RTG 2408 | Maladaptive processes across physiological barriers in chronic diseases

Annual RETREAT 2026

JULY 08–10

USEFUL INFORMATION

VENUE & ACCOMMODATION

Biohotel WildLand

Dorfstraße 26

29323 Hornbostel/Aller

TIMES

Check-in:	Day 1 from 15:00
Check-out:	Day 3 by 10:00
Breakfast:	7:00–9:00

ROOMS

Breakfast, lunch:	Restaurant (A)
Dinner:	Rotes Segel (B)
Talks, meetings, coffee breaks:	Kathedrale (C)
Individual rooms:	Eulenhaus, Dicke Eiche, Speicher (D)

BUS TRANSPORT

Meeting point: July 08, 2026, 8:30-8:40. Parking lot behind House 33

https://maps.app.goo.gl/iPrmg7GFmp8q9s4v5

OTTO VON GUERICKE UNIVERSITÄT MAGDEBURG
MED FACULTY OF MEDICINE

Funded by
DFG Deutsche Forschungsgemeinschaft
German Research Foundation

PROGRAM

Day 1 | July 08, 2026

11:30–12:30	**Arrival & Welcome Lunch**
12:30–12:45	**Welcome**

Session 1 — Chairs: Sandra Freier, Jakob Wolf

12:45–13:30	*Biphasic NF-κB activity in the Helicobacter pylori infected gastric mucosa* P15-3 | Arun Kanthasamy
13:30–14:15	*The role of cold shock proteins in mitochondrial homeostasis and for tubular cell phenotype determination during cell stress* P8-2 | Sohail Ahmad
14:15–14:35	**Coffee Break**

Session 2 — Chairs: David Becker, Shivani Singh

14:35–15:20	*Deciphering extracellular Cold-Shock-Protein-RNA:IgG complex formation and elucidating cellular effects thereof* MD13 | Martin Hoppstock
15:20–16:05	*USP48-dependent regulation of NF-κB in the H. pylori infected gastric mucosa* P1-2 | Lorena Ferino
16:05–16:25	**Coffee Break**

Session 3 — Chairs: Kashod Aslam, Lukas Grein

16:25–17:10	*Long-term local and systemic effects of intestinal inflammation on the immune response to secondary challenges* P2-3 | Nina Lindemann
17:10–17:55	*Analyzing the impact of mesenchymal stromal cells as tolerance promoting barrier in the context of CAR T cell therapies* P13-3 | Lea Reemts
17:55–18:15	**Coffee Break**
18:15–19:30	**PI-Session & Round Tables**
19:30–20:30	**Dinner**
20:30	**Informal Discussion**

PROGRAM

Day 2 | July 09, 2026

Session 4	Chairs: Defne Hacimahmutoglu, Lea Reemts
9:00–9:45	*Investigating the influence of chronic intestinal inflammation on the antibacterial immune response in the lung* MD14 | David Becker
9:45–10:30	*Interaction of the airway epithelial barrier and T helper (Th) 2 cells during helminth infection of the lung* P12-3 | Sandra Freier
10:30–10:50	**Coffee Break**
Session 5	Chairs: Martin Hoppstock, Nina Lindemann
10:50–11:35	*Optimized models for studying intra- and intercellular crosstalk in biliary diseases* P11-3 | Shivani Singh
11:35–12:20	*Mast cell granules as regulators of LPS-induced macrophage tolerance* MD15 | Jakob Wolf
12:20–13:20	**Lunch Break**
13:20–13:50	**General Assembly**
13:50–14:50	**Troubleshooting & Problem Solving Discussions**
14:50–15:10	**Coffee Break**
Session 6	Chairs: Julius Rublack, Tanja Schickschneit
15:10–15:55	*Screening for systemic factors in biliary diseases* P16-3 | Nora Siegelt
15:55–16:40	*Elucidating Gene Regulatory Networks during Cell Cycle Entry* P1-4 | Lukas Grein
17:00–19:30	**Collaboration Walk**
19:30–20:30	**Dinner**
20:30	**Informal Discussion**

PROGRAM

Day 3 | July 10, 2026

9:00–9:30	**Students Meeting**
Session 7	Chairs: Sohail Ahmad, Lorena Ferino
9:30–10:15	*Investigating the role of the mast cell granule uptake in macrophage cell death and survival pathways* MD16 | Defne Hacimahmutoglu
10:15–11:00	*Role of eosinophil subpopulations in allergic asthma and other eosinophil driven diseases* AP12 | Julius Rublack
11:00–11:20	**Coffee Break**
Session 8	Chairs: Arun Kanthasamy, Nora Siegelt
11:20–12:05	*Bidirectional cross talk between Mast Cells and Endothelial Cells in homeostasis and inflammation* P4-3 | Tanja Schickschneit
12:05–12:50	*Immunoglobulin-Mediated Trafficking of Nucleic Acid-Protein Complexes in kidney diseases* P8-3 | Kashod Aslam
12:50–13:00	**Voting of Pitches**
13:00–13:15	**Concluding Remarks + Pitch Ranking**
13:15–14:00	**Farewell Lunch**

Biphasic NF-κB activity in the *Helicobacter pylori* infected gastric mucosa

Background

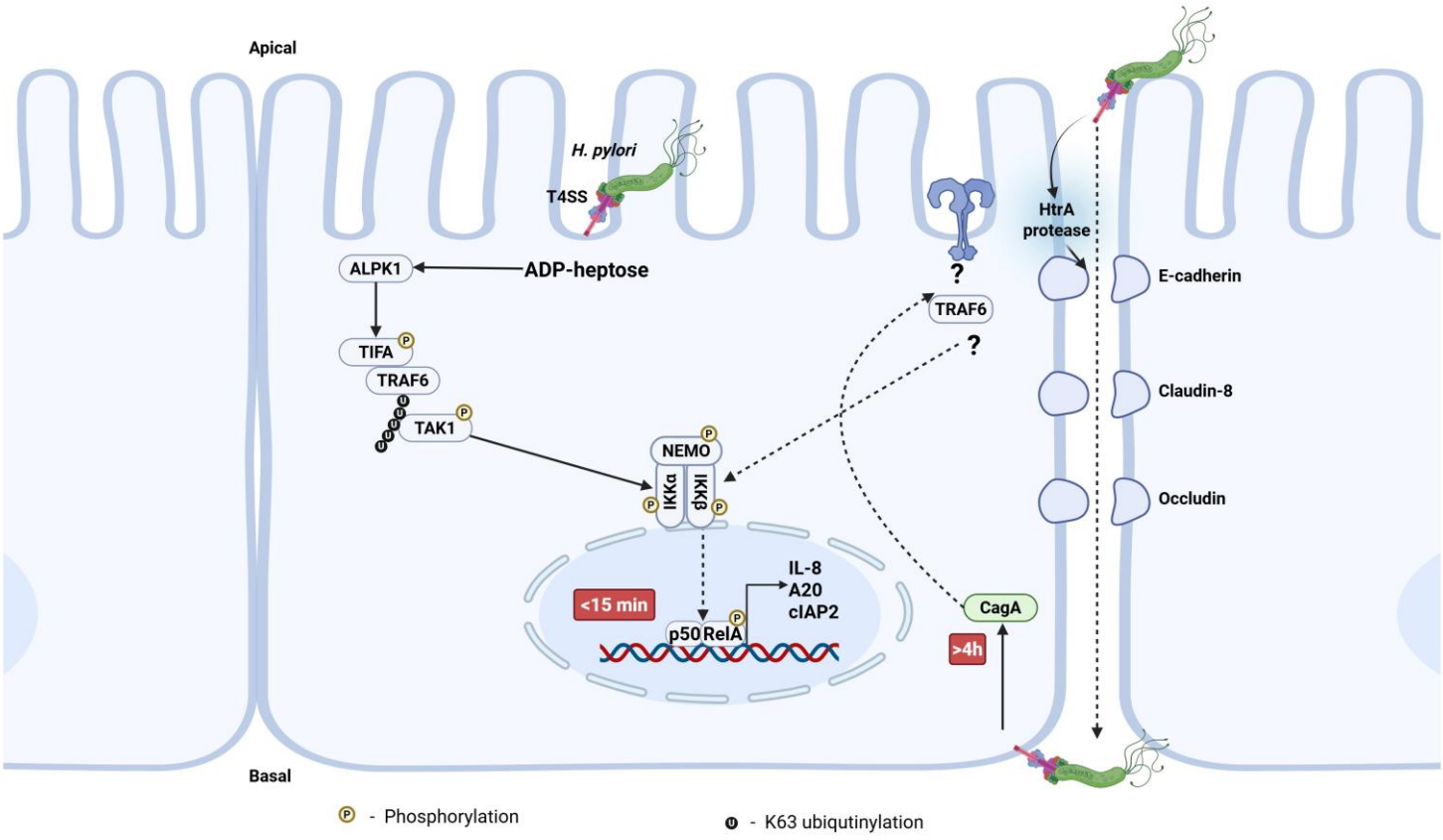
Helicobacter pylori is a Group 1 carcinogen and a known risk factor for several gastric diseases. NF-κB activation is induced in response to ADP-heptose upon *H. pylori* infection.

Results

ADP-heptose release in *H. pylori* infection induces a biphasic NF-κB response in gastric epithelial cells. ADP-heptose-deficient bacteria failed to induce the first phase of NF-κB signaling while CagA mutant bacteria failed to induce sustained NF-κB activation. The early, rapid phase (1 -3 h) required ALPK1 and TIFA, whereas the delayed and sustained phase (4 -12 h) remained largely unaffected in CRISPR/Cas9 ALPK1 or TIFA knock out cells. TRAF6 knockdown abrogated both early and delayed NF-κB activation. The mechanism underlying CagA-dependent sustained NF-κB activity and the molecules involved are currently being investigated. The use of a Transwell co-culture system with *H. pylori*-infected polarized epithelial cells and gastric fibroblasts demonstrated suppression of NF-κB activity. The molecular interactions between the cells and the release of factors are being studied.

