Universität Basel

Department of Biomedicine

DBM Basel

Newsletter
September 2026

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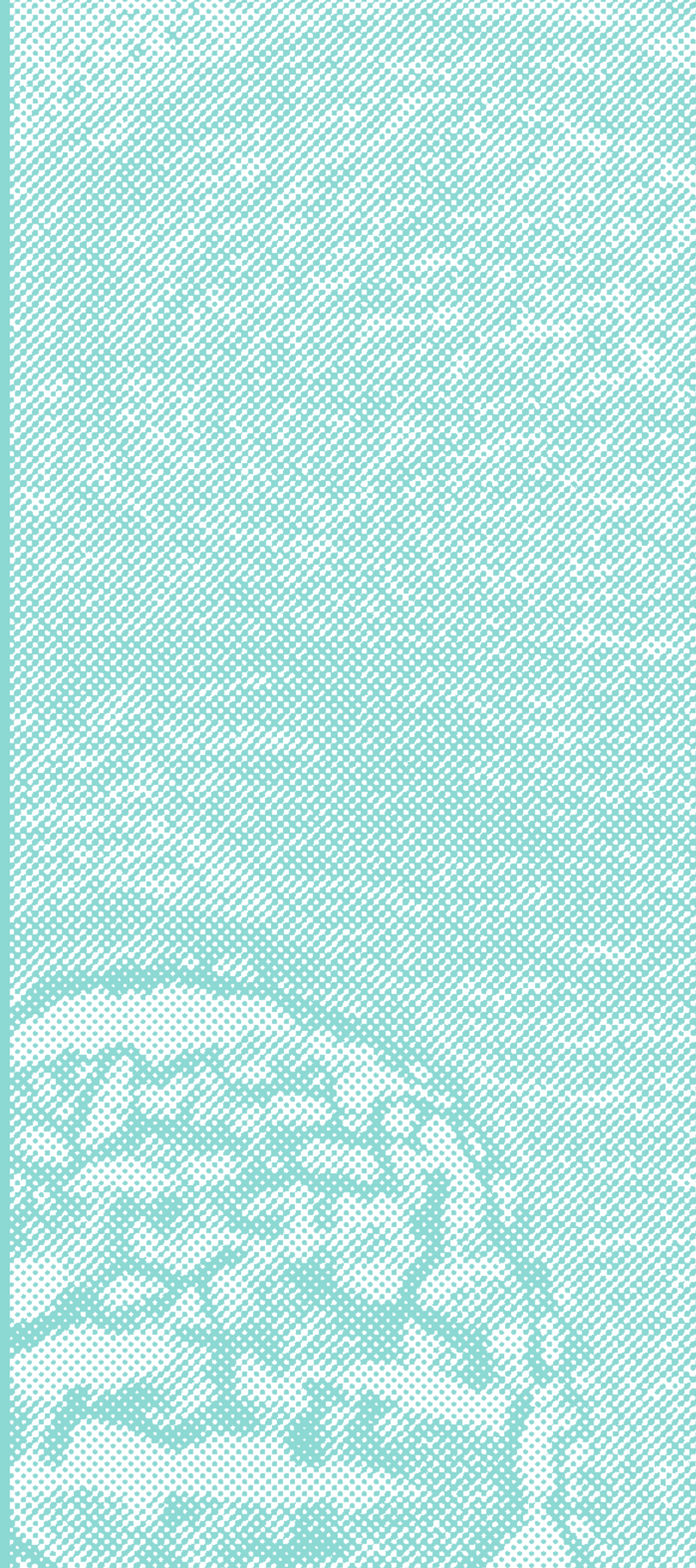

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From Summer Highlights to Autumn Insights

What a summer we had! Almost as if to mark its end, our Summer Symposium took place on the first day that truly felt like autumn – perfect timing for the autumn issue of our newsletter.

At this year's symposium, our PhD students and postdocs gave inspiring talks about their latest research findings from all DBM areas. As always, we also celebrated the best DBM publication of the past year, with Hailey Kim and Mika Schneider from the Clinical Neuroimmunology group selected by an external committee for their recent paper in *Cell*. In this issue, we get to know the two researchers behind the publication and learn more about their work, life outside the lab, and current projects.

We also dive into another research network involving several groups from our department: NCCR AntiResist. We spoke with Nina Khanna, deputy head of the NCCR, Carolyn King, Lucas Boeck, and Mattia Zampieri. They told us how they are working to tackle antibiotic resistance, what drives their research, and what they hope the future of infection treatment might look like.

We introduce two DBM research groups: Kirsten Mertz's Pathology of Infectious and Immunologic Diseases lab, which investigates the complex interactions between pathogens, human tissues, and the immune system, and Marten Trendelenburg's Clinical Immunology lab, which studies the role of complement C1q in systemic lupus erythematosus.

Of course, we also look back at some of our recent events, including the wonderful Athena's Journey Summer Apéro and the third Basel Spatial OMICS Symposium. These are two events you may want to keep on your radar for next summer.

We hope you enjoy this issue of our newsletter as much as we enjoyed putting it together! Thank you to everyone involved and for their contributions.

Your Communications Team

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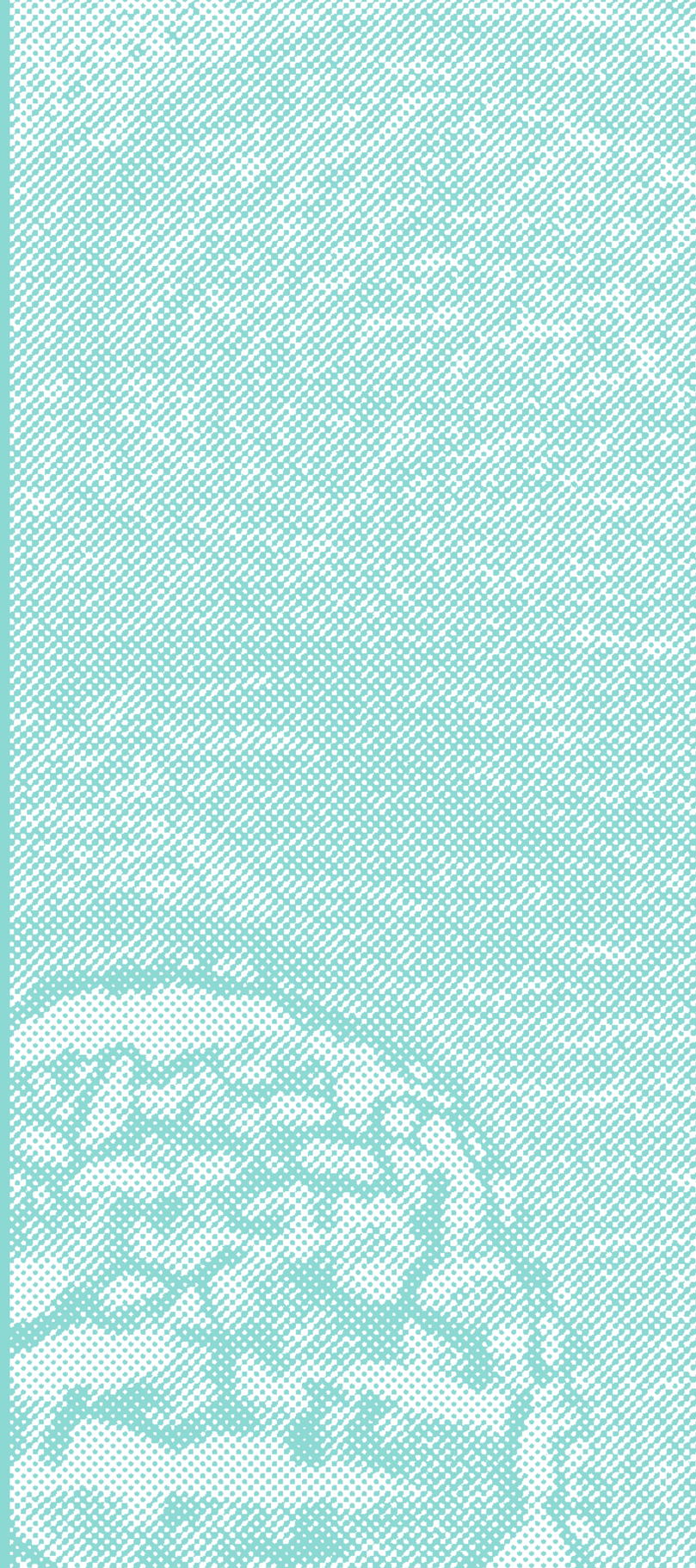

