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Decoding the Interplay Between Metabolism and Immunity

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LUXEMBOURG INSTITUTE OF HEALTH

EIMN European Immunometabolism Network

10.06.2026

SESSION 1 Metabolic regulation of adaptive immunity

SESSION 2 Metabolic regulation of innate immunity

11.06.2026

SESSION 3 Immunometabolism in Chronic Inflammatory Diseases

SESSION 4 Immunometabolism in Cancer

SESSION 5 Immunometabolism in Infections

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SESSION 6 Translational immunometabolism and new technologies

Full programme: https://eimn2026.lu/en/agenda/

Claudia Mauri

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Two Paths, One Circuit: Metabolic and Redox Bases of Breg Action in Autoimmunity and Cancer

B cells can adopt either regulatory or effector fates, with regulatory B cells (Bregs) contributing to immune tolerance, and effector B cells, such as plasma cells, driving antibody responses. The mechanisms guiding this fate decision remain incompletely understood. We have recently identified a critical role for redox balance in shaping B cell identity. Specifically, the thioredoxin system supports Breg differentiation by maintaining mitochondrial homeostasis and limiting reactive oxygen species (ROS). Elevated ROS levels, by contrast, disrupt this balance and promote effector B cell development.

In this presentation, I will discuss how redox-regulated checkpoints influence B cell fate and how inflammatory cues in the microenvironment, such as signals from innate immune cells, can tip the balance away from regulation and toward effector function. These findings offer new insights into how redox metabolism integrates environmental signals to shape B cell responses in contexts such as autoimmunity, chronic inflammation, and cancer.

Dirk Brenner

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Metabolic Regulation of T Cell Function

T cell activation and differentiation are tightly coupled to metabolic rewiring. Upon antigen encounter, T cells profoundly reshape their metabolism to meet the demands of clonal expansion, effector function and the formation of long-lived memory. Our laboratory studies how individual metabolic pathways are integrated into these processes and how they actively shape immune cell fate decisions.

A central focus of our work is one-carbon metabolism, a pathway that supplies units for nucleotide synthesis, methylation reactions and redox balance, and that is strongly induced during T cell activation. While it has long been regarded primarily as a biosynthetic supply line for proliferating cells, our findings indicate that its role in T cells is considerably broader.
We examine how this pathway influences CD8 T cell responses across acute and chronic antigen exposure, shaping not only the early activation programme but also the balance between effector, memory and exhausted states.

Our data indicate that one-carbon metabolism shapes T cell fate through mechanisms that extend beyond its established biosynthetic role and challenge the prevailing view of the pathway. These findings reveal a previously underappreciated layer of metabolic control, with translational implications for cancer immunotherapy.

Alessandro Carrer

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Veneto Institute of Molecular Medicine (VIMM),
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Italy

Metabolic control of b cell epigenome degenerates during aging

Metabolic and epigenetic reprogramming can be linked. Our work identifies a nutrient-sensing axis which signals to the B cell epigenome during the germinal center (GC) reaction.

We found that T lymphocyte-derived signals activate the acetyl-CoA producing enzyme ATP-Citrate Lyase (ACLY), promoting B cell proliferation and commitment to the GC reaction through metabolic-dependent histone acetylation. Isotope tracing revealed significant contribution of T cell-secreted lactate to the de novo production of acetyl-CoA in engaged B cells that in turn leads to increased Histone 3 Lysine 27 (H3K27) acetylation. Lactate supplementation promotes B cell proliferation, which is inhibited by MCT1 blockade. In addition, both ex vivo and in vivo ablation of ACLY impairs GC development.

Mechanistically, lactate-derived pyruvate enters the mitochondria of activated B cells to feed the malate-citrate shuttle, to satisfy increasing demand for acetyl-CoA. This is used for lipid synthesis but also to enhance histone acetylation at loci involved in cell proliferation. Genome-wide mapping (CUT&tag) showed that lactate-mediated histone acetylation activates CBL signaling.

Metabolic control over GCB cell histone acetylation is lost during physiological aging of both humans and mice. Leveraging spatial proteomics and genome-wide analyses we found global depression of histone acetylation in aged GCB cells, which however retain intact sensitivity to exogenous stimuli. Altered lactate availability at lymph nodes of aged organisms and decreased expression of Slc16a1 (MCT1) in aged GCB cells together restrict lactate-dependent epigenetic reprogramming.

Altogether, our data show that metabolic crosstalk between B/T cells is critical for GC reaction and impaired during aging.

Emmanouil Stylianakis

Institute of Molecular Medicine,
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A single cell mapping of age-associated B cells reveals profound phenotypic plasticity and metabolic heterogeneity in aging mice

CD21$^-$CD23$^-$T-bet$^+$ age-associated B cells (ABCs) constitute a heterogeneous B cell population that expands in diverse contexts, including aging, viral infection, and autoimmune diseases such as systemic lupus erythematosus. ABCs are typically defined by the absence of CD21 and CD23 and the expression of the integrins CD11b and CD11c. However, their precise role in adaptive immunity remains incompletely understood, with studies supporting context-dependent functions ranging from pro-inflammatory activity to tolerogenic or exhausted states.

Here, we assessed the metabolic and transcriptional heterogeneity of ABCs in aged C57BL/6 mice (>80 weeks). Flow cytometry–based measurements of cellular redox balance using NADH and FAD autofluorescence revealed two distinct ABC subpopulations: one displaying a normometabolic profile and another displaying a hypermetabolic state characterized by increased cell size, enhanced granularity, elevated H3K9 histone acetylation, consistent with increased OXPHOS activity, and distinctive phenotypic features. Calcium flux analysis showed increased basal intracellular calcium levels compared to follicular and marginal zone B cells, accompanied by reduced responsiveness to further B cell receptor stimulation.

Single-cell RNA sequencing confirmed this metabolic bimodality and identified transcriptional programs consistent with enhanced fatty acid utilization and activation of pattern recognition receptor pathways, including NOD-dependent inflammasome priming. The ABC cell cluster exhibited expression of activation markers CD80 and CD86, inhibitory ITIM-containing receptors such as CD22 and Siglec-G. Transcription factor analysis revealed enrichment of aryl hydrocarbon receptor (AhR) activity, previously linked to regulatory-like B cell phenotypes.

Collectively, these findings define a distinct ABC subset with features consistent with a resilient functional state capable of maintaining activity/function under conditions of chronic, age-associated inflammation (inflammaging).

Mauro Corrado

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Cardiolipin deficiency exposes ferroptosis vulnerability in T cells

Cardiolipin-deficient T cells are characterized by elevated oxidative stress. Nevertheless, they are still viable and retain limited cytokine production. We hypothesized this could be achieved by compensatory metabolic rewiring mechanisms activated by cardiolipin deficiency. We found that cardiolipin deficient T cells shift from glucose to glutamine utilization to fuel a “broken” TCA cycle and sustain a critical anti-oxidant response. Indeed, glutaminolysis inhibition (but not fatty acid or pyruvate oxidation inhibition) in cardiolipin deficient T cells leads to uncontrolled accumulation of reactive oxygen species and lipid peroxidation which sensitize cells to ferroptosis. Overall, we unravel a previously unknown metabolic vulnerability in T cells. Notably, this metabolic vulnerability can also sensitize T-cell lymphomas to cell death, pointing to potential therapeutic strategies for diseases that remain difficult to treat because of resistance, relapse, or chronic persistence.