Conclusions

We have defined bacterial and host mechanisms underlying the biphasic NF-κB activation, providing a refined framework for the effectors and the temporal dynamics of the gastric response to *H. pylori* infection.



P8-2 | SOHAIL AHMAD

Supervisor: Peter Mertens

July 08, 2026

13:30

The role of cold shock proteins in mitochondrial homeostasis and for tubular cell phenotype determination during cell stress

Background

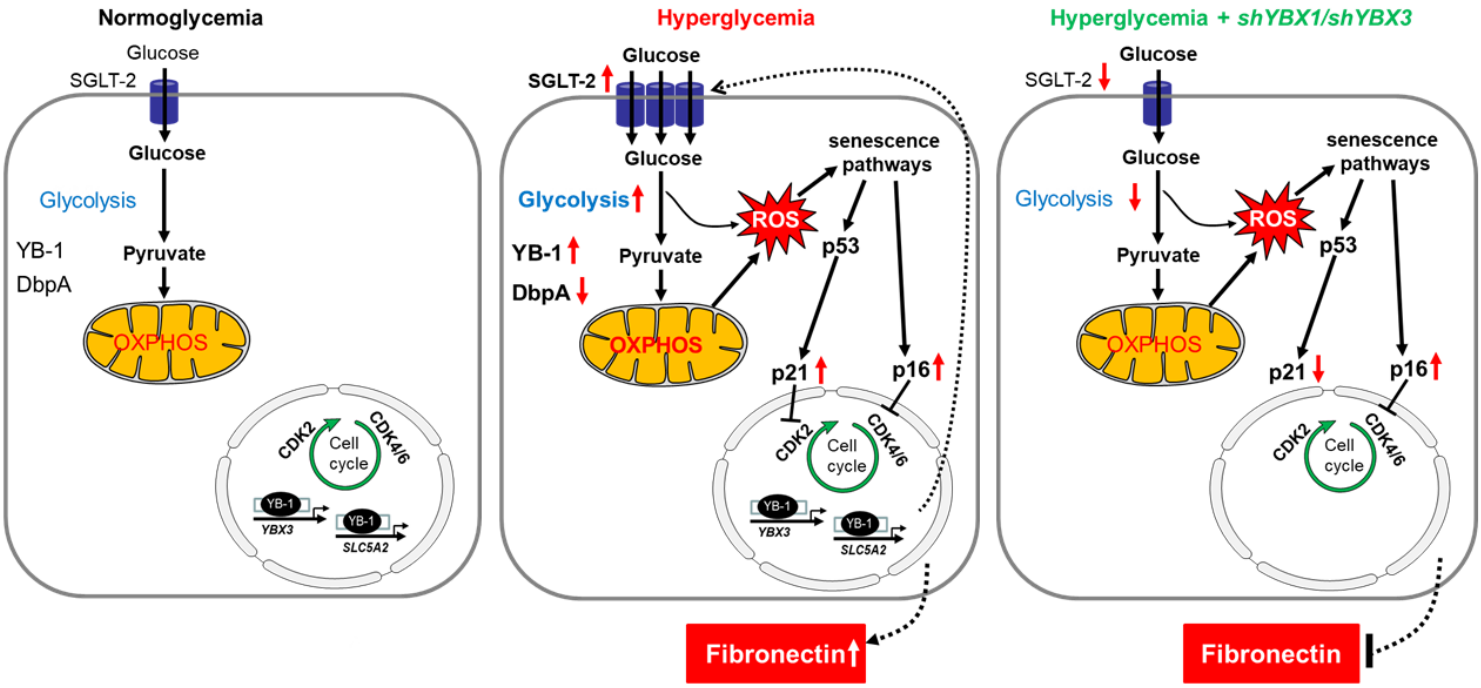
About 30% of diabetic patients develop chronic kidney disease. Data suggest that tubular cell senescence is driving the tissue damage. Cold shock proteins YB-1 and DbpA could be crucial due to their role in cell cycle and proliferation.

Results

Mouse primary tubular cells has shown upregulated senescence (p53, p21, p16) and fibrosis (FN) markers when cultured in high glucose compare to low glucose medium. we observe a counter-regulation of cold shock protein YB-1 and DbpA under high glucose conditions: YB-1 is upregulated (particularly with acetylation at lysine301/304), whereas DbpA abundance is reduced. At the same time precipitated proteins from the supernatants revealed secretion of both proteins in significant amounts. Contrary to our expectation DbpA knockout primary tubular cells were even more susceptible to high glucose-mediated senescence (p16, p21). In a similar approach immortalized human kidney cells has also shown upregulated senescence and fibrosis markers under high glucose condition. Lentiviral knockdown of YB-1 and DbpA had a suppressive effect on senescence as well as fibronectin synthesis. With elevated glucose, an upregulated SGLT-2 expression was observed in both cell types. Knockdown of YB-1 and DbpA resulted in lowered SGLT2 expression in HEK293 cells. Further, SGLT2 inhibition reduces the high glucose induce senescence markers p16 and p21, however cold shock proteins expression was not altered.

Conclusions

YB-1 as well as DbpA have profound effects on tubular cells phenotypes under elevated glucose concentrations. In addition, both proteins are involved in the regulation of SGLT2 expression in primary and immortalized kidney cells.



MD13 | MARTIN HOPPSTOCK

Supervisor: Peter Mertens

July 08, 2026

14:35

Deciphering extracellular Cold-Shock-Protein-RNA:IgG complex formation and elucidating cellular effects thereof

Background

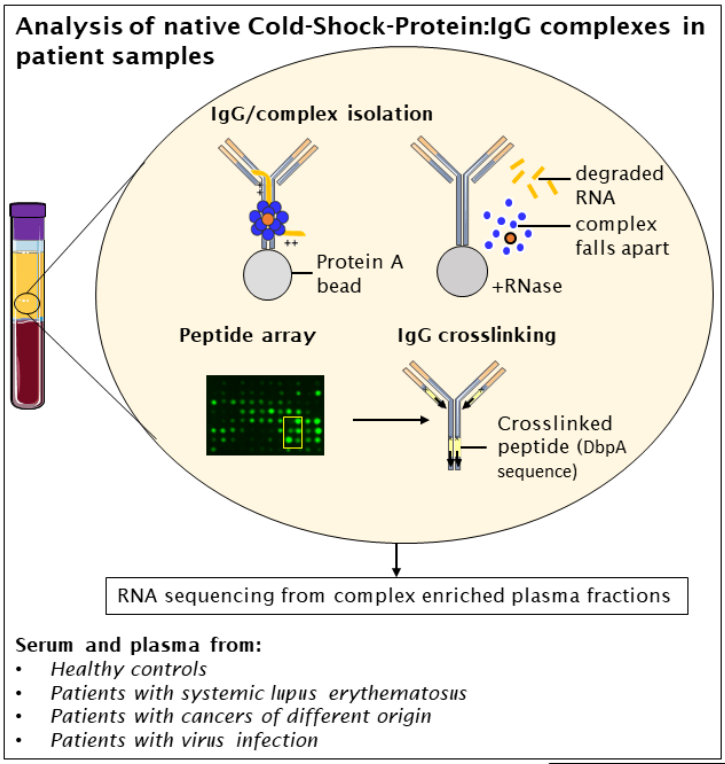
Cold-shock-proteins (CSPs) DbpA and YB-1 are secreted in a stimulus-dependent manner acting as alarmins. The linear DbpA -YB-1 correlation suggests a complex formation. The aim is to investigate the extracellular role of CSP-complexes.

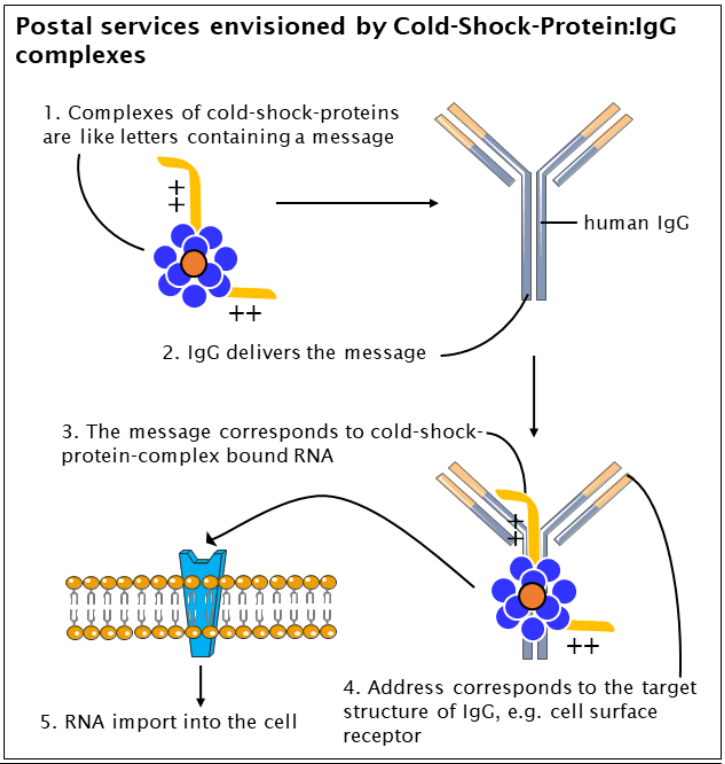
Results

In size-exclusion chromatographic fractions of human plasma, cold-shock proteins (CSPs) were detected as high-molecular-weight (HMW) material co-migrating with IgG, suggesting the formation of CSP–IgG complexes. These complexes could be reconstituted in vitro by incubating recombinant CSPs with IgG. IgG-binding sites within the DbpA molecule were identified using a peptide array. Corresponding DbpA-derived peptides were synthesized and selectively crosslinked to the constant regions of IgG. A subset of IgG subclasses preferentially crosslinked with the DbpA-derived peptide. As CSPs are nucleic acid-binding proteins, the role of nucleic acids in complex formation was investigated. Treatment with RNase destabilized CSP–IgG complexes and markedly reduced crosslinking efficiency, whereas DNase treatment had no effect. Upon elution of CSP:IgG complexes from human plasma, a co-eluting RNA species of approximately 6,000 nucleotides was detected.