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Staying Ahead of Resistance

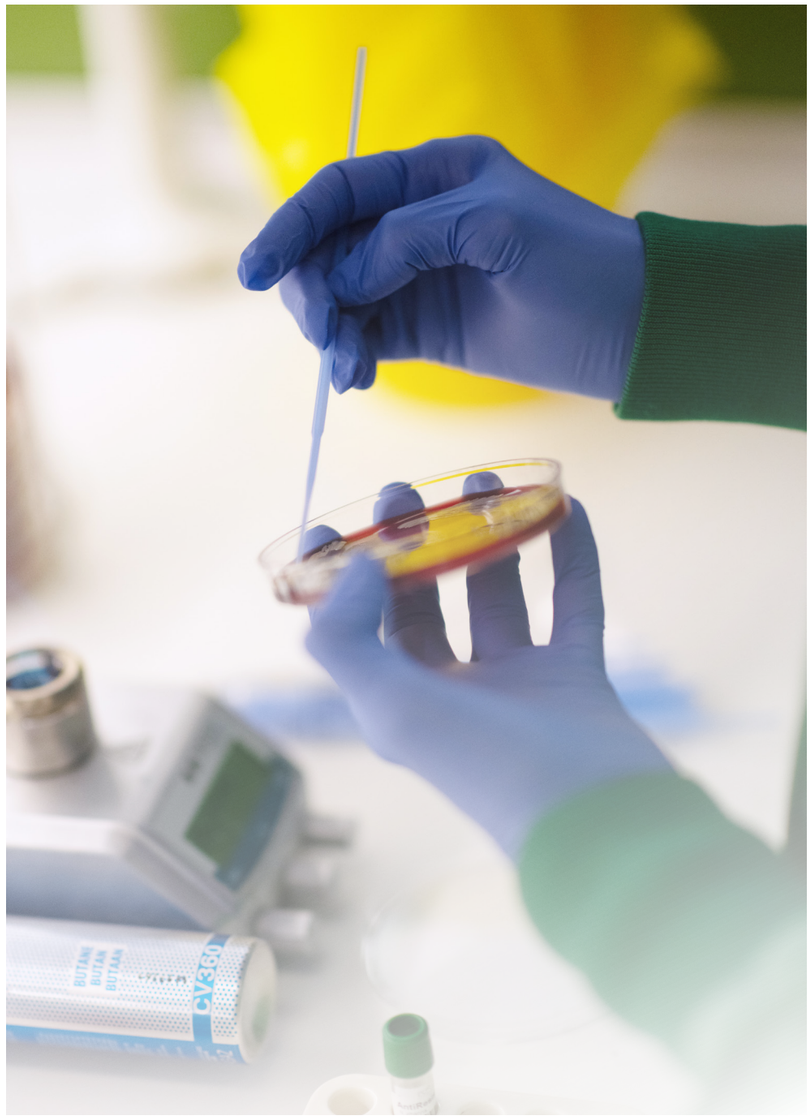
Four DBM research groups within the NCCR AntiResist aim to change how we understand bacterial infections – and how we develop the treatments needed to stay ahead of resistance.

Every year, antibiotic-resistant infections kill more people worldwide than HIV/AIDS and malaria combined. Yet because resistance creeps forward quietly, one failed treatment at a time, it rarely commands the same public urgency. The antibiotics we rely on today were largely discovered decades ago, and the pipeline of truly novel agents has remained alarmingly sparse for more than three decades.

Founded in 2020, Switzerland's National Centre of Competence in Research on Antimicrobial Resistance, NCCR AntiResist, was established to address this innovation gap. Its vision is to match the rapid evolutionary pace of antimicrobial resistance with a new paradigm in antibiotic discovery targeting *in vivo* pathogen behavior. By shifting the focus from conventional laboratory models to understanding how pathogens behave during infection, AntiResist brings together expertise from biology, engineering, chemistry, and clinical medicine across Swiss institutions to establish a sustainable pipeline of innovative antimicrobial strategies that can keep pace with antimicrobial resistance for generations to come.

The DBM is home to several research groups that are central to this mission, each tackling the challenge from a different angle: infectious diseases, immunology, systems biology, and pulmonary infection. We asked the three Prinicipal Investigators, Mattia Zampieri, Carolyn King, and Lucas Boeck, and Deputy Director Nina Khanna to share their perspectives on the urgency of the problem, the role their research plays, and what success could look like for patients by 2035.

Despite their different research perspectives, all four agree on one thing: antibiotic resistance threatens the foundation of modern medicine. Antibiotics are not only used to treat infections – they make organ transplantation, cancer chemotherapy, major surgery, intensive care, and many other medical treatments possible by preventing and treating life-threatening infections. Their success over many decades has made us take them for granted, but as resistance spreads, these life-saving treatments become far more dangerous or even impossible. Protecting antibiotics therefore means protecting modern medicine itself. "What's less appreciated is that antibiotics rarely work alone – they often partner with our immune system. So resistance isn't simply a molecular arms race but also a disruption of that drug-immune partnership. Understanding how bacteria and immunity interact is essential to develop better treatments," says Carolyn King, and Lucas Boeck adds, "what I want people to understand is that this is not an unsolvable problem. I am convinced that with scientific innovation, effort, and collaboration, we can outcompete bacterial evolution again, just as we did in the early days of antibacterial therapy."



A researcher working with a Petri dish in the laboratory. (Photo credit: Agnieszka Wormus, A Brilliant Photo)

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We asked the four researchers what role their research plays in helping us stay ahead of resistant bacteria. Across the four research groups, a common goal is to better understand what determines whether an antibiotic treatment succeeds or fails during an actual infection. Their research approaches this challenge from different and complementary angles. For Nina, this means looking beyond antibiotic resistance to understand why treatment can fail even when bacteria remain susceptible to antibiotics. Her work focuses on bacterial resilience during infection, the ability of bacteria to persist, adapt, and survive antibiotic treatment within the human host. Increasing evidence suggests that antibiotic treatment failure is driven by bacterial resilience mechanisms – such as altered virulence, biofilm formation, and intracellular persistence – and by complex interactions with the host immune response. To investigate these mechanisms in the context of human infection, Nina and her team work closely with clinicians and study bacterial behavior directly in infected patient tissues: "We have established a unique clinical cohort with a linked biobank of patient samples and bacterial isolates. By applying state-of-the-art technologies, we can directly characterize bacterial physiology within infected human tissues." According to her, closing this knowledge gap is essential for developing the next generation of therapies that target bacterial resilience alongside antibiotic resistance.

"We have established a unique clinical cohort with a linked biobank of patient samples and bacterial isolates. By applying state-of-the-art technologies, we can directly characterize bacterial physiology within infected human tissues."