Marine Tronchon

CIRI, Centre International de Recherche en Infectiologie, (Team Epidermal Immunity and Allergy); INSERM, U1111; Univ Lyon; Université de Lyon 1; Ecole Normale Supérieure de Lyon; CNRS, UMR 5308, Lyon, France

Metabolic reprogramming underlies differential longevity of skin resident memory CD8⁺ T cells

T cell metabolism is a central regulator of effector differentiation, memory formation, and cell survival. Tissue-resident memory CD8⁺ T cells (TRM) exhibit specialized, tissue-adapted metabolic programs that distinguish them from their circulating memory counterparts. However, the extent to which metabolic heterogeneity within the TRM compartment shapes their durability and functional longevity remains unclear.

Using a preclinical model of allergic contact dermatitis, we recently identified two distinct clusters of CD8⁺CD69⁺CD103⁺ TRM cells, with divergent persistence profiles: a transient subset that predominates in the early phase post inflammation, and a long-lived subset that persists for more than a year. Here, we sought to determine whether distinct metabolic programs underlie these temporally defined TRM subsets.

Single-cell transcriptomic analyses revealed that the subset that persists at short-term is enriched for bioenergetic and metabolic remodeling pathways (OXPHOS, ATP/glucose metabolism, lipid/xenobiotic metabolism, redox stress), while the long-term subset is characterized by oxidative stress response and redox regulatory programs together with proliferative/recall signatures.

Functionally, ex vivo mitochondrial profiling demonstrated increased mitochondrial content in the short-term TRMs as assessed by MitoTracker staining, enhanced polarization (TMRE), and elevated mitochondrial ROS production (MitoSOX), supporting a high bioenergetic setpoint. Conversely, the long-term TRMs maintained a lower mitochondrial tone, suggesting a metabolically restrained state compatible with durable tissue persistence.
Together, these data support a model in which bioenergetically engaged, mitochondria-high TRMs are optimized for rapid recall but exhibit reduced long-term persistence, whereas a metabolically restrained state favors tissue longevity. Ongoing work combining metabolic flux assays, single-cell metabolic inference, and genetic perturbation of energy-sensing pathways will establish the causal contribution of metabolic programming to TRM durability.

Christoph Wilhelm

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Germany

Metabolic cooperations regulating barrier immunity

The Wilhelm lab studies the metabolic control of mucosal immune cells. Specifically, the lab aims to determine how dietary restriction and fasting affect the immune system, and how exogenous and endogenous metabolites influence tissue-resident immune cells and barrier immunity. Recent work revealed that a ketogenic diet can be an effective dietary intervention strategy for treating chronic airway inflammation, and that ketogenesis triggered by severe respiratory infections supports T cell function by providing ketone bodies as an alternative carbon source. The lab's overarching goal is to understand the link between Westernization and the growing health problem of chronic inflammation, as well as the increased immune-mediated pathology in people with metabolic diseases.

Stefanie K Wculek

Institute for Research in Biomedicine (IRB)
Barcelona, Spain

How innate immune cells adapt to changing environments - diverse tales of mitochondria

My lab investigates tissue-specific adaptations of dendritic cells, neutrophils and macrophages in health and non-infectious conditions with a focus on immunometabolism. We revealed how those innate immune cells distinctly tailor their mitochondrial metabolism to tolerate and drive in such diverse milieus. Moreover, I will present how the active engagement of the electron transport chain crucially supports the context- and environment-dependent functions of dendritic cells, neutrophils and macrophages via entirely different molecular mechanisms.

Aitor Jarit Cabanillas

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The role of the Electron Transport Chain in Trained Immunity

Innate immune cells, such as macrophages, that have responded to a first proinflammatory insult may develop an exacerbated immune response to a second challenge, a process called trained immunity (TI). TI is driven by an interconnected metabolic and epigenetic reprogramming triggered by stimuli such as ß-glucan. Understanding TI's molecular mechanisms is key to developing strategies to either dampen it in autoinflammatory conditions or enhance innate immunity against pathogens.

From the metabolic perspective, monocytes stimulated with ß-glucan upregulate glycolysis and glutaminolysis and accumulate tricarboxylic acid cycle metabolites, processes reported to be involved in both TI induction and LPS-induced endotoxin tolerance. Here, we have shown that, unlike LPS stimulation, WGP stimulation enhanced mitochondrial metabolism and respiration. Surprisingly, inhibiting mitochondrial Complex I (CI) boosted TI, whereas Complex II (CII) inhibition reduced it. Metabolically, WGP stimulation alone led to a phenotype characteristic of NADH-reductive stress, which was further enhanced by CI inhibition. This phenotype showed elevated levels of glycolytic metabolites, purines, and S-adenosylmethionine, the main methyl donor in epigenetic reactions. NADH accumulation and manipulation of the NAD+/NADH ratio in wild-type macrophages enhanced TI, similar to the effect observed in CI-deficient macrophages. Moreover, expression of genes involved in one carbon metabolism was induced upon WGP stimulation but further upregulated upon CI inhibition. Finally, inhibition of onecarbon metabolism reduced the boosted trained responses of CI-deficient macrophages. This study sheds light on the contribution of the electron transport chain and its regulated metabolites to TI induction, highlighting a potential role of NADH-reductive stress in TI.

Danilo Norata

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GLP-1–dependent immunometabolic control of periprandial neutrophilia during high-fat diet adaptation

Background
Transitions between fasting and feeding impose acute metabolic shifts that require tight coordination with innate immunity. While chronic high-fat diet (HFD) induces immunometabolic reprogramming of granulocyte–monocyte progenitors (GMPs), promoting inflammasome activation and cytokine overproduction, it remains unclear how immune cells dynamically respond to periprandial metabolic cues. Incretin signaling represents a key interface between nutrient sensing and systemic metabolism, but its role in shaping acute immune responses is poorly defined.

Methods
We characterized immunometabolic adaptations across the fasting–feeding transition in mice fed HFD (60% energy from fat) or standard diet (SFD). Analyses were performed after overnight fasting and up to 4 h of-feeding, integrating circulating immune profiling, plasma proteomics, neutrophil activation states, and bone marrow GMP transcriptomics.

Results
Feeding triggered a rapid increase in circulating neutrophils within 15 minutes, sustained by Cxcr4-dependent retention, and preceding monocytosis (≥2–4 h). This periprandial neutrophilia was amplified by HFD and further potentiated after short-term (7-day) metabolic adaptation, coinciding with insulin resistance and transcriptional activation of GMPs. The response was dependent on intestinal lipid sensing, as parenteral lipid delivery failed to recapitulate it. Mechanistically, glucagon-like peptide-1 (GLP-1) signaling acted as a key immunometabolic regulator: GLP-1 receptor antagonism enhanced neutrophil mobilization during HFD feeding, whereas insulin co-administration suppressed it. This insulin-dependent restraint was lost after HFD adaptation, linking impaired incretin–insulin signaling to heightened innate immune activation. In humans, postprandial neutrophilia correlated with glucose and triglyceride excursions but not with systemic inflammatory markers, indicating a metabolically driven, non-canonical inflammatory response.

Conclusions
These findings identify a rapid, incretin-modulated neutrophil response as a core component of periprandial immunometabolic adaptation. A GLP-1–insulin axis integrates nutrient sensing with innate immune surveillance, and its disruption during HFD-induced insulin resistance promotes exaggerated neutrophil mobilization. This work establishes periprandial neutrophilia as a dynamic immunometabolic process linking dietary cues to early immune activation.