Conclusions

We propose a model in which RNA associates with CSPs, promoting the interaction with IgG molecules. IgG facilitates the transfer of RNA-bound CSPs between cells, thereby contributing to intercellular communication under stress conditions.





P1-2 | LORENA FERINO

Supervisor: Michael Naumann

July 08, 2026

15:20

USP48-dependent regulation of NF-κB in the *H. pylori* infected gastric mucosa

Background

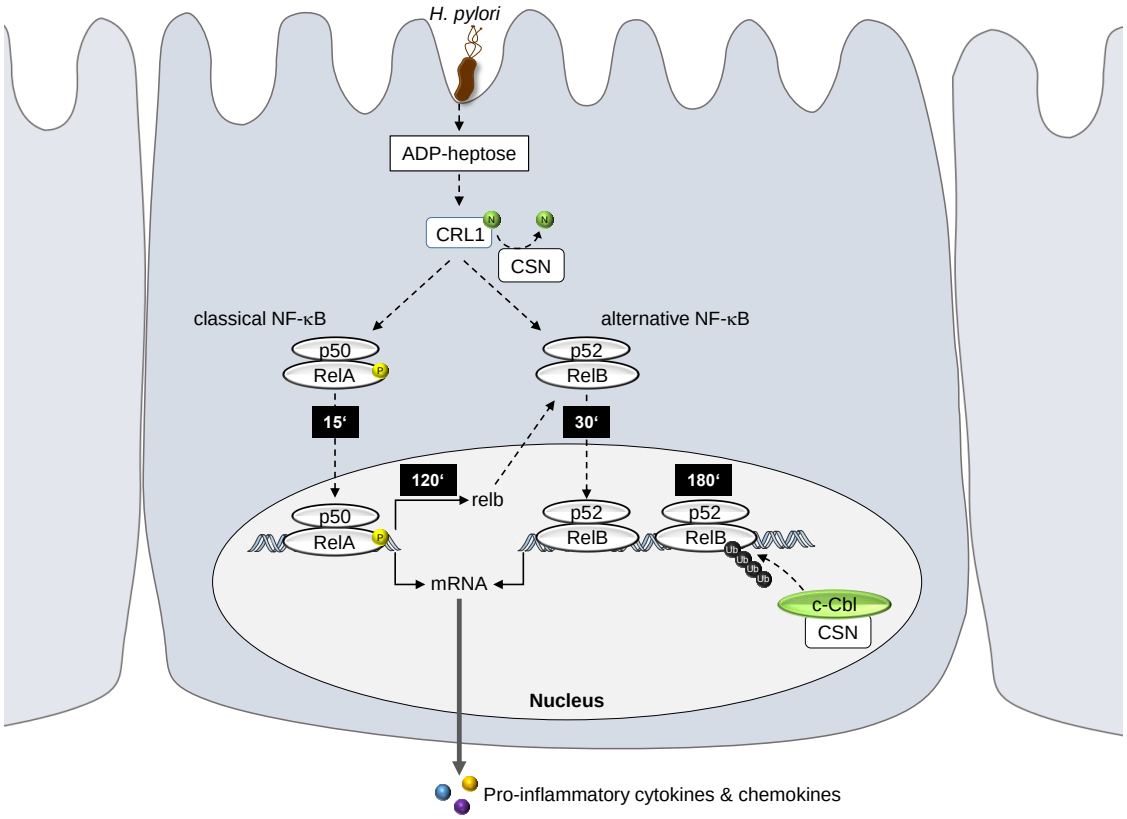
H. pylori induces classical NF-κB/RelA and alternative NF-κB/RelB in the gastric epithelium. RelA regulation is controlled by the CSN, directing CRL1 and USP48 activity. RelB regulation in *H. pylori* infection is unresolved.

Results

The *H. pylori* effector molecule ADP-heptose induces both the classical and the alternative NF-κB pathway. The alternative NF-κB signaling pathway during *H. pylori* infection induces the translocation of RelB/p52 into the nucleus, and RelB is partially synthesized de novo via the classical NF-κB signaling pathway, increasing its concentration in the nucleus. The COP9 signalosome (CSN) and the CSN-associated deubiquitinylase USP48 control this process. In order to shut off alternative NF-κB, RelB becomes ubiquitinylated by the CSN-associated E3 ligase c-Cbl. CSN-associated USP48 might act in stabilizing RelB through deubiquitinylation. In biopsies of B-gastritis and gastric adenocarcinoma patients USP48 is upregulated and might serve as a prognostic disease marker. Further, USP48 is recruited to newly hypomethylated CpG islands and deubiquitinylates histones, suggesting a mechanistic link between DNA hypomethylation and tumor state.

Conclusions

CSN controls alternative NF-κB activity by modulating CRL1 activity and stabilizing nuclear RelA to promote expression of RelB. C-Cbl ubiquitinylates nuclear RelB and directs shut off of alternative NF-κB activity.



Long-term local and systemic effects of intestinal inflammation on the immune response to secondary challenges

Background

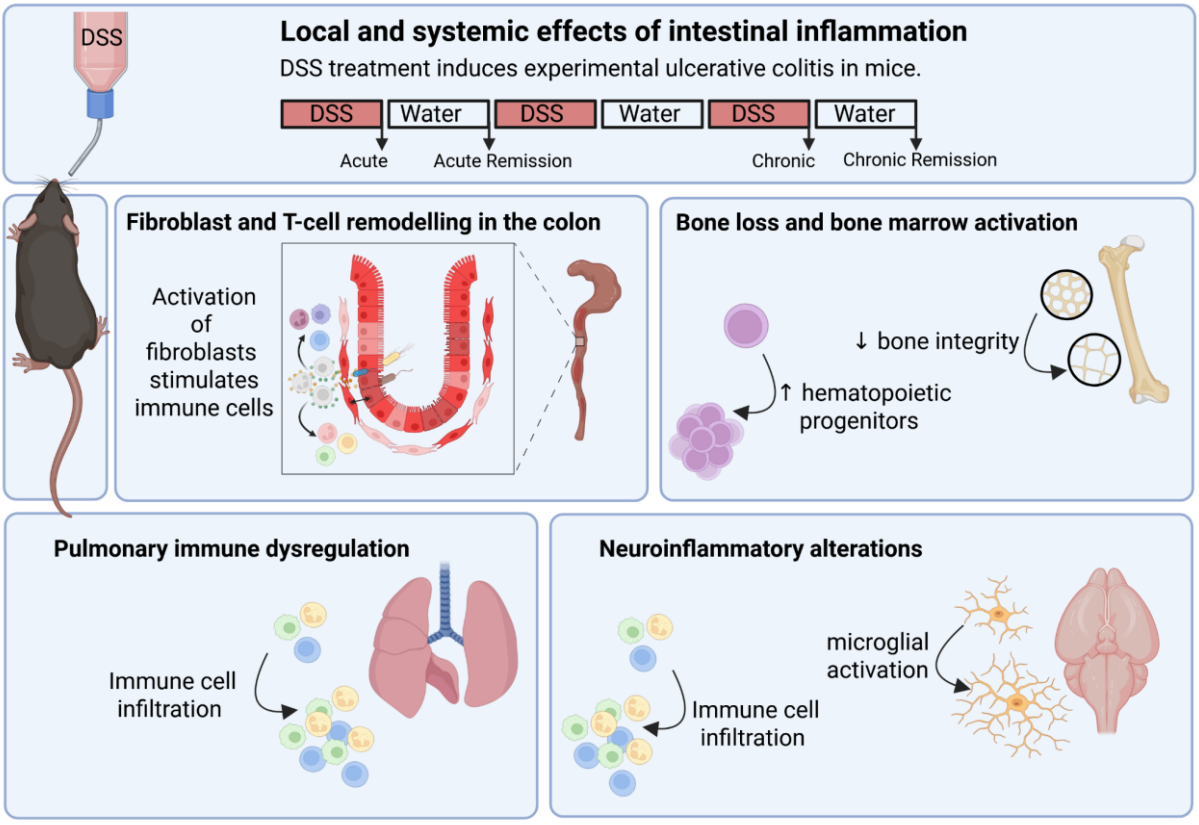
Ulcerative colitis is a chronic inflammatory disorder of the intestine that can be experimentally modelled in mice using dextran sulfate sodium (DSS). While primarily affecting the colon, UC is increasingly recognized as a systemic disease.

Results

To induce chronic colitis, mice received three DSS cycles followed by three weeks of water for remission, while controls had normal water. After sacrifice, colon, lungs, brain, and bones and bone marrow (BM) were analysed using flow-cytometry. DSS colitis induced systemic immune and stromal remodelling beyond the gut. Acute disease reduced several pulmonary immune cell populations, whereas chronic remission was characterized by expansion of myeloid and lymphoid cells. Colonic fibroblasts and T-cell subsets showed disease-stage-dependent alterations, including regulatory T-cell shifts during chronic inflammation and remission. In the bone marrow, chronic remission promoted expansion of hematopoietic stem and progenitor compartments. DSS colitis also reduced bone volume and mineral density with increased trabecular spacing, indicating weaker bone structure. In brain tissue, tight junction protein changes in cortex and hippocampus were accompanied by lymphocyte and myeloid infiltration. Microglia showed condition-dependent morphological and activation marker alterations.

