than by building new ones. Combining proteomics, metabolomics, and RNA modification profiling, they found the organism running energy metabolism and ATP synthesis with a stress-counteracting proteostasis arm at neutral pH, then switching under acid stress to biosynthesis and translation, marked by ribosomal protein upregulation and accumulation of translation precursors and spermidine. The switch was accompanied by dynamic reorganization of the epitranscriptome, implicating post-transcriptional control in an organism with almost no transcription factors. For a minimal-genome pathobiont this offers a coherent account of adaptive plasticity without regulatory circuitry, and it makes pH-linked RNA modification patterns a candidate readout of cellular state. The framework should transfer to other genome-reduced organisms, where the absence of canonical regulators has more often been noted than explained.

Back to Top

## Metabolomist Podcast

*|MC:SUBJECT|*



## Metabolomics Events

Back to top

### MANA 2026

### 8th Annual Conference

**September 8 - 11, 2026**

**Venue: UC Davis, California, USA**

The Metabolomics Association of North America (MANA) is a non-profit organization

that brings together a community of dedicated scientists and professionals in the field of metabolomics. With members from Canada, Mexico, and the USA, MANA is committed to fostering cooperation, coordination, and the advancement of metabolomics research in North America.

The 8th Annual MANA Conference will be hosted at the University of California. Check out the website for program information, speakers, events, registration, awards, and more.

**Check for more details**

## Benelux Metabolomics Days 2026

### September 22 - 23, 2026

**Venue: Antwerpen, Belgium**

The Benelux Metabolomics Days is the annual conference of the Benelux Metabolomics Centre (BMC), bringing together the metabolomics community from the Netherlands, Belgium, and Luxembourg. It will take place at Stadscampus – Hof van Liere, University of Antwerp (Belgium). Hosted by Professor Wout Bittremieux and Professor Adrian Covaci, the conference will feature keynote lectures and sessions highlighting emerging and topical areas in metabolomics. Oral and poster presentations by early-career researchers will be selected through the abstract submission process.

**Visit the website for more details**

## Introduction to Applying GC-MS in Untargeted Metabolomics

### September 22 - 24, 2026

**Venue: Liverpool, England, United Kingdom**

The course provides an introduction to metabolomics, experimental design and the application of GC-MS in untargeted metabolomics, sample preparation, hands-on data acquisition using the LECO Pegasus BT 4D GC x GC-ToF MS, data processing and analysis. The course is delivered using a combination of lectures, laboratory sessions and computer workshops to develop your knowledge, skills and confidence to apply GC-MS in your own research. The course is aimed at individuals with

minimal knowledge of GC-MS based metabolomics, and only a small number of attendees are accepted on each course to maximise opportunities for laboratory experience and interaction with instructors.

**Registration and event details**

## CMMC Symposium 2026 – Metabolic Control of Tissue Regeneration and Degeneration

### September 28 - 30, 2026

**Venue: Cologne, Germany**

The CMMC Symposium 2026 will focus on the emerging role of metabolic programs and metabolites as key regulators of tissue regeneration and degeneration. Beyond providing energy and cellular building blocks, metabolic pathways control fundamental processes such as cell proliferation, differentiation, reprogramming and immune responses. By linking mechanistic insights with clinical perspectives, the symposium will address major challenges in molecular medicine and human disease.

Keynote speakers:

- Elly Tanaka (IMBA, Vienna, AT)
- Axel Roers (University Hospital Heidelberg, DE)
- Gerald I. Shulman (Yale School of Medicine, US)

The meeting is open to all and free of charge but **free registration is required**.

**Visit the website for more details**

## 5th Nordic Metabolomics Conference 2026

### September 28 - 30, 2026

**Venue: Uppsala, Sweden**

The 5th Nordic Metabolomics Conference is the official annual conference of the Nordic Metabolomics Society and will be organized at Uppsala Konsert & Kongress (UKK) in Uppsala, Sweden. The city hosts Uppsala University, who has a longstanding tradition in Analytical Chemistry and Mass Spectrometry. The conference will host the entire depth of the metabolomics community in the Nordic

Countries, high profile (inter)national speakers, will include an Early Career Event, and a dinner at Uppsala Castle.