Silja Vittoria Malkewitz

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Radical Scavenging by 3-Hydroxyanthranilic Acid (3-HAA) Defines a Distinct Antioxidant Module of the Kynurenine Pathway in Arthritis

The kynurenine pathway (KP) is the major route of tryptophan catabolism and is induced during inflammation, positioning it at the intersection of immune activation and metabolic reprogramming. The KP plays multiple roles: modulating immunity via tryptophan depletion, immunoregulatory metabolites, and NAD^+ synthesis.

To investigate the role of previously described KP activation in rheumatoid arthritis (RA), we analyzed single-cell RNA sequencing of RA synovial biopsies and found upregulated kynureninase (KYNU), the enzyme upstream of 3-HAA, in synovial monocytes and blood-derived macrophages. Notably, in bulk RNA sequencing and metabolomics of circulating CD14+ monocytes from RA patients, this metabolic signature was also detectable, with upregulated expression of the proximal KP (IDO1, KMO, KYNU) and accumulation of upstream intermediates (kynurenine, 3-HAA). Consistently, serum metabolomics in large pre-clinical and established RA cohorts showed prominent 3-HAA accumulation.

In vitro, we observed ROS scavenging activity of 3-HAA in cell-free assays as well as primary monocytes within 1 hour of administration. Beyond ROS scavenging, 3-HAA modulated glutathione redox balance at 6 hours post-treatment and induced transcriptional upregulation of Nrf2 target genes at 24 hours in monocyte-derived macrophages. Notably, 3-HAA treatment further exerted a protective effect in an LPSinduced sepsis mouse model, establishing in vivo relevance.

We propose that 3-HAA's antioxidant activity helps inflammatory myeloid cells buffer ROS to limit oxidative tissue damage in RA. We hypothesize that circulating monocytes deliver antioxidant 3-HAA to inflamed joints by upregulating the proximal kynurenine pathway. This positions a distinct KP module in blood-derived myeloid cells as a target for novel therapeutics.

Luke O'Neill

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Mitochondrial Endosymbiosis and inflammatory diseases: an abundance of therapeutic targets for inflammatory diseases

Mitochondrial disturbance is a feature of inflammatory cells, and we have been analysing mitochondrial metabolites, notably itaconate and fumarate in inflammatory macrophages. Itaconate derivatives are anti-inflammatory and have potential for the treatment of immune and inflammatory diseases. We have found that the cytokine GDF-15 is a key signal being driven by itaconate and fumarate, as well as general disturbance of mitochondria. It has anti-inflammatory effects, can limit food intake and thereby control obesity, and may act to take the pressure off damaged mitochondria in inflammation. Overall evidence is growing that a break in mitochondrial endosymbiosis might be a reason for the increasing incidence of autoimmune and inflammatory diseases. NLRP3 is an important target that becomes activated and there are over 20 inhibitors currently in development for a range of inflmamtory diseases. Other targets outside mitochondria include the enzyme PKM2 which is key to the Warburg metabolism that defines inflammation and is also being targeted in the clinic. These insights are providing a new view of metabolism in immunity and inflammation and might indicate new therapeutic approaches.

Michael Heneka

Director Centre of Systems Biomedicine at the University of Luxembourg (LCSB), University of Luxembourg

The dual and stage-dependent role of innate immune activation in Alzheimer's disease

The accumulation of neurotoxic amyloid beta peptides along with neurofibrillary tangle formation are key pathological hallmarks of Alzheimer's disease. The brain has been considered as an immune-privileged organ, however, increasing evidence from translational, genetic, and pathological studies suggests that activation of distinct innate immune pathways represent a third important disease component.

Microglia play a pivotal role in this immune response and are activated by binding of aggregated proteins or aberrant nucleic acids to pattern recognition receptors. This immune activation leads to the release of inflammatory mediators, but also distracts microglia cells from their physiological functions and tasks. Within several immune pathways, sustained NLRP3 inflammasome activation causes cognitive dysfunction and neurodegeneration but also a hyperinflammatory microglial cell death commonly denoted as pyroptosis which leads to the release of activated inflammasome complexes called ASC specks. The latter contributes to seeding of pathology by enhancing the propensity of beta-amyloid peptides to aggregate. This mechanism may account for the spread of pathology within a brain region, but also from one brain area to another. Increased microglial cell death in turn will cause proliferation of microglial cells generating several subpopulations over time, which show functional and transcriptional changes compared to non-proliferating cells. Thus, while protective in early stages, the ongoing and chronic microglial activation actively drives Alzheimer disease phenotypes and pathology, offering new and yet unused opportunities for therapeutic interventions.

Michela Matteoli

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When Microglia Fuel the Developing Brain: Role of Trem2 in Neuronal Bioenergetics

Microglial Trem2 is essential for proper brain development, yet its contribution to neuronal maturation extends beyond synaptic remodeling. We have recently shown that Trem2, a lipid-sensing receptor, orchestrates microglial metabolic and signaling pathways that support neuronal energy homeostasis. Indeed, Trem2-deficient microglia fail to sustain neuronal oxidative metabolism and mitochondrial function, leading to region-specific impairments in bioenergetic capacity. Consequently, neurons exhibit decreased synaptic activity and structural maturation. Our findings reveal that Trem2-dependent signaling maintains the metabolic dialogue between microglia and neurons, ensuring proper circuit refinement and functional development of the hippocampus. This talk will outline the molecular and cellular mechanisms through which Trem2 orchestrates microglia–neuron metabolic interactions.

David Sancho

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A type-I interferon-mitochondrial axis regulates efferocytosis and Interferon Stimulated Gene induction in macrophages

Macrophage metabolism is intricately linked to cellular function. Contrasting with Toll-like receptor (TLR) stimulation, cytosolic nucleic acid sensing induced a decrease in mitochondrial membrane potential (MMP) and maintenance of mitochondrial respiration. Interferon-α/ß (IFN-I) receptor (IFNAR) signaling was necessary and sufficient for this metabolic response. IFNAR signaling induced interferon-stimulated gene 15 (ISG15) expression and ISGylation of mitochondrial proteins, including subunits of mitochondrial complex V, increasing ATP production and decreasing MMP, thus enhancing macrophage efferocytic capacity. Moreover, the IFNAR-ISG15-mediated drop in MMP activated the mitochondrial protease OMA1, inducing mitochondrial fission and decreasing endoplasmic reticulum-mitochondria communication, thus dampening IFN-stimulated gene (ISG) induction. Loss of ISG15 or OMA1 enhanced histone acetylation and ISG induction upon IFN-I stimulation, dependent on mitochondrial calcium uptake. This increase in ISG induction provided protection against acute viral infections. These data indicate that IFNAR-ISG15 signaling boosts efferocytosis while limiting ISG induction, promoting the resolution of inflammation.

Fabrizia Bonacina

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Srebp1c modulates Treg immunobiology through a phospholipid-dependent adenosine pathway

Aim: Regulatory T cells (Tregs) maintain immune tolerance through mechanisms tightly coupled to cellular metabolism. We aimed at investigating the role of SREBP1c in Treg immunometabolism, given its key role in intracellular lipid homeostasis, that contributes to tolerogenic phenotype.

Material and Methods: A detailed immunophenotyping through flow cytometry and metabolic profiling (metabolomics and seahorse analysis) of isolated Tregulatory (CD4+CD25+,nTreg) and in vitro induced Treg (iTreg) cells were performed together with in vitro and in vivo assays of Treg function from SREBP1c KO and WT littermates. RNAseq and lipidomics was performed on iTreg.