Conclusions

Research into extraintestinal manifestations, particularly in the lungs and bone marrow, but also in the brain and bone structure, provides valuable insights into the systemic pathophysiology of the disease.



Analyzing the impact of mesenchymal stromal cells as tolerance promoting barrier in the context of CAR T cell therapies

Background

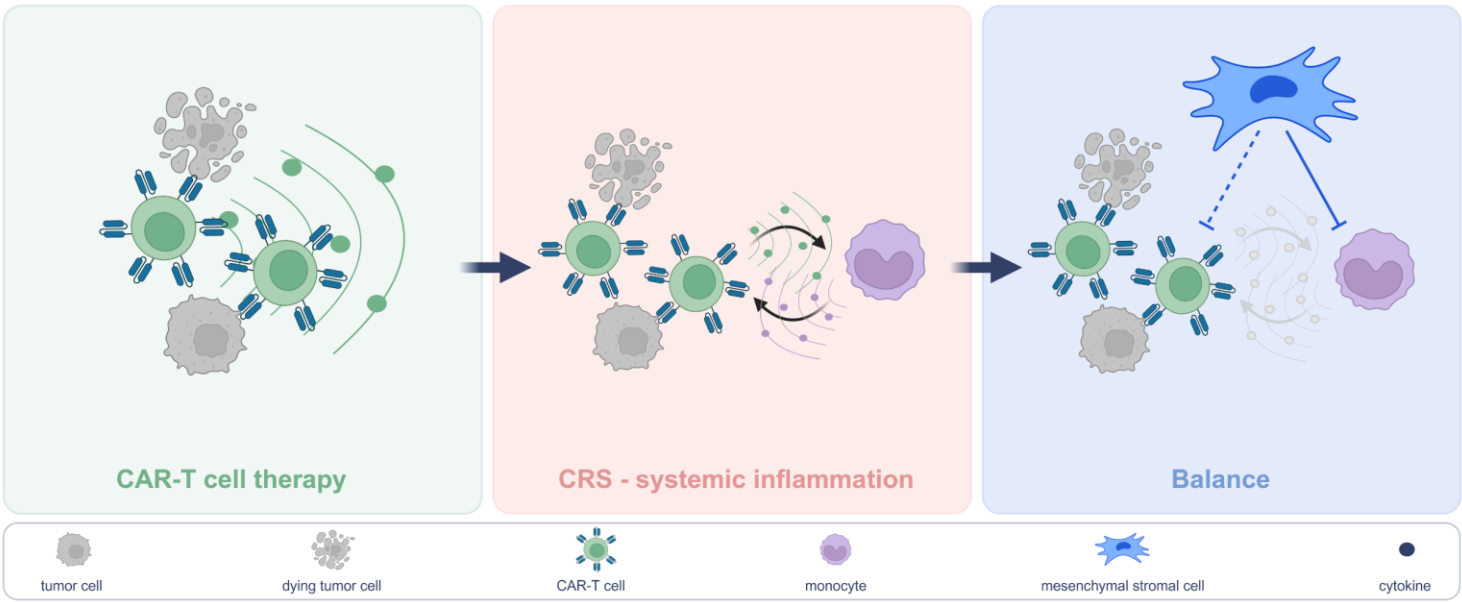
CAR-T cell therapy is associated with severe, potentially lethal, toxicities due to excessive inflammatory immune activation. These toxicities might be alleviated by the immunomodulatory properties of mesenchymal stromal cells.

Results

We established an *in vitro* model of cytokine release syndrome (CRS) to mimic the development of CAR (chimeric antigen receptor) T cell therapy-associated CRS. The model contains the main components associated with CRS development: CAR T cells, tumor cells, and monocytes. Co-cultures of these three cell types showed a significantly increased production of CRS-associated cytokines and prominent activation of the CAR-T cells and monocytes, compared to double or single cultures of the corresponding cell types, confirming an efficient *in vitro* CRS development. Increasing the tumor burden in the system led to even stronger CRS development, as observed in patients. Further testing also revealed that the model was responsive to conventional CRS treatments. Based on these findings and the reliable characteristics of the CRS model, we introduced MSCs (mesenchymal stromal cells) to the *in vitro* system. This treatment led to a significant reduction in several CRS-associated cytokines. Simultaneously, no relevant impairment of CAR-T cell activation or tumor clearance was observed, which supports our hypothesis that MSCs could be used as a new therapeutic option against CRS.

Conclusions

Our *in vitro* findings pave the way for subsequent in vivo investigations, which will examine the efficacy of MSCs against CAR-T cell therapy-associated CRS, as well as potential adverse effects in a complex mammalian organism.



MD14 | DAVID BECKER

Supervisor: Dunja Bruder

July 09, 2026

09:00

Investigating the influence of chronic intestinal inflammation on the antibacterial immune response in the lung

Background

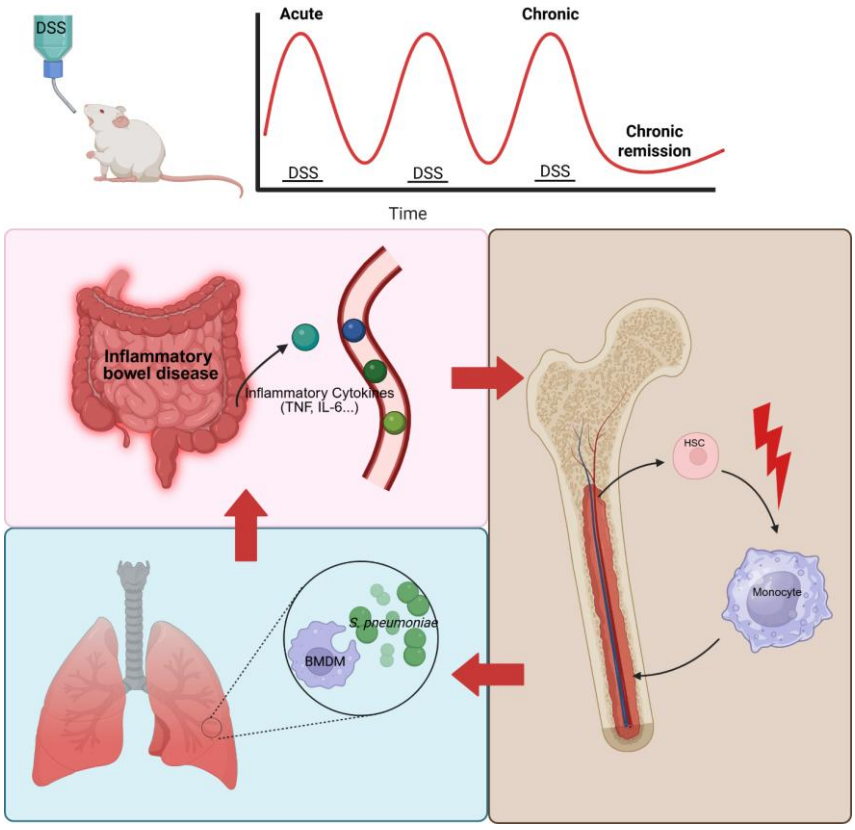
Ulcerative colitis (UC) is a chronic inflammatory bowel disease characterized by continuous mucosal inflammation of the colon. We hypothesize that chronic inflammation modulates haematopoiesis within the bone marrow.

Results

Chronic colitis in mice is induced using repeated cycles of dextran sulphate sodium (DSS) to model the acute, chronic, and remission phases of the disease. Following induction, bone marrow cells are harvested ex vivo and differentiated in vitro into bone marrow-derived macrophages using M-CSF. Subsequently gene expression analysis via qRT-PCR will be performed on macrophages across all disease phases (acute, chronic, and remission) under M0, M1 and M2 conditions. Expression of pro-inflammatory markers NOS2, TNF-α, IL-1β, and IL-6 will be assessed as indicators of classical M1 activation, while Arg1, EGR2 and CD206 will serve as markers of alternative M2 polarization. These markers were selected for their reciprocal expression patterns, which allow robust discrimination between M1 and M2 states, and can reflect the trained immunity and tolerance programs through which chronic inflammation is thought to epigenetically alter bone marrow progenitors and thereby the functional output of their macrophage progeny. Finally, the in vivo phase of the study will involve challenging colitic mice in the remission phase with the respiratory pathogen *S. pneumoniae.*

Conclusions

Chronic colitis may alter bone marrow immune cell development, reshaping systemic innate immunity via the gut-bone marrow-lung axis — potentially explaining why ulcerative colitis patients face higher respiratory infection risk.