Abstract submission deadline for poster presentations - **September 1, 2026**

**Check for more details**

## MSACL 2026

## 16th Annual Conference & Exhibits

### October 4 - 9, 2026

**Venue: Montreal, Canada**

MSACL's mission is to support the community in translating pre-clinical research to clinical patient care. The conference is focused on providing a forum of interaction for participants in all stages of the development, advancement and use of analytical tools, including data analytics, in the clinic to improve patient care. Although the group primarily focuses on mass spectrometry, presentations and discussion on other platforms are welcome.

There were 628 attendees for MSACL 2025. 600-700 attendees are expected for MSACL 2026.

**Check for more details**

## MANA SODAMeet

### October 13, 2026

**Venue: Online**

The goal of SODA is to provide a community-driven resource of actively-maintained software, test datasets used for software benchmarking, and results produced by software. SODAMeets is a platform where data generators and computational scientists can share their use of software/data. During SODAMeets (every 2 months), two speakers will present on software or data they would like to share with the community, emphasizing how these software/data are used. Speakers will be requested to fill out a form on our SODA website so that we collect relevant information on these software/data presented.

*|MC:SUBJECT|*

Join the web seminar

## Australian Lipid Meeting 7 & 5th International Lipidomics Society Conference

### October 18 - 21, 2026

**Venue: Perth, Western Australia**

This special event unites two major gatherings in lipid research, bringing together researchers, scientists, clinicians and industry leaders from around the world to share discoveries, promote collaborations and shape the future of lipidomics.

Visit website for more details

## Introduction to Applying LC-MS in Untargeted Metabolomics

### October 26 - 28, 2026

**Venue: Liverpool, United Kingdom**

The course introduces attendees to the metabolomics workflow and applying LC-MS in metabolomics. It is designed to develop knowledge, skills and experience in small group session with hands-on experience in sample preparation, LC-MS data acquisition (MS and MS/MS), data analysis and metabolite annotation and identification. The course is aimed at individuals with minimal knowledge of LC-MS based metabolomics and only a small number of attendees are accepted on each course to maximise opportunities for laboratory experience and interaction with instructors.

Registration and additional event details

## 2027 Metabolomics and Human Health Gordon Research Conference and Gordon Research

## Seminar

### February 27 - March 5, 2027

**Venue: Liverpool, United Kingdom**

The 2027 Metabolomics and Human Health Gordon Research Conference and affiliated Gordon Research Seminar will bring together scientists across metabolomics, exposomics, One Health, nutrition, environmental health, and human disease. The GRS is organized by and for early-career researchers, including students and postdoctoral fellows, while the GRC provides opportunities to engage with world leaders in metabolic research and build collaborations across disciplines. The 2027 GRC theme is “One Health, the Exposome, and Lifecourse Health and Disease,” and the GRS theme is “Decoding Metabolism at the Health-Disease-Environment Interface Through Emerging Technologies.”

**Gordon Research Conference**

**Gordon Research Seminar**

---

# Metabolomics Jobs

## Metabolomics Jobs

If you have a job to post, please email the MetaboNews team at metabolomics.innovation@gmail.com

We may remove a listing after 6 months if we do not receive a confirmation that it is still necessary. However, if you would like us to repost it, please contact us.

| Job Title | Employer | Location | Source |
|---|---|---|---|
| Postdoctoral position "Computational Discovery of Mechanisms Linking | IDIBGI & RMIT | Girona, Spain | |

| | | | |
|---|---|---|---|
| Metabolism, Inflammation, and the Gut–Brain Axis" | University | and Melbourne, Australia | RMIT University |
| Postdoctoral Associate in Microbial Metabolomics | Yale School of Public Health | New Haven, CT, USA | MANA |
| ARISE Fellowship program | ARISE Career Accelerator for Research Infrastructure Scientists | Six EMBL sites | European Molecular Biology Laboratory (EMBL) |

Back to top

# MetaboNews Feedback Form

Thank you for being a part of MetaboNews!

Your input means a lot to us, and we're eager to hear your thoughts on how we can improve our newsletter. Please take a moment to share your opinions with us at metabolomics.innovation@gmail.com

Back to top

*|MC:SUBJECT|*