– Nina Khanna

Nina Khanna

Infection Biology Lab
and Deputy Head of NCCR AntiResist

Most antibiotic research is done in the laboratory. Why is it important to study bacteria directly in infected patients, and what have you discovered?

"My research focuses on *Staphylococcus aureus*, one of the leading causes of serious bacterial infections. Antibiotic treatment can fail even when the bacteria remain fully susceptible to the antibiotics tested in the laboratory. To understand why, we study bacterial behavior directly in infected human tissues rather than relying solely on laboratory models.

We found that *S. aureus* persists predominantly inside intermediate monocytes/macrophages, where it adopts a slow-growing, low-metabolic state that markedly reduces susceptibility to antibiotic killing despite adequate drug exposure. Although antibiotics substantially reduce bacterial burden, they fail to eradicate the infection completely. Even when routine cultures become negative, bacteria can persist in tissues, indicating that culture negativity often reflects the limits of diagnostic sensitivity rather than true bacterial clearance. We also identified bacterial genes that are essential for intracellular survival, providing new therapeutic targets. These findings redefine the biology of persistent *S. aureus* infections and provide a foundation for developing therapies that target bacterial resilience alongside antibiotic resistance, ultimately improving the treatment of difficult-to-cure infections."

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Mattia Zampieri approaches the problem from a different angle, developing new approaches to identify how potentially useful antibiotics work. “Even more importantly, we may identify molecules that appear inactive under standard conditions but interfere with processes that bacteria do not need in the laboratory, yet critically depend on to survive, adapt, or establish an infection inside the body,” Mattia emphasizes.

Mattia Zampieri

Systems Pharmacology and Biology of Metabolism

Your lab uses metabolic “fingerprints” of bacteria to understand how drugs work and why resistance develops. How does this approach open doors that traditional methods can not?

“Traditionally, researchers screen thousands of molecules to see whether they can kill bacteria under standard laboratory conditions and only afterwards try to determine their mechanism of action. We aim to reverse this process: by quickly understanding which cellular processes a molecule affects, we can make more rational decisions about whether and under which conditions it could become an effective antibiotic.
This is important because bacteria growing in a test tube behave very differently from bacteria causing an infection in the human body. Conventional screens therefore often identify compounds that work well in the laboratory but fail in patients. By understanding how a molecule works, we can recognize this hidden potential and make a more informed prediction of whether it could be effective in patients.”

Mattia Zampieri and his team discussing ongoing experiments in the laboratory.
(Photo credit: Agnieszka Wormus, A Brilliant Photo)

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Carolyn mentions that a lot of infection research studies bacteria in isolation, or a single cell type in a dish, exposed to a drug. This is not what happens in a real infection where bacteria meet a whole tissue defended by immune cells. “In our research we try to mimic lung infection by combining human epithelial cells together with macrophages, patrolling immune cells that engulf harmful bacteria. We grow these cells together at what’s called an ‘air-liquid’ interface so that one side is exposed to the air just like it is in your actual lungs. Unlike existing animal models, this approach allows us to capture the earliest hours after infection,” Carolyn explains. Ultimately, they would like to use this model to investigate how antibiotics – or other therapies like monoclonal antibodies – work together with the immune system during an active infection. Because it is built from human cells, this model can also be adapted to study how age, smoking, or underlying conditions like cystic fibrosis change the way infection and treatment unfold.

Laboratory work with cell culture samples under sterile conditions. (Photo credit: Agnieszka Wormus, A Brilliant Photo)

Carolyn King

Infection Immunology

A lot of antibiotic research focuses on the drug and the bacterium. Why might the immune cells – like macrophages – be a missing piece of the puzzle?

“Macrophages and other recruited immune cells are in contact with bacteria well before any drug arrives. But they're walking a sort of tightrope: too little activity, and bacteria might escape control; too much activity, and the resulting inflammation might damage the tissue itself. Understanding that balance could lay the foundation for treatments that work not just against the bacteria, but for the immune system.”

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Lucas adds that, for decades, researchers and physicians have evaluated antibiotics mainly by their ability to stop bacterial growth under simplified laboratory conditions. But infections inside the human body are far more complex. Antibiotics must reach the site of infection, work together with the immune system, and preferably eliminate the bacteria rather than limit their growth. “Our research aims to understand the bacterial behavior during infection that determines whether a treatment succeeds or fails. By uncovering the underlying mechanisms, we aim to develop better ways to select and design treatments that cure infections more effectively,” he concludes.

Lucas Boeck

Pulmonary Infection Biology

You have developed a new imaging platform that can observe bacteria being killed in hundreds of conditions simultaneously. What has that revealed that we couldn’t see before?

“Conventional bacterial assays that evaluate if bacteria are alive are slow, measure populations as a whole, and don’t tell us how individual bacteria behave. Our imaging platform changes this fundamentally. By combining high-throughput microscopy with computer vision, we can follow millions of individual bacteria in real time across tens of thousands of conditions. This allows us to see not only whether an antibiotic works on the whole population, but also how quickly cells die, which cells survive, and how strongly these responses vary between conditions. We have already shown that these killing measures contain clinically relevant information that conventional susceptibility tests miss. We now use this platform for three main goals: to uncover the biology of bacterial survival and death; to design and optimize antibiotic regimens using more informative readouts; and to identify the bacterial genetic determinants of killing, with the long-term goal of developing molecular tools that help personalize antibiotic treatment.”

Preparing microplates for high-throughput automated analysis in the laboratory.
(Photo credit: Agnieszka Wormus, A Brilliant Photo)

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We next asked about the moment in their work that made them realize how urgent this challenge really is. What struck Mattia most was seeing how easily bacteria can evolve resistance in the laboratory. By exposing them repeatedly to an antibiotic, it is often possible to obtain resistant populations surprisingly quickly, certainly much faster and more readily than he had imagined before working directly on this problem. This experience made the challenge feel very concrete. “It showed me how adaptable bacteria are and how quickly resistance can emerge,” he adds.

Carolyn’s realization came from outside the NCCR, through her work on tuberculosis (TB). As part of her involvement with the Gates Foundation, she had the opportunity to visit several TB-endemic countries, to see and hear firsthand the devastating toll this disease takes. “TB currently demands a grueling four- to six-month drug regimen and very few new drugs have reached patients in the last 50 years. Drug resistance remains a major threat, including here in Europe, where multidrug-resistant TB rates are among the highest in the world. On the host side, we still rely on a vaccine that is over a hundred years old and does little to prevent disease in adults. There's obviously a tremendous urgency to address this,” Carolyn says.

For Lucas, it was one of the first patients he saw as a junior doctor, a 35-year-old woman who repeatedly required intensive care treatment for a difficult *Pseudomonas aeruginosa* infection. Her condition would improve with treatment, only to deteriorate again. Over the course of several weeks, repeated antibiotic exposure and bacterial adaptation led to increasing drug resistance. Eventually, there was not a single effective treatment option left, and the patient passed away. “This experience highlights some of the challenges of treating difficult infections. Having active antibiotics does not always mean we can cure an infection. Some pulmonary infections persist for years despite being classified as drug-susceptible. At the same time, antibiotic exposure can drive bacterial adaptation, turning a treatable infection into a resistant and ultimately untreatable one,” Lucas explains.

For Nina, the clinical challenge was different but equally striking. As an infectious diseases physician, she cares for patients with severe infections every day. “Over the past 25 years, I have seen a worrying increase in antibiotic-resistant bacteria, leaving us with fewer and fewer treatment options for some patients. At the same time, I have been struck by another challenge: many patients continue to suffer from persistent infections even when the bacteria remain fully susceptible to antibiotics in the laboratory and the patients receive the correct treatment. This taught me that antibiotic resistance is only part of the problem.” This clinical experience led her to investigate what happens to bacteria during infection that allows them to survive otherwise appropriate antibiotic treatment and ultimately to focus on bacterial resilience as a complementary target to resistance.