Interaction of the airway epithelial barrier and T helper (Th) 2 cells during helminth infection of the lung

Background

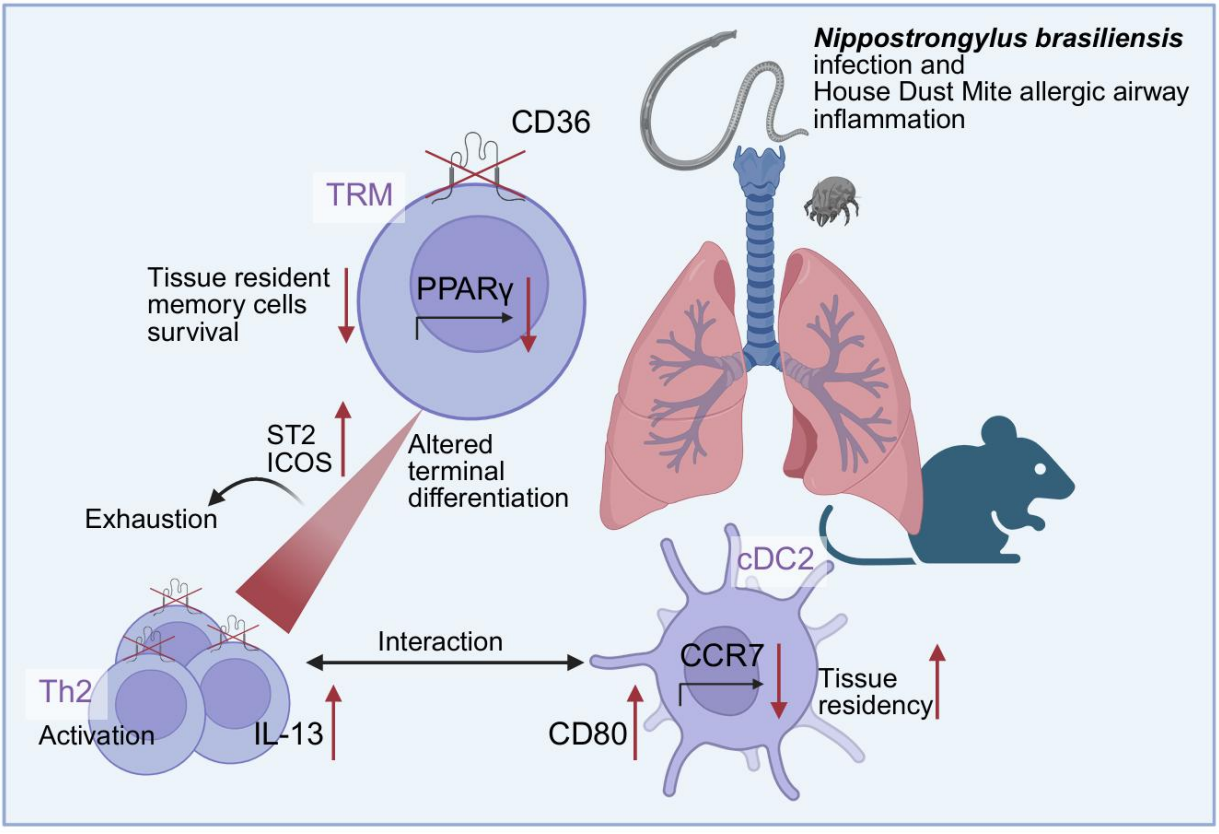
Th2 cells contribute to allergic airway inflammation but also mediate anti-helminth immunity. As CD36 is enriched on lung CD4+ T cells during asthma, we investigated its role in protective immunity to *Nippostrongylus brasiliensis*.

Results

As CD36 is a scavenger receptor and lipid sensor, lipid metabolism may play a crucial role in Th2 immune responses. Recent studies, as well as our own preliminary data, suggest that Th2 cells depend strongly on lipid metabolic pathways during allergic airway inflammation (Project 12-2). In an *in vivo* helminth infection model, we observed altered TRM differentiation in CD36-deficient CD4+ T cells, together with reduced PPARγ. This alteration may result from T cell exhaustion, as exhaustion markers such as ST2 and ICOS are increased on CD36-deficient CD4+ T cells. In contrast to allergic airway inflammation, parasite infection studies revealed impaired cDC2 migration, suggesting their retention in the lung. This is consistent with increased expression of activation markers on CD36-deficient CD4+ T cells and enhanced T cell-cDC2 interactions. At present, the interaction between Th2 cells and airway epithelial cells during physiological anti-helminth immunity and pathological allergic asthma remains insufficiently understood. Therefore, we will use single-cell RNA sequencing to compare in vivo data from allergic airway inflammation and *N. brasiliensis* infection.

Conclusions

Our project could have significant implications for precision and personalised medicine, for example in the development of novel therapeutics that target pathogenic Th2 cells in allergy without compromising immunity to infection.



P11-3 | SHIVANI SINGH

Supervisor: Verena Keitel-Anselmino

July 09, 2026

10:50

Optimized models for studying intra- and intercellular crosstalk in biliary diseases

Background

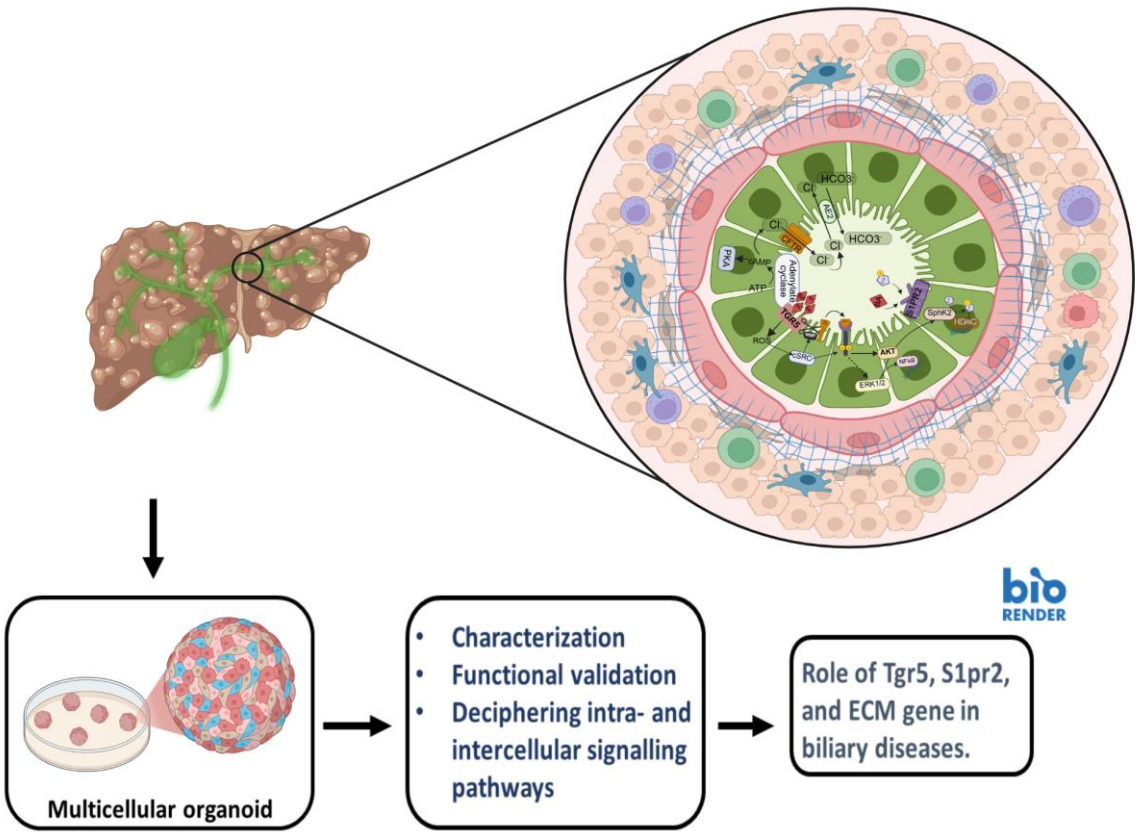
Optimized multicellular organoid models incorporating bile acid signaling and ECM gene networks represent a crucial step towards more physiologically relevant disease modelling and therapeutic development in biliary diseases.

Results

Multicellular liver organoids were generated from liver tissue isolated from wild-type and knockout mice lacking the Tgr5 receptor. Following liver perfusion with different collagenases, distinct populations of hepatic cells were isolated. Hepatocytes and non-parenchymal cells (NPCs) were seeded at a 2:1 ratio and cultured for 15-20 days to promote organoid formation. Organoid morphology was evaluated using hematoxylin and eosin (H&E) staining, while cellular composition was confirmed by immunohisto-chemistry (IHC). Bile canaliculi-like structures were assessed using the CellTracker™ Green CMFDA assay, and inflammatory cytokine expression (TNF- and IL-6) was quantified by qRT-PCR. Furthermore, treatment with oleic acid and palmitic acid induced metabolic stress in the multicellular liver organoids, enabling the in-vitro modeling of fatty liver disease.

Conclusions

Multicellular liver organoids were successfully established and characterized both structurally and functionally, providing a physiologically relevant platform for studying the mechanisms involved in biliary diseases.



MD15 | JAKOB WOLF

Supervisor: Anne Dudeck

July 09, 2026

11:35

Mast cell granules as regulators of LPS-induced macrophage tolerance

Background

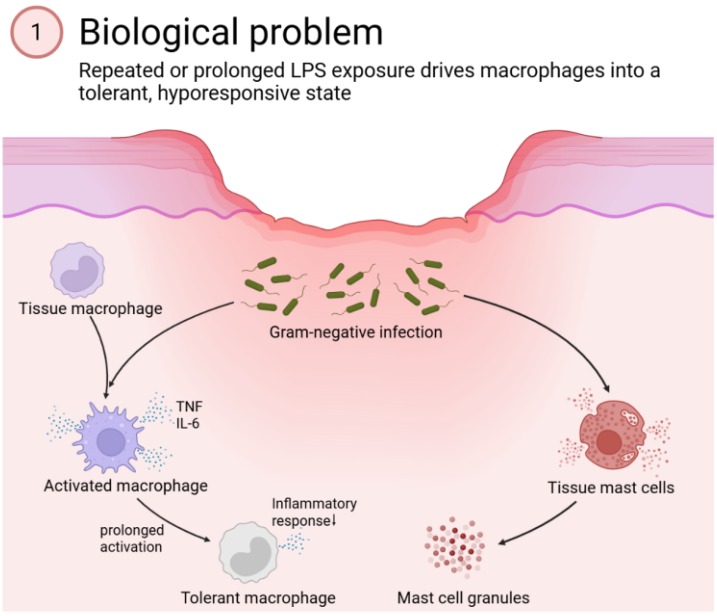
Repeated exposure to LPS can induce tolerance in macrophages, reducing their responsiveness to subsequent infections. This protects tissue by limiting excessive inflammation, but can also allow bacteria to persist, spread, and cause sepsis.