Results: Tregs from Srebp1c-deficient mice displayed impaired suppressive function, reduced frequencies in circulation and lymphoid tissues, and diminished expression of functional markers. These defects stemmed from intrinsic metabolic rewiring rather than systemic alterations, as both ex vivo Tregs ($CD4^+CD25hiFoxP3^+$) and in vitro-derived iTregs lacking Srebp1c were shifted toward glycolysis. Integrated transcriptomic and lipidomic analyses revealed that Srebp1c-deficient Tregs exhibited defective phospholipid remodeling, with an accumulation of lysophosphatidylcholines over phosphatidylcholines due to enhanced phospholipase A2 activity and disruption of the Lands cycle. Altered membrane composition impaired adenosine-mediated immunosuppression by reducing CD73 expression and extracellular adenosine generation. Accordingly, pharmacological inhibition of phospholipase A2 restored adenosine signaling and Treg suppressive capacity.

Conclusion: by preserving phospholipid homeostasis, SREBP1c functions as an immunometabolic checkpoint that links lipid metabolism to adenosine-dependent Treg suppression.

Tân Khoa Lam

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Metabolic Rewiring Drives the Emergence of Protective Long-Lived Neutrophils in Rheumatoid Arthritis

Neutrophils are the most abundant circulating leukocytes and key effectors of tissue damage in rheumatoid arthritis (RA). While they accumulate in inflamed synovium and promote joint destruction, a distinct subset with protective features has been identified in the synovial fluid of patients with RA. These cells resemble human neutrophils reprogrammed in vitro with RAassociated cytokines, i.e., TNF, GM-CSF, and IL-4, and are termed long-lived (LL) neutrophils. Transcriptomic profiling suggested that LL reprogramming reshapes cellular metabolism and inflammatory functions.

To define their metabolic phenotype, peripheral blood neutrophils from healthy donors were cultured for 48 h in the presence or absence of TNF, GM-CSF, and IL-4. Real-time analysis of energy metabolism using a Seahorse revealed that LL neutrophils exhibit enhanced mitochondrial respiration, sustaining ATP production by preferentially relying on pyruvate. Consistent with this oxidative shift, reprogrammed neutrophils displayed a fused mitochondrial network, challenging the traditional view of neutrophils as predominantly glycolytic cells. Functionally, LL neutrophils exhibited reduced reactive oxygen species production and neutrophil extracellular trap formation, while significantly increasing secretion of elafin and secretory leukocyte protease inhibitor (SLPI), two endogenous protease inhibitors with tissueprotective properties, relative to freshly isolated or control-cultured cells.

Together, these findings identify metabolic rewiring by inflammatory cytokines as a central mechanism underlying the emergence of a new immunoregulatory neutrophil state. Ex vivo induction of LL neutrophils may represent a novel therapeutic strategy to modulate neutrophildriven inflammation in RA and in other chronic inflammatory diseases in which neutrophils are greatly implicated.

Abhijeet Kulkarni

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Impaired L-phenylalanine metabolism facilitates pathogenic Th2 inflammation in severe allergy

T cell metabolic programming shapes cell fate and function. Efficient metabolism is crucial for activation, proliferation, effector activity, and memory formation. However, metabolic regulation of CD4+ T cells and their subsets in allergic disease remains incompletely understood. Hence, we first performed metabolomic profiling of circulating memory CD4+ T effector (Teff) and regulatory (Treg) cells from healthy individuals, revealing enrichment of amino acid pathways, particularly L-phenylalanine (Phe) metabolism. We therefore examined effect of Phe on CD4+ T cell energy regulation and observed that Phe increased glycolysis in memory CD4+ T cells while limiting OXPHOS. It also inhibited their proliferation through an IL4I1-dependent mechanism, confirmed using siRNA-based knockdown techniques. Using SCENITH, we found Phe selectively enhanced Th2 cell glycolytic capacity while reducing mitochondrial dependence among helper T cell subsets. In vitro–differentiated Th2 cells exposed to Phe displayed reduced proliferation, STAT6 phosphorylation, and expression of key type 2 transcription factors and cytokines, including mTOR, BACH2, BATF, IL-4, IL-5, and IL-13. Furthermore, Phe reduced CD161 expression, a documented marker of pathogenic Th2a cells in allergy. Metabolomic and ex vivo analyses of allergic patients showed reduced intracellular Phe in circulating memory CD4+ Teff cells in a subset of severe cases and elevated serum Phe levels. Finally, analysis of allergic clinical cohorts revealed impaired Phe metabolism and expression of LAT1, an important Phe transporter, negatively correlated with serum Phe only in patients with allergy. Altogether, these findings identify Phe as a regulator of Th2 metabolism and this mechanism appears impaired in severe allergic disease.

Julia Jellusova

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GSK3 as a central Regulator of lipid metabolism and survival in normal and malignant B cells

Glycogen synthase kinase (GSK3) is an ubiquitously expressed kinase targeting many pro-survival and pro-proliferation factors such as cMyc or β-catenin for degradation. GSK3 inhibition is known to drive proliferation or even malignant transformation in various cell types. Surprisingly, we have found that while GSK3 inhibition boosts proliferation of mature activated B cells, the survival of B cell precursors and various B cell-derived malignant cells is reduced. We demonstrated that cell death is mediated by β-catenin accumulation in this context, which unexpectedly reduces cMyc expression. In addition to regulating cMyc expression we have found GSK3 inhibition to dysregulate various aspects of B cell metabolism including mitochondrial activity, lipid storage and redox balance. In summary, these findings demonstrate that the GSK3/β-catenin signaling pathway plays a fundamentally different role in B cells than in many other cell types. Several GSK3 inhibitors have been shown to be safe in clinical settings and, based on our findings, may warrant consideration for the treatment of B-cell malignancies.

Claus Desler Madsen

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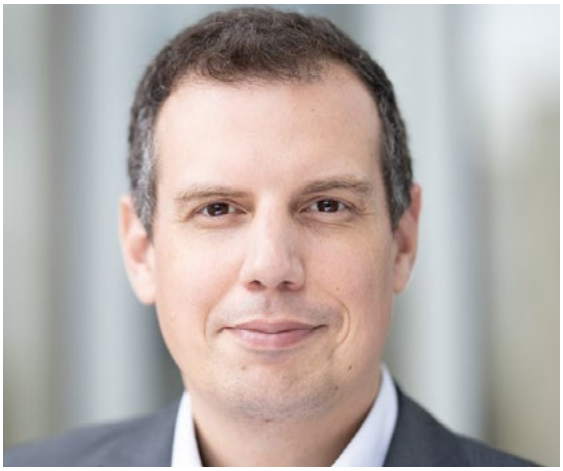
Mitochondrial regulation in the tumor microenvironment

Mitochondria are emerging as central signal-integrating hubs in the tumor microenvironment (TME), shaping both malignant cell fitness and anti-tumor immunity. Our work links mitochondrial dysfunction to innate immune activation programs, including cGAS–STING–associated inflammatory signaling in macrophage-like cells. In parallel, we explore how mitochondrial control of nucleotide metabolism constrains immune cell proliferation and function under TME-like stress.

A recurrent theme is that metabolic rewiring influences immune recognition pathways, including regulation of stress-induced ligands such as MICA with relevance for NK/T cell surveillance. By integrating functional bioenergetics with immunophenotyping and molecular profiling, we map how mitochondrial state partitions immune cell fates within inflammatory microenvironments.

These insights support a model where mitochondrial stress responses act upstream of both immune suppression and immune activation, depending on cellular context and timing.