The four researchers share a vision of 2035 in which bacterial infections are understood and treated very differently. Instead of relying primarily on laboratory tests that do not fully reflect what happens in the human body, new models and technologies help reveal how bacteria behave during infection, why treatments succeed or fail, and how they interact with the immune system. This knowledge enables the development of new antibiotics and other therapeutic strategies targeting not only bacterial resistance, but also resilience and host-pathogen interactions. Treatments can be selected more precisely for an individual infection, making therapy less trial and error, faster and more effective – with fewer treatment failures, faster recovery, and ultimately more lives saved.

Carolyn makes this future tangible: “It's 2035, and a patient comes into the hospital with severe pneumonia. Instead of doctors reaching for the same handful of antibiotics and hoping one still works, they can now check how the patient's own immune system is responding – and choose a treatment that supports it, whether that's an antibiotic, a host-directed therapy or some combination of both.”

For Mattia, success would also leave a legacy beyond individual treatments: “Equally important, I hope we will have trained a new generation of scientists with the creativity and interdisciplinary expertise needed to tackle antibiotic resistance.”

The challenge of antibiotic resistance will not disappear overnight, but the work of NCCR AntiResist shows that there are new ways to tackle it. By bringing together different perspectives on bacteria, the immune system, drug discovery, and human infection, the researchers are helping to build the knowledge and tools needed to stay ahead of resistance. A big thank you to Nina, Mattia, Carolyn, and Lucas for taking the time to share their research, experiences, and vision with us.

About NCCR AntiResist

NCCR AntiResist is a Swiss research consortium funded by the Swiss National Science Foundation (SNSF) and based at the University of Basel. Bringing together around 30 research groups from institutions across Switzerland, as well as international collaborators, NCCR AntiResist combines expertise in clinical research, infection biology, and engineering to develop new approaches to combat antimicrobial resistance.

By studying how bacterial pathogens behave under conditions that closely reflect infections in the human body, researchers aim to uncover new vulnerabilities and translate these insights into innovative antimicrobial strategies – helping to create new paths for antibiotic discovery and tackle one of today’s major global health challenges.

Find out more and follow NCCR AntiResist:
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DBM Paper Prize Winners: Mika Schneider and Hailey Kim

Each year, the DBM Paper Prize recognizes an outstanding publication by researchers from our department. Publications are nominated by DBM members and evaluated by a jury of external experts based on their scientific quality and relevance to the DBM. This year's prize goes to Hailey Kim and Mika Schneider from the Clinical Neuroimmunology lab for their publication "Myelin antigen capture in the CNS by B cells expressing EBV latent membrane protein 1 leads to demyelinating lesion formation". We spoke to them about their research, their lives beyond the lab, and where their paths have taken them since.

For Mika, growing up right next to the forest sparked an early curiosity about nature and how things work. Even Christmas and birthday wishes tended to revolve around science – from growing little green crystals to getting a microscope or telescope to explore what was otherwise beyond sight. Years later, a personal experience shifted this curiosity towards the brain and, eventually, multiple sclerosis research. An advertisement for a Master's position in the lab of Tobias Derfuss then opened the door to the DBM.

For Hailey, the path was less obvious. Becoming a scientist was never really part of the original plan. As a child, she remembers thinking that those who wanted to become scientists were very ambitious because being a scientist seemed far too difficult. Instead, it happened gradually: she simply kept following the subjects she enjoyed, from mathematics and biology to molecular biology and translational science. This path eventually brought her to the DBM, attracted by an environment where basic and clinical research interact closely and scientific questions remain connected to disease and clinical relevance.

Their paths at the DBM eventually came together in the work that would receive this year's DBM Paper Prize. The project started with a relatively simple question: what happens when potentially harmful, self-reactive B cells enter the brain and encounter their target antigen there? While the brain is considered an immune-privileged organ during homeostasis, in multiple sclerosis (MS), the central nervous system is infiltrated by lymphocytes, including B cells, which can contribute to damage of the myelin sheath surrounding neuronal axons.

During her PhD, Hailey developed experimental models to study B-cell infiltration into the brain, which allowed the team to follow autoreactive, myelin-specific B cells after a virus infection. They showed that these B cells actively engaged with and captured their target antigen within the central nervous system, but underwent apoptosis shortly afterwards due to a lack of T-cell help. This suggested an important protective checkpoint that prevents self-reactive B cells from becoming pathogenic.

The project was later extended with Mika and the rest of the team to investigate a possible connection with Epstein-Barr virus (EBV). By providing an additional survival signal through expression of the EBV protein Latent Membrane Protein 1 (LMP1) in myelin-reactive B cells, they showed that these cells could escape activation-induced cell death and bypass this critical tolerance checkpoint. This resulted in antibody secretion, complement and microglia activation, and, ultimately, demyelination reminiscent of lesions seen in patients with MS.

> **"Being exposed to many different people lets you grow as a person, not only scientifically."**
>
> **– Mika Schneider**

Mika Schneider

Mika grew up in Baselland but still calls himself a Basler – or probably a "Bebbi". At the age of six, he discovered his musical side and started playing the drums, which he gave up 12 years later due to his blossoming trumpet career. He has kept it up ever since, especially "fyr die drey scheenste Dääg". In fact, this year marked his 28th time actively contributing to Basler Fasnacht – despite having only just turned 29.

After school, Mika completed an apprenticeship as a chemical lab technician at Novartis. Not yet feeling prepared for boring adult life, he decided to study. Back then, everything seemed interesting, so he started a Bachelor's degree in nanosciences – although quantum mechanics and he never quite became friends. Two years later, he had an insightful moment and switched to molecular biology. And this is how it all began and, eventually, how he ended up at the DBM.

And something not everybody knows about Mika?

He once won a car in a Christmas light photo competition. :)

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For Hailey, one of the most exciting aspects was seeing "a relatively basic question about how B cells behave inside the brain develop into a broader story connecting B-cell biology, viral infection, and MS."

What both particularly valued about their time at the DBM were the people they met along the way. For Hailey, it was especially the sense of community. Being part of the DBM PhD Club, and eventually leading it during her final year, created many opportunities to connect with PhD students, researchers, and supervisors outside her own group. Mika remembers the DBM as a young, fresh, and dynamic environment, where people were constantly coming and going and where he got to know people from all over the world, bringing and sharing their different cultures. Some of the friendships and professional relationships built during that time have continued long after the PhD. "Research can easily become very focused on one's own experiments and project," Hailey says, making this broader community especially meaningful.

> **"Seeing a relatively basic question about how B cells behave inside the brain develop into a broader story connecting B-cell biology, viral infection, and MS."**
>
> **– Hailey Kim**

For Mika, being exposed to so many different people also had an impact beyond research: "I think this lets you grow as a person, not only scientifically."

When we asked both about their greatest achievement so far, their answers reflected not only the outcome of their work, but also the persistence it took to get there.

For Mika, completing his PhD is something he is particularly proud of. There were many frustrating periods when experiments were not working, and maintaining the motivation to keep going back and trying to optimize things over and over again was not always easy. "I think that is a big achievement in itself," he says, adding that having the *Cell* publication at the end of that time made the experience even more rewarding.

For Hailey, one of the most rewarding achievements has also been seeing her PhD research develop into the work that was eventually published in *Cell*. During her PhD, there were many moments when it was not clear where the project would go, and the work was scientifically challenging. Seeing the initial question develop over several years into a much bigger story about B cells, EBV, and MS was therefore particularly rewarding. At the same time, she stresses that a publication like this is never simply the result of one person working harder than others. It requires a good question, the right environment and collaborators, persistence – and also some luck in how the project develops.