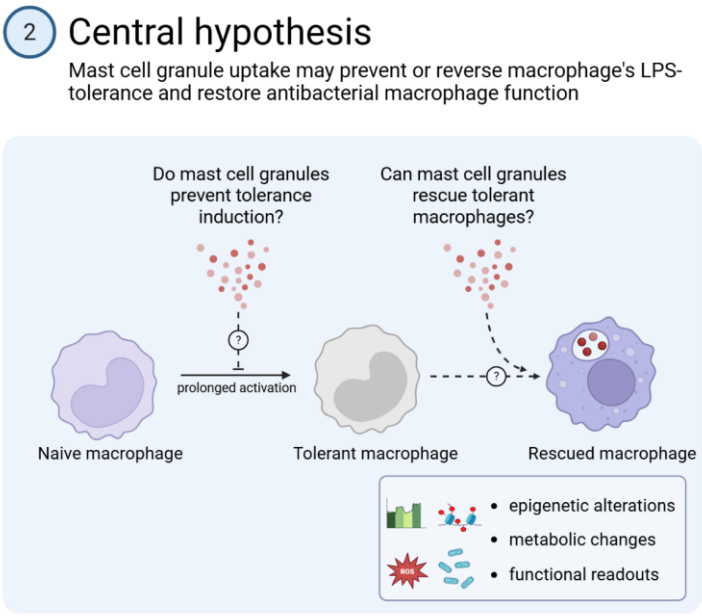
Results

Preliminary data from our group shows that mast cells (MCs) and macrophages are closely associated in barrier tissues such as the skin. We found that intact mast cell granules (MCGs) are engulfed by macrophages in murine models in vivo and in vitro. This uptake enhances macrophage function and induces a unique plasticity, combining features of both classically and alternatively activated states, suggesting improved versatility. These findings were confirmed with human MCs and macrophages in vitro and in situ, in healthy skin explants and psoriatic patient lesional skin. Additionally, MCG uptake alters phenotypic and functional changes of macrophages to LPS, suggesting that MCGs have the potential to modulate endotoxin-induced changes. This project explores whether MCGs help macrophages regain antibacterial functions under conditions where LPS tolerance would normally be detrimental. Our early observations suggest that MCGs modulate the tolerant state of macrophages in vitro, thus supporting this hypothesis. In particular, we can see a trend towards a restored cytokine response and significant reversal of phenotypic markers in samples receiving MCGs.

Conclusions

Understanding how MCG uptake regulates LPS tolerance could reveal new therapeutic targets. Preserving macrophage responsiveness is essential for maintaining barrier integrity and effective early immune defense.





P16-3 | NORA SIEGELT

Supervisor: Verena Keitel-Anselmino

July 09, 2026
15:10

Screening for systemic factors in biliary diseases

Background

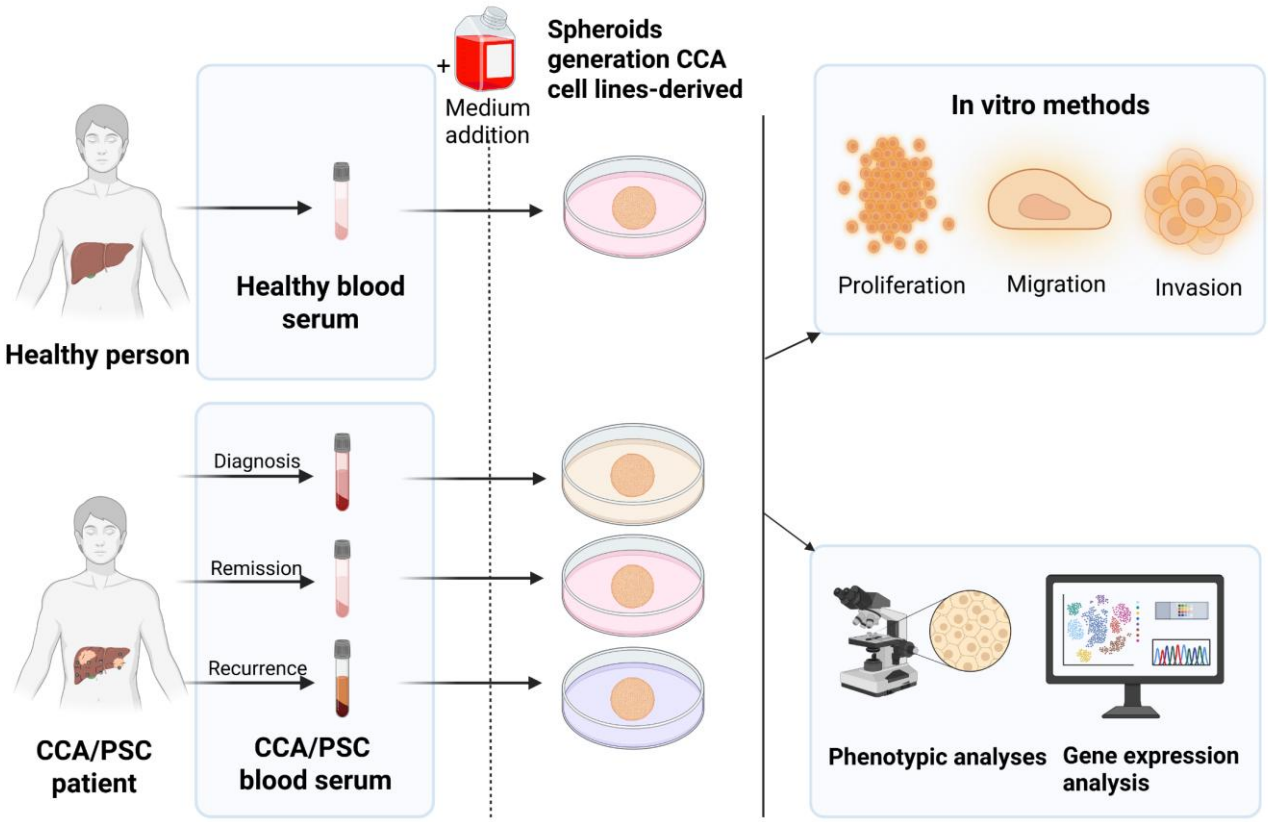
Cholangiocarcinoma (CCA) is an aggressive bile duct cancer with rising incidence and limited treatment options. Understanding how serum factors affect tumor behavior is key to discovering better biomarkers and targeted therapies.

Results

To modulate tumor behavior and its microenvironment we established a 3D spheroid model created from cholangiocyte cell lines. ECCA cell lines EGI-1 and TFK-1 and the normal cholangiocytes H69 formed the most stable and globular spheroids and showed the highest growth over time. Blood sera from various CCA and primary sclerosing cholangitis (PSC) patients were collected from our biobank, which were used to stimulate the spheroids. Spheroids stimulated with one of the PSC sera showed significant growth and phenotypic changes after 24 h, as determined by brightfield microscopy. The EGI-1 and TFK-1 spheroids exhibited a 2.5-fold and 4-fold increase in cell viability, respectively, when compared to spheroids exposed to healthy serum. In contrast, H69 spheroids showed lower viability, which is appropriate, as they are normal human cholangiocytes. Serum from another PSC patient did not elicit specific effects on spheroid growth. Further experiments are underway to identify the factors responsible for the observed growth stimulation.

Conclusions

Increased growth and viability of cancerous cholangiocytes in response to PSC serum highlight complex tumor microenvironment interactions and confirms the role of blood-borne factors in promoting tumor proliferation.