Philipp A. Lang

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Improved T cell metabolism can boost anti-tumor effector function

The tumor microenvironment is frequently hypoxic and characterized by a scarcity of nutritional resources including a shortage of glucose. As effector T cells have high energy demands, tumor metabolism can contribute to T cell dysfunction and exhaustion. These metabolic restrains can impact the efficacy of T cell based therapy regimens of solid tumors including transfer of tumor infiltrating lymphocytes or CAR-T cells. Furthermore, infiltration of such T cells might be curbed by the scarce nutritional tumor microenvironment.

Here we show, that a metabolic improvement through expression of myoglobin in T cells can boost their mitochondrial and glycolytic metabolic functions. These metabolic enhanced T cells exhibited increased presence of metabolites including enhanced ATP levels in vitro. Moreover, T cells exhibited increased effector differentiation which was associated with more effector functions against tumor cells in tissue culture settings. Moreover, transfer of metabolic improved T cells into tumor bearing mice exhibited reduced tumor growth in murine model systems. Furthermore, we discuss whether other tumor therapies might influence metabolic functions of tumor specific T cells and accordingly affect their anti-tumoral efficacy.

In conclusion, we show that metabolic enhancement in T cells can increase their metabolism, infiltration into the tumor tissue, and effector function against cancer cells.

Martina Erbi

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Development of a click-chemistry based glucose probe: single-cell in situ tracking of glucose uptake in the tumor microenvironment

Glucose uptake is a central regulator of immune cell function, which is dynamically regulated in pathophysiological settings, based on nutrient availability and cellular metabolic demands. Thus far, glucose uptake measurements at single-cell level have relied on the use of bulky fluorescently-labelled glucose. However, recent evidence show this tool does not faithfully mirror native glucose uptake. Here, we developed a novel click-chemistry compatible glucose probe whose uptake can be visualized by spectral flow cytometry at single-cell resolution by addition of an azide-fluorophore through a copper-catalyzed click reaction. This compound, identified by screening a library of alkyne-glucose analogues, showed competition with native and radiolabeled glucose and its uptake was inhibited by a glucose transporter inhibitor in primary murine splenocytes, demonstrating it largely behaves as its native counterpart. We then applied this novel probe in vivo, to track glucose uptake in situ in the tumor microenvironment using a murine model of colorectal cancer. We found that macrophages showed the highest capacity for glucose uptake, followed by T cells and tumor cells, and this was further boosted following vaccination. Moreover, within the CD8+ T cell compartment we identified populations with different glucose uptake profiles that we could not discriminate by surface marker expression, highlighting the potential of this glucose uptake assay to discern metabolically distinct T cells that would otherwise be immune-phenotypically identical. Overall, we developed a glucose probe that can faithfully track glucose uptake and can be used to assess glucose uptake profiles at a single-cell level, both in vitro and in vivo.

Jose Aramburu

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Poorly perfused tumor regions harbor T cells with a glucose-dependent effector phenotype

Effector T lymphocytes are avid nutrient consumers, but can function in nutrient-poor tumor microenvironments. Availability of key nutrients such as glucose inside the tumor is not homogeneous, and how tumor-infiltrating T lymphocytes (TILs) can maintain functionality in regions with poor blood perfusion is not well known. Here we show that in vitro-stimulated TILs could induce substantial production of hallmark glucose-dependent cytokines under glucose concentrations 20 times lower than in blood; that effector TILs from tumor regions with poor access to blood show comparable capacity for inducing IFNγ and granzyme B to TILs with fuller accessibility; exhibit an enhanced type I IFN response signature characteristic of bystander resident memory cells; and unexpectedly, have reduced expression of immune checkpoint and Treg-associated markers. TILs with poor blood accessibility also have reduced biosynthetic activity than highly blood-accessible TILs, yet both compartments depend fundamentally on glucose for ATP production. Thus, effector T lymphocytes in poorly perfused tumor regions can maintain specific glucose-dependent responses, and might be partially protected from inhibitory and exhausting pressure from the tumor microenvironment.

Stephanie Sendker

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Dual Function Immune Metabolic Engager (DIME12) Potentiates Anti-tumor Immunity by Rewiring the Tumor Metabolic Environment

We developed a first-in-class Dual Function Immune Metabolic Engager expressing IL-12 (DIME12), based on a human gut-derived non-pathogenic E. coli (K12 DH5a) engineered to surface-display IL-12 and adenosine deaminase (ADA). This system converts immunosuppressive adenosine into inosine while enhancing immune cell cytotoxicity, enabling localized immune activation within the tumor microenvironment (TME) with reduced systemic toxicity.

Engineered bacteria expressing IL-12, ADA, or IL-12-ADA were validated by flow cytometry, HEK-Blue assay, LC-MS, and enzymatic assays. Primary human NK and T cells were exposed to adenosine, inosine, or engineered bacteria, followed by phenotypic, metabolic (Seahorse), transcriptional (RNA-seq), and cytotoxicity analyses. In-vivo efficacy, safety, and immune responses were evaluated in B16-F10 melanoma, WEHI-3B leukemia, and ASPCl pancreatic cancer xenograft models.

E. Coli displaying IL-12 enhanced NK and T cell activation but lead to increased adenosine level in-vitro and susceptibility to adenosine via upregulation ofCD38, ENTPD1/CD39, ENPPJ/CD203a, andADORA2BIA2BR upregulation and ADA, PNP downregulation. ADA-displaying bacteria restore immune function under adenosine, boosting activation and tumor cytotoxicity. While bacteria scaffold shifted NK metabolism toward glycolysis, ADA restored oxidative phosphorylation, indicating metabolic flexibility. Co-expression ofIL-12 and ADA preserved NK effector function and enhanced tumor lysis.

In-vivo, DIME12 was well tolerated and demonstrated robust tumor control with durable immunity upon rechallenge. In humanized ASPCl models, combination with mesothelin CAR-NK cells significantly improved tumor control and survival.

Together, DIME12 remodels the metabolic and immune landscape of tumors, driving durable antitumor responses and supporting clinical translation.

Johan Garaude

ImmunoConcEpT, INSERM-CNRS,
University of Bordeaux, France

Microbial viability drives immunometabolic responses of macrophages

During bacterial infection, macrophages successively encounter and engulf live and killed microbes, and trigger appropriated host responses to eradicate the threat and restore tissue homeostasis. Despite the critical role of phagocytosis during this event, the fate of phagocytosed microbial cargo and its impact on host cell is poorly understood. Our investigation reveals that phagocytosis of bacteria provides bioenergetic and biosynthetic precursors that are differentially utilized by macrophages depending on microbial viability. This highlights the capacity of phagocytic cells to adjust their cellular metabolism to the 'fueling' capacity of the ingested cargos and how, together with the sensing of microbial viability, it regulates immunometabolic adaptations to adjust host response intensity to the course of microbial infection.

Felix Wensveen

Faculty of Medicine, University of Rijeka, Croatia

War-time metabolism: How the immune system rewires the body to fight infection

When we get sick, we feel miserable. We get a temperature, we feel weak and we just want to lay in bed. We experience this as a pathology. After all, how can feeling bad be good? But in fact, the changes to our physiological state in response to infection are the result of a carefully regulated set of metabolic changes mediated by the immune system. Its purpose is to generate a metabolic environment in which the body is optimally able to fight infection. Infection-induced metabolic changes, also known as sickness metabolism, depend on tissue-specific interactions between the immune system and organs involved in regulation of systemic homeostasis. Alterations to homeostatic set points leads to altered production and uptake of nutrients in circulation, which modifies the metabolic rate of key organs. This is what we experience as being sick. Surprisingly, whereas we are all familiar with being sick, the underlying mechanisms are only now starting to be understood. In this presentation I will provide some new insights into how the immune system changes our metabolism as it goes to war against infections. And I will explain how this system can derail into chronic metabolic changes, i.e. metabolic disease, if it battles against perceived threats, for example metabolically stressed tissues.