Hailey Kim

Hailey is originally from South Korea. She was born in Seoul but had the opportunity to live, study, and work in several countries. She completed both her Bachelor's degree in Biochemistry and her Master's degree in Experimental Medicine at McGill University in Canada. After her Master's, she moved back to Korea and worked at QIAGEN Korea before coming to Switzerland for her PhD in Clinical Neuroimmunology at the University of Basel.

She is still at the University of Basel today, but now in a very different role at the Innovation Office, working mainly with life-science startups and international partners. Her career has taken her from basic science and research to industry and now innovation, with science as the common thread connecting them.

And something not everybody knows about Hailey?

When travelling, she likes to experience a place as locally as possible. Taking public transport and going grocery shopping are two things she almost always does, as everyday routines often reveal more about a place than the major tourist attractions. These ordinary experiences often end up being the most memorable part of a trip.

For her, the achievement therefore feels "less like an individual success and more like the outcome of a long scientific journey and a strong team effort."

Both have since left the DBM – Hailey some time ago and Mika only recently. We asked them what they are doing now and where they see their paths taking them next. Hailey is now based at the Innovation Office at the University of Basel, where she works with life-science startups and international partners. Her role includes helping scientists and founders position their science, connecting them with relevant academic, clinical, or industry partners, and developing international innovation programs and partnerships, particularly between the Basel ecosystem and partners across Europe and Asia. Although her work is now quite different from her PhD and no longer involves bench work, her scientific training still plays an important part. Looking ahead, she would like to continue

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working at the interface of science, innovation, and business and help strong science move beyond the laboratory.

For Mika, the transition is still ongoing. He started applying for his first industry positions around six months before defending his PhD – something he recommends to others given the currently difficult job market. He is continuing to explore opportunities both in industry and academia and hopes to gain further experience in translational research and clinical studies, with the longer-term goal of moving into industry.

Drawing on their own experiences, we finally asked them what advice they would give to young scientists hoping to build a successful career. For Hailey, an important part of finding your own path is to explore and try different things to understand what you genuinely enjoy and what matters to you. Once something feels worth pursuing, however, she believes it is equally important to stay with it and finish it, even through difficult periods. The knowledge, skills, relationships, and even failures collected along the way can become valuable later – sometimes in unexpected ways. She also advises not to define success too narrowly: "Do your best, be kind to the people around you, and keep learning from what happens."

> **"Do your best, be kind to the people around you, and keep learning from what happens."**
>
> **– Hailey Kim**

Mika's advice is more practical: plan ahead and structure your daily schedule. Before starting an experiment, he suggests asking: What is the question? How will the experiment help answer it? How long will it take, and are all the necessary machines available? Once everything is clear, it is time to get started. Or, in his own words: "Wrap up, analyze, optimize, write, publish, success! :-)"

> **"Wrap up, analyze, optimize, write, publish, success!"**
>
> **– Mika Schneider**

We thank Hailey and Mika for sharing their stories with us, congratulate them once again on receiving the DBM Paper Prize, and wish them both every success wherever their paths take them next.

Hailey Kim, Ivan Martin and Mika Schneider.

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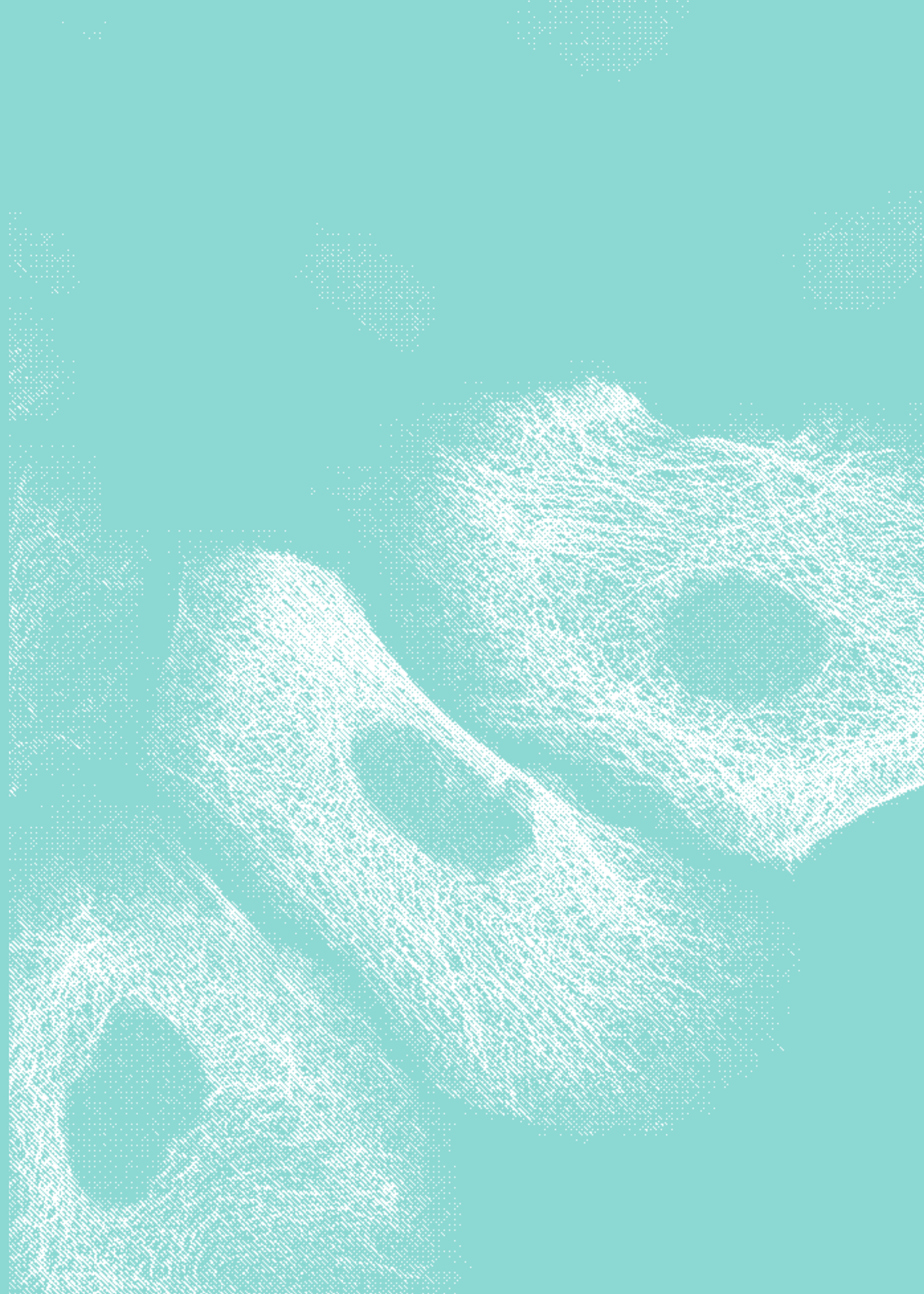

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A Quick Overview of Our Research

Systemic lupus erythematosus (SLE) is the prototype of an autoimmune disease and can involve any organ system – eventually leading to comorbidities that can also be observed independently of underlying SLE. The complex pathogenic mechanisms leading to and being involved in this autoimmune-inflammatory syndrome are not well understood. However, complement C1q, the first component of the classical pathway, seems to play a central role. By analyzing the role of C1q as well as its interaction with autoantibodies targeting C1q (anti-C1q) in SLE, we aim at elucidating: 1) mechanisms being involved in the initiation of autoimmunity, 2) mechanisms of secondary acceleration of inflammation, and 3) processes being associated with the development of atherosclerosis and thromboembolism.