P1-4 | LUKAS GREIN

Supervisor: Martin Fischer

July 09, 2026

15:55

Elucidating Gene Regulatory Networks during Cell Cycle Entry

Background

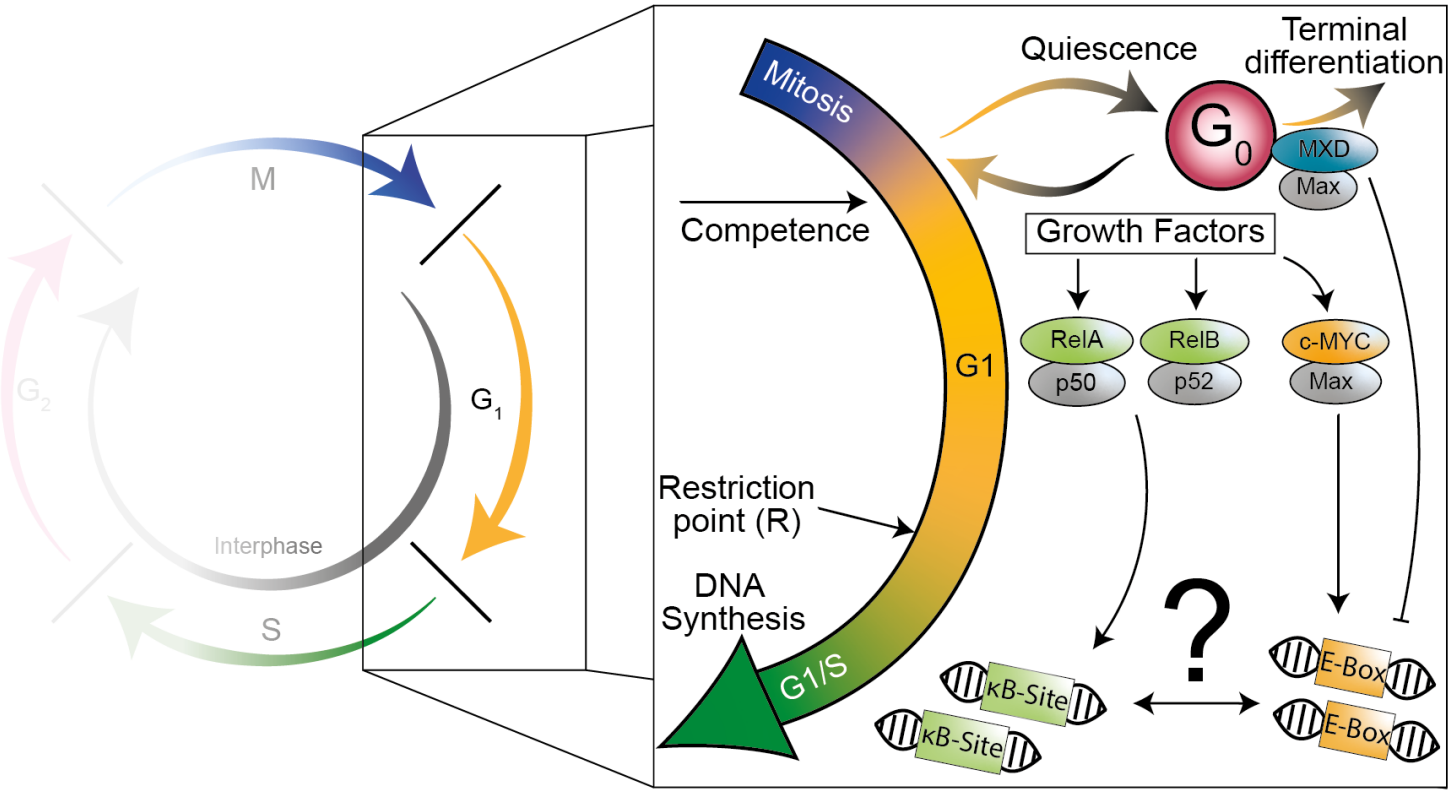
The cell cycle is central to growth and frequently dysregulated in cancer. Yet, genome-wide gene regulation during cell cycle entry and the roles of transcription factors such as MYC, MXDs and NF-κB remain incompletely understood.

Results

Using hTERT-immortalized BJ fibroblasts and RPE1 epithelial cells, I synchronized cells in quiescence by serum withdrawal and induced cell cycle re-entry by serum re-addition. Synchronization was validated by FACS. MYC-dependent gene expression was assessed by siRNA-mediated MYC depletion followed by RT-qPCR and RNA-seq. Integrating RNA-seq with publicly available MYC occupancy data, I identified 307 putative direct MYC target genes. Surprisingly, depletion of the MXD family members MXD1, MXI1, MXD4, and MNT - classical antagonists of MYC - had little effect on MYC target gene expression. Instead, RNA-seq analysis of MXD4-depleted BJ cells suggests a role for MXD4 in promoting an interferon response during quiescence. To investigate potential pioneer factor activity of MYC, I performed ATAC-seq. A small subset of MYC binding sites gained accessibility during cell cycle entry, supporting limited pioneer functionality. To identify MXD1 and MXD4 target genes, we generated RPE1 cells with doxycycline-inducible MXD1 or MXD4 expression and confirmed induction by Western Blot and RT-qPCR. Next, ChIP-seq and integrative analysis with RNA-seq will be used to infer MXD1 and MXD4 targets.

Conclusions

MYC and MXD transcription factors are strongly associated with cell cycle regulation, yet RNA-seq data do not support widespread antagonism at MYC target genes. ATAC-seq indicates limited pioneer activity of MYC during cell cycle entry.



MD15 | DEFNE HACIMAHMUTOGLU

Supervisor: Anne Dudeck

July 10, 2026

9:30

Investigating the role of the mast cell granule uptake in macrophage cell death and survival pathways

Background

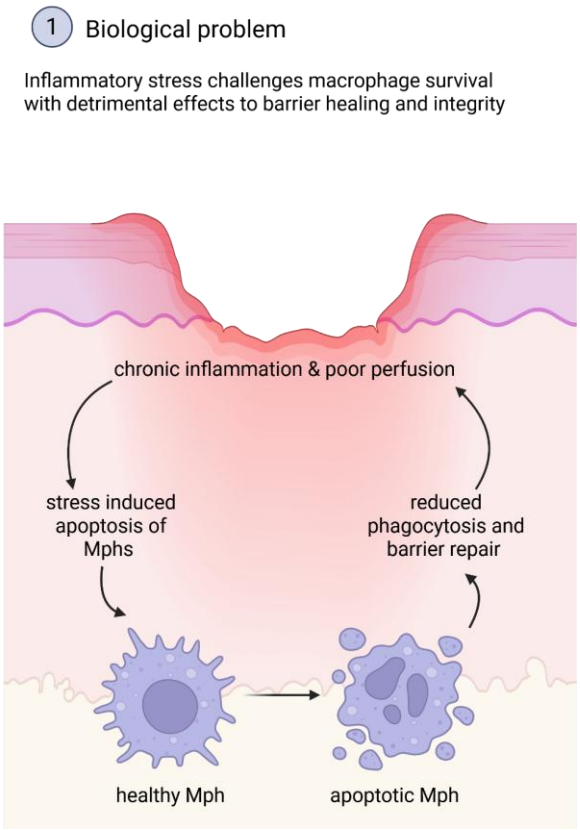
Mast cell granules (MCG) can be engulfed by macrophages (Mphs), inducing an atypical phenotype with enhanced phagocytosis. Our data suggest these granules persist for up to 7 days, but their functional impact remains unclear.

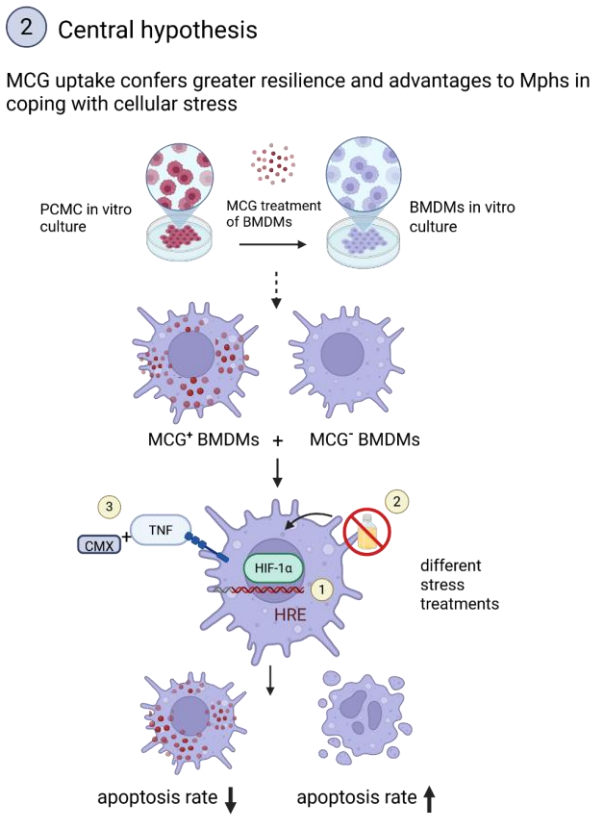
Results

In cases of skin inflammation, the ability of macrophages to survive stressful conditions, maintain self-renewal, or the type of cell death is crucial for maintaining the integrity of the skin barrier. Therefore investigating whether MCG uptake confers greater resilience and advantages to Mph in coping with cellular stress by modulating death pathways may reveal novel mechanisms relevant to chronic skin inflammation. Initial in vitro data has shown that serum deprivation predominantly induced caspase independent cell death, whereas hypoxia and TNF + Cycloheximide (CMX) treatment triggered apoptosis. Under hypoxic conditions, MCG+ macrophages exhibited consistently lower levels of all cell death markers compared to MCG− and untreated cells, indicating enhanced resistance. Following serum-free medium and TNF+CMX treatments, MCG+ Mphs showed significantly reduced secondary necrosis marker levels compared to MCG− macrophages, indicating delayed progression to late-stage cell death.

Conclusions

Thus, MCG uptake enhances Mph resilience under stress, particularly hypoxia, by reducing apoptotic responses and delaying progression to secondary necrosis, indicating a protective role of MC–Mph interactions in inflammatory environments.