Marcela Hortová Kohoutková

International Clinical Research Center (ICRC),
St. Anne's University Hospital Brno, Czech Republic

Sepsis-induced long-term functional and metabolic rewiring in innate immunity

Sepsis is a heterogeneous syndrome with dynamic progression, yearly affecting more than 160 million people worldwide. Survivors frequently suffer from immunosuppression, which can persist many months after sepsis onset and impede the patient's full recovery.

The exposure of innate immune cells to environmental stimuli such as invading pathogens or pathogen-originated ligands is followed by their activation leading to a metabolic switch associated with phenotypic changes. The metabolic switch is necessary to obtain sufficient energy and intermediate metabolites to execute the defending processes against invading stimuli. Detailed characterization of immune cells' immunometabolic status and phenotypic alterations together with patient clinical status represent possible approach how to depict the sepsis dynamics, especially with emphasis to the long-term consequences of sepsis.

We found changes in neutrophils and monocytic subsets frequency functional status represented by altered expression of HLA-DR, CD86, or CD36 together with specific cytokine pattern. We also revealed the metabolic alterations across all innate immune cells. All markers were correlated to the severity of sepsis (SOFA or Phoenix score) to obtain comprehensive information associated also with the clinical picture of patients. We also evaluated the decline of quality of life of these patients using self-reporting SF-36 questionnaire. By further integration of all obtained data by machine learning we will evaluate the potential risk of severe complications' development and design timely treatment strategy to facilitate the patient full recovery.

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Maxim Nosenko

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Methionine availability regulates immunometabolic response of NK cells upon bacterial infection

NK cells contribute to bacterial infection control via production of IFNγ as well as via direct lysis of bacterial and infected host cells with cytotoxic molecules. While metabolic requirements of NK cell activation are well-studied in the context of cancer and viral infections, less is known about bacterial challenge.

Here, using mouse models of LPS-induced inflammation and E. coli infection we found that NK cells undergo dynamic immunometabolic reprogramming with early (IFNγ, Granzyme B) and late (Perforin) waves of activation, accompanied by mTORC1 signalling and upregulation of mitochondrial metabolism as well as amino acid uptake via Slc7a5 and Slc1a5 transporters. Genetic inactivation of Slc7a5 specifically in NK cells resulted in blunted functional and metabolic response with diminished capacity to control bacterial dissemination. In contrast, inactivation of Slc1a5 had only minor outcome on NK cell activation, potentially highlighting compensatory mechanisms.

Metabolomics analysis revealed early and substantial drop in systemic availability of methionine, Slc7a5 cargo, upon inflammation. Depleting methionine resulted in decreased production of Granzyme B and Perforin by cytokine-stimulated NK cells in vitro. Accordingly, supplementing diseased mice with methionine increased production of cytotoxic molecules by peritoneal NK cells. In contrast, neither depletion, nor supplementation of methionine affected NK cell IFNγ production. Mitochondrial metabolism and mTORC1 signalling were also not impacted by methionine, however, we found a decreased abundance of cellular ROS when methionine was depleted.

Altogether, these findings indicate a key role of methionine for regulation of cytotoxic response of NK cells in the context of bacterial infection and sepsis.

Hatem Abouguendia

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Metabolic rewiring drives the feed-forward loop of inflammation and HCMV reactivation

Human cytomegalovirus (HCMV) infects 66-90% of the population and establishes latency in the hematopoietic progenitor cells and myeloid cells. Inflammatory signals reactivate HCMV which further exacerbates inflammation, and the virus is often found active in inflammatory conditions such as cardiovascular diseases, rheumatoid arthritis, and SLE.

We hypothesize that the feedforward loop of inflammation and HCMV reactivation is mediated by metabolic rewiring of myeloid cells. We aim to characterize the metabolic profile and associated molecular mechanisms. A model of latency-reactivation in human monocyte-derived macrophages (HMDMs) was used. Oxygen consumption rate (OCR) and extracellular acidification rate (ECAR) were assayed by Seahorse and qPCR was used to measure inflammatory cytokine levels such as IL-1β and TNFα. Latency was validated by the absence of viral output and immediate early (IE) transcripts while maintaining the viral genome.

We found that HCMV upregulates basal ECAR during latency, which primes the macrophages for enhanced inflammation. Upon activating macrophages using the pro-inflammatory molecules IFNγ and LPS or IL-6, HCMV was reactivated, as demonstrated by the expression of IE86. Viral reactivation prevented the upregulation of OCR observed in the activated HMDMs. HCMV infection boosted the production of pro-inflammatory cytokines. This was in part mediated by the mitochondrial phosphatase PTPMT1 that inactivates complex II (SDH), which controls the mitochondrial respiration and hence an inflammatory response. We found that PTPMT1 is upregulated in HCMV-infected macrophages, leading to inhibition of SDH activity to prevent upregulation of OCR. We also found that HCMV further enhances the expression of itaconate-producing gene (ACOD1), which causes further inhibition in SDH activity.

Collectively, our data shows that HCMV can enhance inflammation by affecting cellular metabolism. This opens the doors to explore whether antiviral drugs and metabolic interventions can be used to control HCMV infection as well as inflammatory conditions.

Roland Lang

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Getting itaconate to where it matters in antibacterial defense

Infection with the intracellular bacterium *Coxiella burnetii* is controlled by Myd88-dependent macrophage activation in mice and SNPs in human Myd88 are associated with increased risk of chronic Q fever. Higher bacterial burden in Myd88-deficient mice correlated with impaired expression of ACOD1, that was required in macrophages to inhibit replication of C. *burnetii* via production of itaconate. *Acod1*$^{-/-}$ mice developed higher C. *burnetii* burden in the lung and liver. Supplementation of exogenous itaconate to *Acod1*$^{-/-}$ macrophages and mice restored control of C. *burnetii* replication *in vitro* and after intraperitoneal infection. Itaconate and its isomers mesaconate and citraconate inhibit replication of C. *burnetii* in axenic culture at concentrations considerably lower compared to several other bacterial species. Remarkably, only itaconate, but not its isomers, was able to inhibit C. *burnetii* growth in Acod1$^{-/-}$ macrophages. Analysis of intra-macrophage levels of the isomers by MS revealed that uptake of exogenous itaconate and mesaconate, but not of citraconate, was increased in infected macrophages in a Myd88-dependent manner. Transcriptome data mining identified several members of the solute carrier (SLC) gene family to be strongly induced by C. *burnetii* infection, including Slc13a3, a transporter for the dicarboxylates succinate and citrate. We show that Slc13a3 transports itaconate and mesaconate, but not citraconate, suggesting that Myd88-dependent upregulation of Slc13a3 underlies the efficient uptake of itaconate by activated macrophages that enables complementation of *Acod1*$^{-/-}$ macrophages for control of intracellular bacterial replication.