Highlights, Breakthroughs, and Current Projects

An important part of our past research activity focused on the role of complement C1q, the starter molecule of the classical pathway, and anti-C1q in systemic autoimmunity.

One fundamental finding was the demonstration of a strong correlation of anti-C1q with the occurrence of severe nephritis in patients with SLE. Analyses of these anti-C1q showed that they are the consequence of an antigen-driven immune response and specifically target C1q bound on early apoptotic cells.

We then aimed at understanding mechanisms driving the autoimmune response, and identifying pathogenic mechanisms occurring after the binding of anti-C1q. We were able to identify a major linear cryptic epitope of C1q as a target for a significant fraction of anti-C1q. This cryptic epitope is located on the collagen-like tail of the C1q A-chain. When exploring mechanisms that could cause the formation of autoantibodies to this antigenic site, we most recently showed that anti-C1q can be induced by an Epstein Barr Virus (EBV)-derived epitope through molecular mimicry. This adds to the understanding of SLE supporting the pathogenic role of EBV in the development of the disease. Considering the fact that the large C1q molecule is highly functional and plays a role in many processes, we believe that epitope-specific antibodies in patients with SLE are associated with specific clinical aspects of the disease, i.e. epitope-specific effects of anti-C1q that could be explained by the different functional roles of the targeted epitopes.

Regarding downstream effects of anti-C1q binding, we could show that SLE patient-derived anti-C1q can activate the classical pathway of complement. This provides a direct link to hypocomplementemia – frequently observed in these patients in particular in the context of renal flares. In addition, we could show that anti-C1q induces a proinflammatory phenotype in monocytes and macrophages being associated with reduced phagocytic capacity. Indeed, ineffective clearance of dead and dying cells is considered to be a driver of SLE. Anti-C1q might, therefore, be an important driver of secondary disease acceleration in patients with SLE. Furthermore, using our human *in vitro* model of autoimmunity, we found that activated T cells can trigger the inflammatory responses of monocytes/macrophages via CD40, while they can be reduced by the inhibition of Cathepsin S. Thus, we described the basis for a potential future treatment approach.

Our current projects aim at understanding the functional role of the antigenic site 'A08' of C1q and developing potential therapeutics that on the one hand keep the primary functionality of C1q intact but on the other hand inhibit its assumed detrimental downstream effects.

Clinical Immunology (from left to right): Marten Trendelenburg, Denise Dubler, Juliane Klehr and Kristina Schulz.

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Our Vision for the Future

SLE has the highest incidence rates in high-income countries. It has an estimated prevalence of about 40 cases per 100'000 people in Central Europe, and has shown a trend to further increase in recent years. Despite significant improvements in the last decades, patients with SLE still have high morbidity, and their mortality is 2-3 times higher than in the general population. SLE is one of the leading causes of death in young women. Thus, understanding the complex pathogenic mechanisms leading to SLE and maintaining the autoimmune-inflammatory process with the aim of improving our therapeutic options by the development of more targeted therapies are pivotal. With this overall goal in mind and because complement C1q is well described to play an important role in systemic autoimmunity, we aim at extending our knowledge on the functions of complement C1q fostering new strategies for targeted therapies.

Team Spirit – Introduction of Our Team

An essential part of our research group is the translational aspect of our work. We are a diverse team consisting of people with different backgrounds and perspectives leading to lively discussions and inputs, sometimes best if there is coffee or lunch. In addition, the research group lives due to the technical and scientific expertise of our scientists, technicians, and PhD students which is a fundamental basis for addressing our questions. On the other hand, the integration of MDs and MD students as well as the access to patient data and blood samples thanks to the Swiss SLE Cohort Study (SSCS) enables us to bring clinically relevant questions to the laboratory. This connection also helps interpreting results from experiments and developing new research questions. The goal is to create an inspiring and successful scientific environment.

Clinical Immunology (from left to right): Stefan Moser, Eylül Tuncer, Denise Dubler, Peymane Zahiroddini, Juliane Klehr, Marten Trendelenburg and Jasmin Schulz.

Our Group Members

Marten Trendelenburg	Research Group Leader
Denise Dubler	Laboratory Technician
Juliane Klehr	Postdoc
Stefan Moser	Research Associate
Michael Osthoff	Project Leader
Elisa Pons	MD Candidate
Kristina Schulz	Postdoc

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Research Group at a Glance
Pathology of Infectious and Immunologic Diseases
Mertz Lab

A Quick Overview of Our Research

What unites us is a shared fascination for infections and the complex interactions between pathogens, human tissues and the immune system. What happens when a pathogen meets human tissue? This deceptively simple question is at the heart of our research. We study infectious diseases directly where they happen: in human tissues. Our aim is not only to identify which pathogen is present, but to understand where it is, how it interacts with its host, why it persists, and how the surrounding immune response determines disease. To achieve this, we combine classical pathology with cutting-edge molecular and spatial technologies, including metagenomic next-generation sequencing, targeted sequencing, host transcriptomics, spatial transcriptomics and proteomics, multiplex imaging, digital pathology and computational approaches. A particular strength of our group is the development of methods that work with formalin-fixed, paraffin-embedded (FFPE) tissue – the material routinely collected from patients around the world and stored in pathology archives for decades. This gives us access to an extraordinary biological resource and allows us to investigate rare, unexpected and previously unexplained infectious diseases. Our work spans three closely connected areas: Translational & Diagnostic Infection Pathology, Experimental Infection Biology and Technology Development.

Highlights, Breakthroughs, and Current Projects

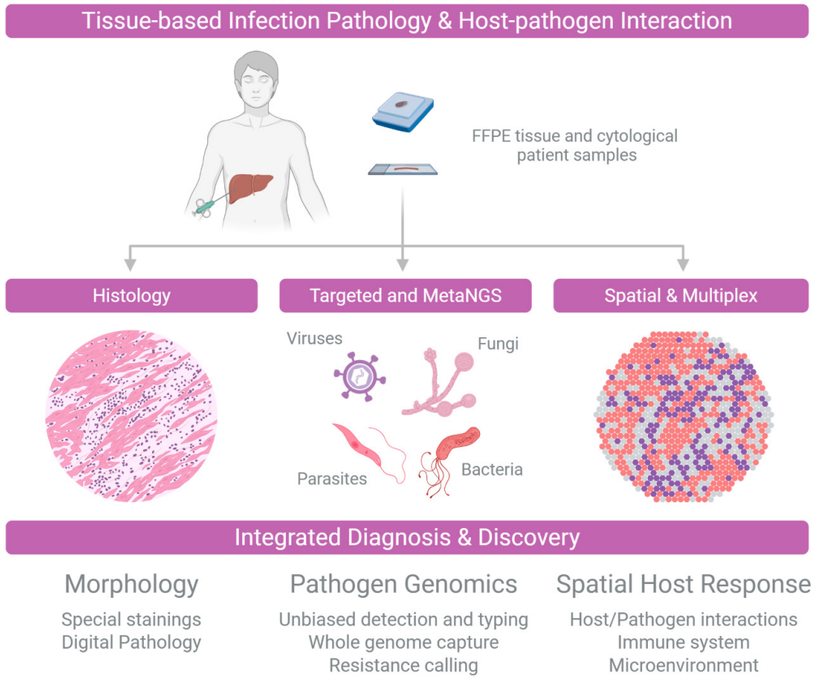
One of our major achievements has been to establish tissue metagenomics for routine diagnostic pathology. In one of the largest real-world cohorts of its kind, we investigated more than 600 FFPE tissue samples using unbiased metagenomic sequencing. This approach enables us to search simultaneously for bacteria, fungi, parasites and DNA viruses – including pathogens that were not initially suspected. And sometimes, the tissue surprises us.