AP12 | JULIUS RUBLACK

Supervisor: Dunja Bruder

July 10, 2026

10:15

Role of eosinophil subpopulations in allergic asthma and other eosinophil driven diseases

Background

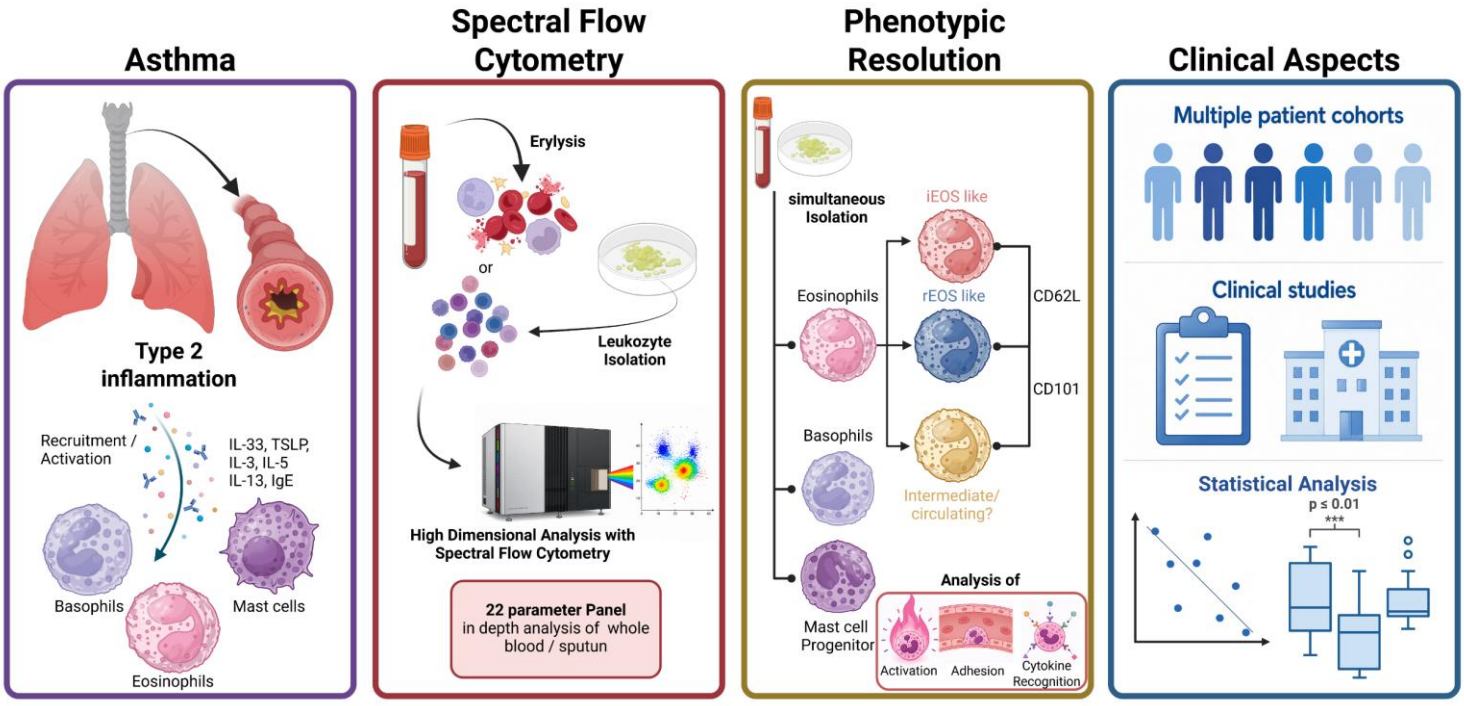
Asthma is one of the most common chronic airway diseases driven by type 2 inflammation. In this context, eosinophils, basophils and mast cells are key effector populations. Recent studies suggest that eosinophils in asthma can be subtyped.

Results

To address these subtypes and gain insight into the cell biology, we developed a 22-parameter spectral flow cytometry panel, combined with erythrocyte lysis, to enable the simultaneous identification and characterization of multiple asthma-relevant immune cell populations directly from whole blood or sputum. The panel was designed to detect eosinophils, basophils and mast cell progenitors, and to enable the subtyping of eosinophils and the assessment of activation- and trafficking-associated phenotypes. Preliminary analyses indicate that the panel robustly identifies the targeted cell populations and captures type 2-associated phenotypic variation in peripheral blood. In particular, the marker combination including CD62L, CD101, and CD44 appears to enable discrimination of eosinophil subsets beyond simple enumeration. Early observations further suggest the presence of an eosinophil population in asthma that does not fully align with the currently described phenotypes, but may instead represent an intermediate or persistently circulating state.

Conclusions

Overall, this spectral panel provides a promising platform for the integrated analysis of asthma-relevant immune cell populations. Its broader application may help refine eosinophil subset classification and support future studies.



Bidirectional cross talk between Mast Cells and Endothelial Cells in homeostasis and inflammation

Background

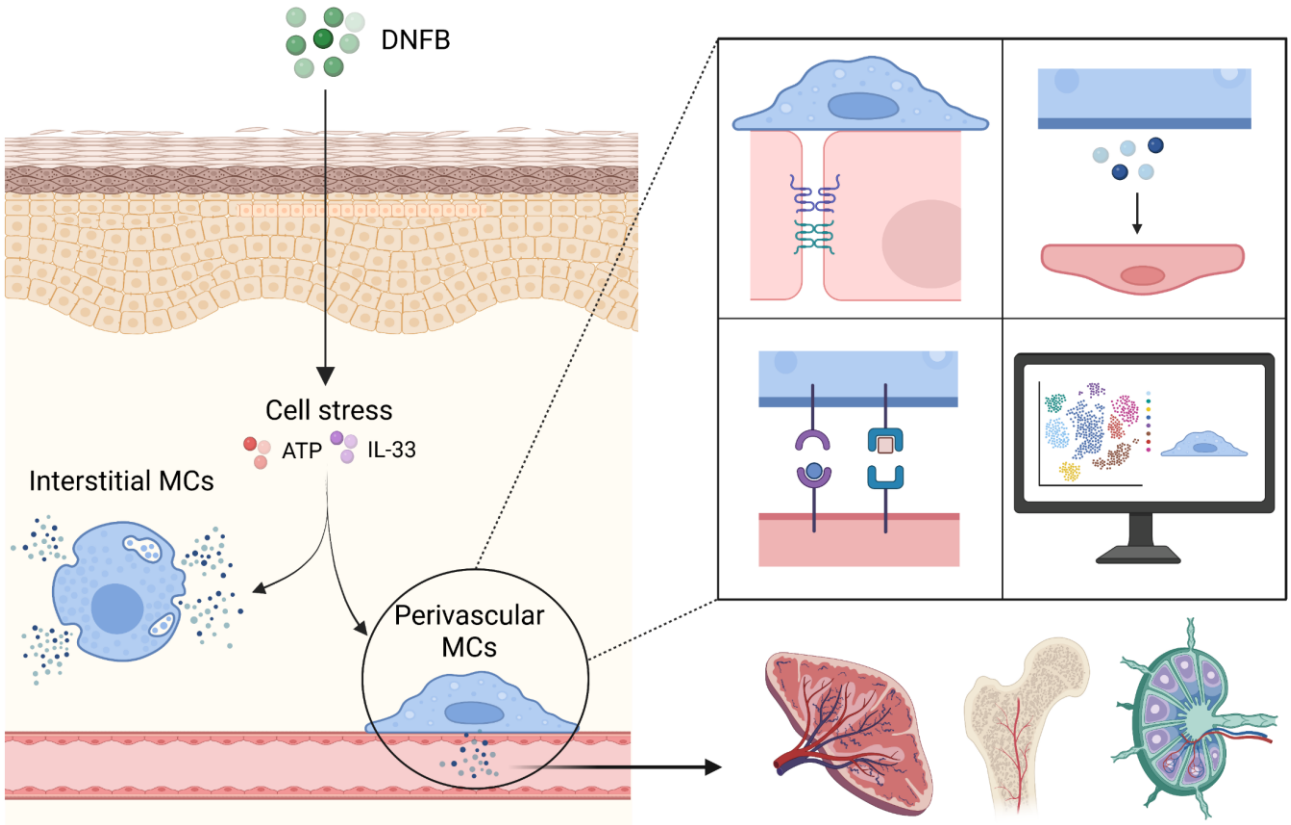
Mast cells (MCs) are long-lived, tissue-resident innate immune cells mainly found in tissues at environmental interfaces. The conventional classification divides MCs into mucosal MCs and connective tissue-type MCs.

Results

Recent findings suggest that MCs exhibit a greater degree of heterogeneity, based on their subanatomical tissue localization. In the skin, perivascular MCs (pvMCs) reside in close proximity to blood vessels and form intraluminal protrusions passing the endothelial barrier. Upon pvMC activation, intact MC granules (MCGs) are released directionally into the blood stream, thereby facilitating systemic distribution of MC mediators. Using a DNFB-based model of local skin inflammation, this project aims to identify the unique characteristics of pvMCs, which distinguish them from interstitial MCs and allow for their bidirectional communication with endothelial cells (ECs), by RNAseq analysis. The formation of intravascular protrusions passing the endothelial barrier and the effect of MC degranulation on blood vessel permeability will be assessed in vitro using co-culture assays and advanced imaging techniques. . Finally, we aim to define remote effects of intravascular skin MC degranulation in distant immune organs, including lymph nodes, spleen, and bone marrow.

Conclusions

pvMC characteristics and their capacity for intravascular degranulation may represent an important target for therapeutic approaches to intervene systemic inflammatory responses, such as cytokine storm syndromes or anaphylactic shock.



P8-3 | KASHOD ASLAM

Supervisor: Peter Mertens

July 10, 2026

12:05

Immunoglobulin-Mediated Trafficking of Nucleic Acid-Protein Complexes in kidney diseases

Background

IgG-bound riboprotein-RNA complexes traffic to kidneys, where Fc-mediated uptake triggers intracellular sensing and inflammatory pathways, driving the pathogenesis of immune-mediated renal injury.

Results

We propose a mechanism where IgG acts as a delivery vehicle for riboprotein-RNA complexes. To characterize these circulating assemblies, we utilize protein A bead-based isolation from patient serum and plasma, followed by RNA sequencing of enriched fractions to identify the RNA cargo, confirmed by the observation that RNase treatment causes the complexes to dissociate. We are investigating how these complexes are internalized by podocytes, mesangial and tubular cells via Fc receptor-mediated endocytosis and how this process is modulated by cell stress, such as high glucose concentrations, or the addition of complement factors. Furthermore, we are evaluating the downstream activation of immune-sensing pathways and quantifying signal transduction to measure cytokine production and cellular phenotypic changes, including matrix deposition and altered cell-cell interactions, which drive renal inflammation and injury.

Conclusions

This research will elucidate the composition of circulating riboprotein:IgG complexes and the specific cellular mechanisms including uptake and intracellular signaling that link these immune assemblies to the pathogenesis of kidney disease.