Carole Linster

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Damage is Inevitable and Repair is Essential – Also in Metabolism

Although neglected for a long time, enzymatic side activities and spontaneous chemical reactions that generate abnormal or potentially toxic metabolites are an integral part of cellular metabolism. Metabolite repair enzymes keep these inevitable side-products in check, restoring them to useful forms and maintaining metabolic homeostasis. Over the past 15 years, a combination of hypothesis-driven biochemistry and metabolomics has helped uncover many such repair systems.

For example, hydrated and redox-inactive forms of the NADH and NADPH cofactors (NADHX and NADPHX) are continuously generated in cells. We discovered a highly conserved repair system, composed of the dehydratase NAXD and the epimerase NAXE, which converts NAD(P)HX back to the normal cofactors. Loss of NAXD or NAXE leads to NADHX accumulation and perturbations in central metabolism, notably in de novo serine synthesis. Clinically, deficiency in either of these enzymes causes a fatal pediatric neurometabolic disorder, highlighting the physiological importance of metabolite repair and the potential for translation from molecular insight to therapy. In zebrafish models, immune-related pathways are among the most perturbed signatures based on transcriptomics analyses, motivating current work to test whether glial cells show specific vulnerability to impaired NAD(P)HX repair.

A further illustration is a newly discovered function of citrate lyase beta-like protein (CLYBL), a mammalian enzyme linked to itaconate metabolism and vitamin B12 homeostasis. We found that CLYBL hydrolyzes malyl-CoA, a previously unrecognized side-product of central metabolism that accumulates in CLYBL-deficient cells and inhibits the B12-dependent enzyme methylmalonyl-CoA mutase, contributing to reduced cellular cobalamin. Unlike NAXD/NAXE deficiency, CLYBL loss-of-function is relatively common, illustrating how metabolite repair can shape both rare and more widespread aspects of human metabolism.

Together, these studies highlight metabolite repair as a pervasive and functionally critical layer of metabolism, and show how mechanistic insights into metabolic chemistry can translate into new concepts for disease mechanisms and therapeutic strategies.

Rafael Argüello

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Epic-SCENITH reveals metabolic–epigenetic programs induced by glycolytic stress

Glucose limitation is a recurrent feature of inflamed and tumor microenvironments, where immune and cancer cells must adapt to nutrient competition, oxidative stress, and endoplasmic reticulum stress. While the hypoxia response has been extensively characterized, the molecular architecture of a dedicated glycolytic stress response remains poorly defined. In particular, it is still unclear how early metabolic constraints are converted into durable transcriptional and epigenetic programs that may influence immune dysfunction, cancer cell persistence, and therapy resistance.

To address this question, we developed Epic-SCENITH, an extension of SCENITH™ that links single-cell functional metabolic profiling to downstream chromatin analysis. Epic-SCENITH enables the identification and physical isolation of cells according to their metabolic functional responses, followed by low-input chromatin profiling of regulatory histone marks such as H3K27ac, H3K27me3, and H3K4me3 in fixed and permeabilized cells. Using a model of glycolytic stress in monocytic leukemia cells, we show that a homogeneous metabolic challenge generates heterogeneous functional states, with subsets maintaining a glycolytic-like profile while others acquire increased mitochondrial dependence. These divergent states are associated with distinct chromatin and transcriptional programs.

Integrated epigenomic and transcriptomic analyses revealed that glycolytic stress induces broad remodeling of regulatory regions, including activation of interferon-stimulated genes, unfolded protein response pathways, oxidative stress programs, and lipid metabolism regulators. Motif enrichment analysis of H3K27ac-enriched regions nominated transcription factors linked to stress adaptation and metabolic rewiring, including NRF2, IRF family members, ATF4/XBP1, RXRA/RARA, and SREBP-related programs. These data support a model in which glycolytic stress does not simply impose an energetic deficit but triggers a coordinated adaptive response involving redox control, ER stress, translation regulation, lipid metabolism, and chromatin remodeling.

Mechanistically, our results suggest that cells adapting to glycolytic stress may increase reliance on fatty acid and amino acid oxidation and engage a non-canonical TCA-derived route involving citrate export, ACLY-dependent acetyl-CoA production, and ME1-linked NADPH generation. This pathway may simultaneously buffer oxidative stress, limit integrated stress response–driven translation arrest, and provide acetyl-CoA for chromatin acetylation, thereby connecting metabolic adaptation with long-lasting regulatory states.

Together, these findings establish Epic-SCENITH as a powerful platform to dissect how metabolic heterogeneity is translated into epigenetic heterogeneity at single-cell resolution. Beyond methodological innovation, this work identifies candidate regulators of the glycolytic stress response and provides a conceptual framework to understand how nutrient constraints in tumors and inflamed tissues may shape immune dysfunction, cancer cell persistence, and therapeutic resistance.

Thekla Cordes

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Tracing the journey of itaconate metabolism

The immunometabolite itaconate has attracted high interest as a regulator of cellular metabolism and immune function. It inhibits succinate dehydrogenase (SDH) thereby reshaping mitochondrial respiration and tricarboxylic acid (TCA) cycle metabolism as well as associated pathways. However, the metabolic dynamics and fate of itaconate remain poorly understood. Here, I will present our work combining mass spectrometry approaches, pharmacokinetic profiling, and 13C isotope tracing to define itaconate metabolism and degradation pathways. We demonstrate that itaconate is rapidly cleared from plasma and undergoes further metabolic processing. Succinate levels strongly correlate with itaconate abundance indicating a dynamic and functional impact on SDH activity. Moreover, we identify novel metabolites derived from itaconate and reveal an additional metabolic branch for itaconate degradation. Together, these findings provide new insight into the mode of action, turnover, and downstream metabolism of itaconate refining current concepts of immunometabolic regulation.

Stefano Angiari

Otto Loewi Research Center, Division of Immunology, Medical University of Graz, Austria

Coenzyme A fueling with pantethine limits autoreactive T cell pathogenicity in experimental neuroinflammation

Targeting T cell metabolism is emerging as a promising strategy for the treatment of autoimmunity. Identification of metabolic pathways controlling T cell pathogenicity in autoimmune diseases has thus high therapeutic potential.

In this study, we evaluated the metabolic profile of myelin-specific murine encephalitogenic T cells, to discover novel targets in autoimmune neuroinflammation. By performing unbiased metabolomics analysis, we detected a potential break in the coenzyme A (CoA) synthesis pathway in activelyproliferating encephalitogenic T cells, compared to resting T cells. CoA fueling with the CoA precursor pantethine affected essential immune-related processes of autoreactive T cells, such as antigen-specific proliferation, cytokine production, and integrin-mediated cell adhesion, both in vitro and in vivo. Mechanistically, pantethine exerted its immunomodulatory effects in encephalitogenic T cells by linking metabolic reprogramming to alteration of intracellular signaling pathways. We then evaluated the impact of pantethine treatment on the development of experimental autoimmune encephalomyelitis (EAE), a mouse model of multiple sclerosis (MS). Our data show that pre-clinical treatment with pantethine inhibited EAE development in two different mouse strains. Importantly, pantethine also significantly ameliorated the disease course when administered after disease onset in a therapeutic setting. Finally, pantethine limited pro-inflammatory cytokine production by human T helper 1 (Th1) and Th17 cells in vitro, as well as by T cells from MS patients, confirming its translational potential.

In conclusion, we demonstrated that CoA fueling with pantethine in pro-inflammatory and autoreactive T cells may represent a novel therapeutic approach for the treatment of autoimmune neuroinflammation.