Our work has contributed to the identification and characterization of rare and unexpected infections, including a novel human circovirus associated with hepatitis, *Borrelia afzelii* hepatitis in an immunocompromised patient, unusual adenovirus-associated pneumonia, and rare bacterial and fungal infections. These cases illustrate why looking broadly – rather than testing only for the pathogen we expect – can be so powerful.

Another major focus of our work is granulomatous inflammation. Granulomas occur in tuberculosis and many other infectious and inflammatory diseases, yet morphologically similar granulomas can represent fundamentally different biological processes. We are combining spatial transcriptomics, multiplex imaging, molecular pathogen detection and computational pathology to build a molecular and spatial map of human granulomas – ultimately asking whether we can redefine these lesions by their underlying biology rather than morphology alone.

We are also developing tissue-based precision diagnostics for *Helicobacter pylori*, allowing simultaneous detection of the bacterium together with antibiotic-resistance mutations and virulence factors directly from routine gastric biopsies – without the need for culture.

Across all these projects, we are particularly interested in persistent infections, infections in immunocompromised patients, emerging and unexpected pathogens and infection-associated immune dysregulation.



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Our Vision for the Future

Our long-term vision is to make the tissue itself a central source of information in infectious disease diagnostics and research. We want to move from simply asking "Is a pathogen present?" towards understanding "What is happening between this pathogen, this tissue and this particular patient's immune system?"

By integrating pathogen genomics with host responses, tissue morphology and spatial information, we hope to create a new generation of tissue-based precision diagnostics for infectious diseases.

At the same time, we are building a strong platform for Infection Pathology in Switzerland, connecting pathology with infectious diseases, microbiology, immunology, genomics and computational biology. Our ambition is to provide expertise for difficult and unexplained cases, develop and validate new diagnostic technologies, and use the extraordinary wealth of information contained in human tissue to discover new disease mechanisms.

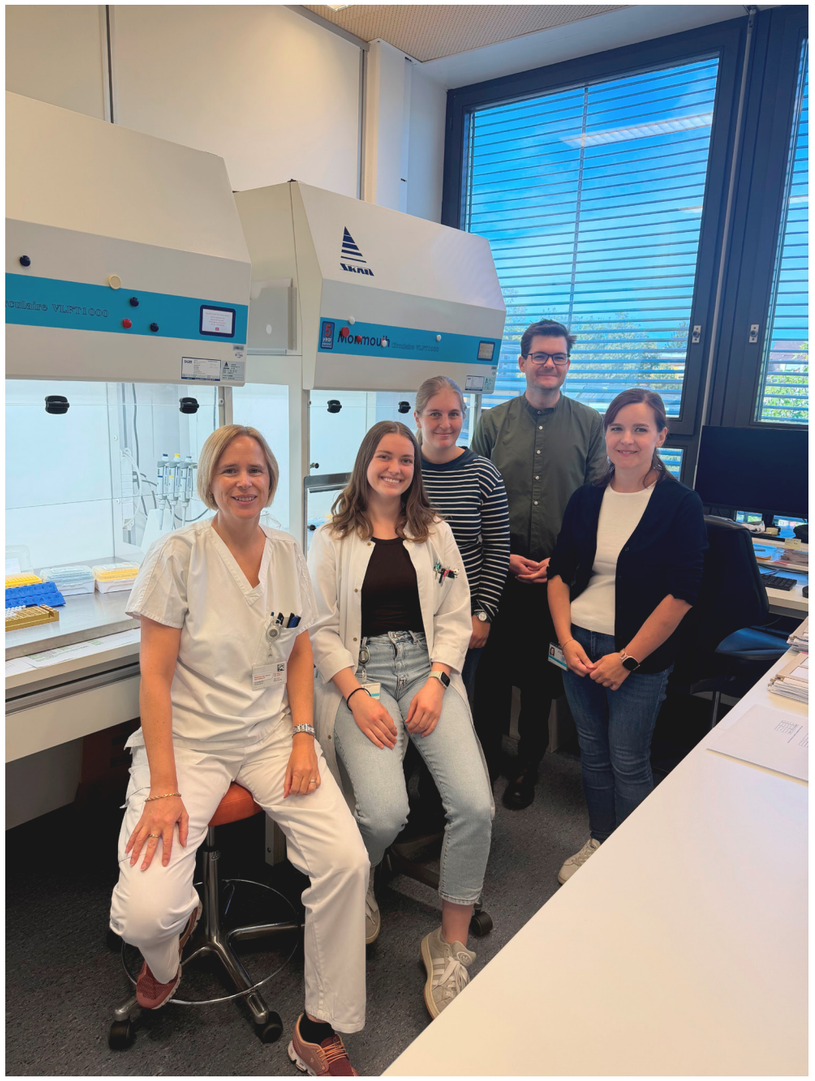
Ultimately, our goal is simple: to turn discoveries made in tissue into better diagnoses, deeper biological understanding and, eventually, better care for patients with infectious and infection-associated diseases.

Team Spirit – Introduction of Our Team

We are a curious, enthusiastic and highly interdisciplinary team. Pathologists work alongside microbiologists, molecular biologists, bioinformaticians and experimental scientists – and everyone brings a different perspective to the same question.

We particularly enjoy the cases and projects where the answer is not obvious. An unexpected sequencing result, an unusual pattern under the microscope or a puzzling clinical case often becomes the starting point for a new scientific question.

Our group thrives on discussion, collaboration and the freedom to follow surprising observations. We believe some of the most interesting science happens at the boundaries between disciplines – and that it is much more rewarding when discoveries are made together. We look for pathogens and study the host. And we let the tissue tell us the story.



Pathology of Infectious and Immunologic Diseases (from left to right): Barbara Klaus-Wirthner, Sarah Rubin, Salome Hosch, Baptiste Hamelin and Maria Mancuso.

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Our interdisciplinary team brings together expertise in pathology, microbiology, molecular diagnostics, infection biology, bioinformatics and spatial technologies. We work closely with clinical and basic-science partners in Basel, throughout Switzerland and internationally. This interdisciplinary environment is essential to what we do: bringing observations from patient tissues into the laboratory – and new discoveries back into diagnostics and patient care.

Our Group Members

Kirsten Mertz	Research Group Leader
Baptiste Hamelin	Postdoc
Jasmin Haslbauer	Pathologist, Clinician Scientist
Salome Hosch	Postdoc
Maria Teresa Mancuso	Laboratory Technician
Rachel Pearson	Postdoc
Sarah Rubin	Laboratory Technician
Maria Tapken	Pathology Resident

Pathology of Infectious and Immunologic Diseases
(upper row, from left to right): Kirsten Mertz, Baptiste Hamelin, Regula Bernhard and Melanie Sachs.
(lower row, from left to right): Nadine Mensah, Salome Hosch, Jasmin Haslbauer and Maria Mancuso.

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All publications we have received from the period between May and September 2026. The publications are ordered by date of publication.

Chronic infection perturbs the affinity hierarchy of antiviral B cells

Dimitrova M, Abreu-Mota T, Fixemer J, Bonilla WV, Marx AF, Lu M, Tintignac K, Kastner AL, Ertuna YI, Florova M, Ciancaglini M, Narr K, Stauffer K, Roux J, Demougin P, Wagner I, Merkler D, Pinschewer DD.

Proc Natl Acad Sci U S A. 2026 Apr 7;123(14):e2532422123.
doi: 10.1073/pnas.2532422123.