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Metabolic reprogramming of interleukin-17-producing γδ T cells promotes ACC1-mediated de novo lipogenesis under psoriatic conditions

Metabolic reprogramming determines γδ T cell fate during thymic development. However, the metabolic requirements of IL-17A-producing γδ T cells (γδT17 cells) under psoriatic conditions remain unclear. Combining high-throughput techniques, including RNA sequencing, SCENITH, proteomics, and stable isotope tracing, we demonstrated that psoriatic inflammation caused γδT17 cells to switch toward aerobic glycolysis, linking carbohydrate metabolism and fatty acid synthesis. Accordingly, we used a pharmacological Acetyl-CoA carboxylase (ACC) inhibitor, Soraphen A, which blocks fatty acid synthesis in γδT17 cells, reducing their intracellular lipid stores and ability to produce IL-17A under psoriatic conditions in vitro. We further pinpointed the pathogenic role of ACC1 in γδT17 cells in vivo by genetic ablation, ameliorating inflammation in a psoriatic mouse model. Furthermore, ACC inhibition limited human IL-17A-producing γδT17 cells. Targeting ACC1 to attenuate pathogenic T cells, including conventional T cells and γδT17 cells, has important implications for the management of psoriasis. Interestingly, γδT17 cells did not burn lipid droplets as a primary energy source via fatty acid oxidation under psoriatic conditions, but rather increased lipid droplet accumulation. Therefore, it is unclear why γδT17 cells accumulate lipid droplets under both homeostatic and inflammatory conditions. Therefore, we aim to investigate the roles of lipid droplets in γδT17 cells by super-resolution microscopy and innovative microenvironment-mapping methods to provide spatial resolution. Our study reveals the underappreciated function of lipid droplets in γδT17 cells, which could open new avenues for regulating inflammation.

Marina Diotallevi

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A new NO-independent immune regulatory role for iNOS via protein-protein interaction with IRG1

Itaconate, a TCA cycle derived metabolite produced by Immunoresponsive Gene 1 (IRG1, Acod1), is one of the most abundant metabolites in activated immune cells and has pivotal roles in the inflammatory response, metabolic regulation, and redox signalling. We have previously discovered that inflamed macrophages lacking inducible Nitric Oxide Synthase (iNOS) (an inducible enzyme generating high levels of nitric oxide (NO) during inflammation) or its cofactor tetrahydrobiopterin (BH4), produced markedly increased amounts of itaconate in comparison with wild-type activated macrophages, by a mechanism independent of IRG1 expression.

To further understand the role of iNOS in mediating itaconate production, and unravel the subsequent immuno-regulatory functions of iNOS, we used bone marrow derived macrophages cultured from WT, iNOS KO and BH4 KO mice. Following activation with LPS and interferon γ ($M^{LPS/IFN\gamma}$), we uncovered a strong correlation between the presence of iNOS / NO and a striking decrease in the production of itaconate over time. Using experimental studies in cells, surface plasmon resonance, computational predictions, and molecular dynamics simulations of iNOS and IRG1 molecular interactions, we report here a dynamic inhibition of IRG1 by protein-protein interaction between iNOS and IRG1 that is dependent upon specific iNOS conformations, but not on NO generation.

In conclusion, we have revealed a novel fundamental role for iNOS, independent of its NO catalytic activity, in regulating the critical metabolite itaconate. This study places iNOS at the centre of regulating macrophage function and the response to injury, with iNOS effectively acting as a brake to control itaconate production and ultimately macrophage polarization.

Tjaša Frlic

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Metabolic reprogramming during ex vivo expansion promotes memory-enriched CAR T Cells

Background: Immunotherapies, such as CAR T therapy, have revolutionized cancer treatment; however, poor therapeutic outcomes have driven research into strategies for enhancing anti-tumor T cell responses. T cell functions are intrinsically linked to metabolic reprogramming, positioning metabolic modulation as a promising strategy to improve therapeutic efficacy. We screened metabolic compounds to identify those capable of promoting a memory-enriched phenotype with reduced terminal differentiation and improved metabolic fitness.

Methods: PBMCs were thawed, activated with anti-CD3 and anti-CD28 antibodies in the presence of IL-2, and cultured for an additional 10 days. Over the last 6 days, metabolic modulators have been added to the media. Bioenergetic profile (OCR/ECAR) was assessed using Seahorse XFe analysis. T cell differentiation and activation markers were quantified using flow cytometry, effector functions were evaluated by measuring IFN-γ, IL-2, and Granzyme B secretion, and RNA was isolated for transcriptomic sequencing.

Results: Selected metabolic modulators significantly increased the frequency of memory-associated T cell subsets and upregulated stem- and memory-related transcription factors. Treated cells exhibited reduced expression of activation markers and decreased levels of exhaustion-associated molecules, such as LAG3, TIGIT, and TIM3. Metabolic profiling confirmed bioenergetic remodeling consistent with functional reprogramming, while effector cytokine production was diminished or preserved. Transcriptomic analysis further revealed specific enrichment of genes associated with self-renewal and long-term persistence, alongside suppression of pathways linked to terminal effector differentiation and chronic activation.

Conclusions: Collectively, these results support metabolic reprogramming as a strategy to promote durable, memory-enriched CAR T cell products with reduced exhaustion and improved metabolic fitness.

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Jeffrey Rathmell

Virginia and D.K. Ludwig Chair for Cancer Research Professor

Chair, Ben May Department for Cancer Research

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Metabolic Stress and T Cell Dysfunction

The metabolism of T cells and other immune cells is dynamically regulated and influences biosynthesis, signaling, and cell fate. However, mitochondria in T cells from tumors or in chronic inflammatory settings become fragmented, hyperpolarized, and produce Reactive Oxygen Species (ROS). Further, these T cells had reduced metabolic flexibility, with limited spare capacity or ability to efficiently switch substrates. We are now working to understand the nature of this stress, the mitochondrial response, and how it may be overcome to improve immunotherapy. Temperature is a microenvironmental variable that changes with body location, fever, and inflammation. While heat is well-known to influence activities of enzymes and complex structures, the impact of locally increased temperatures on T cell metabolism and function are uncertain. We tested the effects of elevated temperatures found that T cells become more pro-inflammatory but begin to experience stress. Effector CD4 T cells of all subsets tested had initially increased proliferation and cytokine secretion. While Treg also had increased proliferation, they had reduced ability to suppress. Interestingly, Th1 cells selectively showed mitochondrial stress similar to T cells in tumors that Th17 and Treg did not experience. This stress ultimately led to cGAS/STING and p53 activation to enhance both inflammation and apoptosis. Mechanistically, our data point to mitochondrial electron transport complex 1 (ETC1) as sensitive to elevated temperatures and Th1 as selectively dependent on this complex. Together, these data show that physiologic heat is pro-inflammatory and that Th1 cells selectively develop mitochondrial stress with ETC1 as a potential thermosensitive modulator of mitochondrial metabolism. This heat sensitive ETC1 mitochondrial stress pathway may have broad implications in fever and inflamed tissues.

Elena Loche

Director of Flow cytometry
Abcam Limited, Cambridge Cambridge, UK

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Gideon Gießelmann

Luxembourg National Research Fund
FNR, Esch-sur-Alzette, Luxembourg

The Luxembourg Research Ecosystem and Funding Opportunities

The presentation outlines the Luxembourg research ecosystem and funding landscape, highlighting key programmes of the Luxembourg National Research Fund (FNR). It presents funding opportunities across career stages, including national and international schemes, alongside initiatives promoting research culture and international collaboration to strengthen Luxembourg's research system and impact in line with international best practice.

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