Pathway-selective signaling of serotonergic psychedelics at 5-HT receptors: Implications for psychoactivity, safety, and therapeutic potential

Rudin D, Valenta J, Rolli H, Liechti ME, Luethi D.

Eur Neuropsychopharmacol. 2026 Aug;109:112835.
doi: 10.1016/j.euroneuro.2026.112835.

Open-source bioreactor delivers electrical and perfusion stimulation supporting 3D cardiac engineered tissue maturation

Reid G, Gabetti S, Sileo A, Fusco D, Isu G, Massai D, Milan G, Marsano A.

Bioeng Transl Med. 2026 Apr 13;11(4):e70145.
doi: 10.1002/btm2.70145.

Memory T cell aging and rejuvenation

Adamo S, Rurik JG, Gustafson CE, Buggert M.

Immunity. 2026 Apr 14;59(4):878-896.
doi: 10.1016/j.immuni.2026.02.018.

Neural correlates of emotional memory enhancement: The role of valence and arousal

Amini E, Coynel D, Papassotiropoulos A, de Quervain DJ.

Imaging Neurosci (Camb). 2026 Apr 17;4:IMAG.a.1213.
doi: 10.1162/IMAG.a.1213.

Network-level neural basis of individual differences in emotional memory enhancement

Geissmann L, Coynel D, Papassotiropoulos A, de Quervain DJ.

Neuroimage. 2026 Jun;333:121941.
doi: 10.1016/j.neuroimage.2026.121941.

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Helicobacter pylori whole genome enrichment and sequencing for resistome and virulome characterization in gastric biopsies

Hosch S, Hamelin B, Neidhöfer C, Pearson RA, Rubin SE, Mancuso M, Beckmann C, Leuzinger K, Keller PM, Mertz KD.

Precis Pathol. 2026 May;1:100001.
doi: 10.1016/j.prpath.2026.100001.

Uncovering immune dysfunction in ACLF: Cellular mechanisms, molecular pathways, and therapeutic frontiers

Ortega-Ribera M, Brenig R, Bernsmeier C, Szabo G.

J Hepatol. 2026 Sep;85(3):612-629.
doi: 10.1016/j.jhep.2026.04.025.

Preexisting IgG forms immune complexes and links local thermal reactogenicity with immunogenicity in influenza vaccination

Hirsiger JR, Scarascia S, Recher M, Bantug G, Berger CT.

NPJ Vaccines. 2026 May 2;11(1):137.
doi: 10.1038/s41541-026-01477-x.

Mendelian randomization identifies lipidomic signatures of depression risk that are partly reflected in cortisol-induced membrane remodeling and modulated by St. John's wort extract (Ze 117)

Freytag V, Butterweck V, de Quervain DJ, Boonen G, Papassotiropoulos A.

Int J Mol Sci. 2026 May 13;27(10):4344.
doi: 10.3390/ijms27104344.

Neuregulin-1β mitigates doxorubicin-induced cardiotoxicity via Serping1 in cardiac fibroblasts

Aghagolzadeh P, Xu L, Klinger P, Morandi C, Lépine LM, Minder L, Guns PJ, Bosman M, Coissieux MM, Pedrazzini T, Kania G, Brink M.

Int J Mol Sci. 2026 May 21;27(10):4616.
doi: 10.3390/ijms27104616.

Structural connectivity of functionally defined episodic memory networks: A large-scale connectome-behavior study

Ghasemi N, Coynel D, Papassotiropoulos A, de Quervain DJF.

Brain Behav. 2026 Jun;16(6):e71515.
doi: 10.1002/brb3.71515.

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Cytotoxic CD39+ tumor-associated NK cells respond to NKG2A blockade in lung cancer

Serger C, Rebuffet L, Sandholzer MT, Rackwitz W, Fusi I, Herzig P, Brüggemann A, Pawlow CA, Chichelnitskiy E, Hajnal D, Oelgarth N, Uzun S, Schultheiß C, Zingg A, Tundo S, Luu TT, Hojski A, Lardinois D, Neubert L, Binder M, Mertz K, Trefny MP, Kirchhammer N, Natoli M, Matter MS, Läubli H, Falk C, Schaeuble K, Vivier E, Romagnani A, Zippelius A.

Sci Immunol. 2026 Jun 5;11(120):eaeb6645.
doi: 10.1126/sciimmunol.aeb6645.

A fully human engineered bone niche with endogenous osteoclastogenesis reveals osteoclast-dependent osteomimicry in prostate cancer cells

Mazzoleni A, Dolgos R, Menter T, Dasen B, Scherberich A, Le Magnen C, Muraro MG, Martin I.

Adv Healthc Mater. 2026 Jul;15(26):e05893.
doi: 10.1002/adhm.202505893.

Mice produce interneurons in the septum as a response to aversive experiences and antidepressant treatment

Lampada A, Rolando C, Whittle N, Engler A, Tillmann J, Freire JA, Giachino C, Parmigiani E, Saotome I, Louvi A, Gründemann J, Lüthi A, Taylor V.

Sci Adv. 2026 Jun 12;12(24):eaed3625.
doi: 10.1126/sciadv.aed3625.

Efficacy and safety of ofatumumab in participants with relapsing multiple sclerosis and breakthrough disease on oral fingolimod or fumarates: Results from the ARTIOS study

Bove R, Langdon D, Maciejowski M, Jasińska E, Dufek M, Abeyewickreme A, Rauh A, Fu H, Khan IA, Böhringer M, Eichau SM, Derfuss T.

J Neurol. 2026 Jun 29;273(7):434.
doi: 10.1007/s00415-026-13960-5.

Heart failure with preserved ejection fraction: The role of intravascular volumes and body composition in exercise-induced progenitor cell mobilization

Kröpfl JM, Schoch R, Gasser B, Prechtl L, Gruber HJ, Wick R, Dieterle T, Brink M, Schmidt-Trucksäss A.

Int J Cardiol Heart Vasc. 2026 Jun 29;65:101967.
doi: 10.1016/j.ijcha.2026.101967.

OncoMimic peptide-based immunotherapy EO2401 induces shared T cell clusters in patients with adrenal cancer

Schultheiß C, Besemer B, Willscher E, Paschold L, Merscerman T, Talpin A, Serger C, Zippelius A, Berruti A, Grisanti S, Menke-van der Houven van Oordt CW, Baudin E, Landwehr LS, Capdevila J, Subbiah V, Granberg D, Gedske Daugaard K, Triebig A, Gauduchon T, Do Cao C, Garcia ME, Magalhaes J, Chêne L, Binder M.

Commun Med (Lond). 2026 Jul 6.
doi: 10.1038/s43856-026-01755-8.

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A tumor profiling resource for ovarian cancer: Insights into chemotherapy-driven heterogeneity and personalized treatment strategy

Jacob F, Wegmann R, Ficek-Pascual J, Lischetti U, Kuipers J, Chevrier S, Prummer M, Toussaint NC, Alborelli I, Coelho R, Casanova R, Goetze S, Gut G, Baciu-Drăgan MA, Bertolini A, Bonilla X, Brugger J, Brune MM, Calgua B, Engler S, Esposito C, Ferreira PF, Jacobs A, Lombardo FC, Malsot P, Mena J, Miglino N, Milani ES, Sarabia Del Castillo J, Sivapatham S, Sobottka-Brillout BA, Tanna T, Zwimpfer TA; TumorProfiler Consortium; Aebersold R, Bacac M, Beerenwinkel N, Beisel C, Bodenmiller B, Koelzer VH, Lehmann KV, Levesque MP, Moch H, Muenst S, Pelkmans L, Manz MG, Rätsch G, Singer F, Snijder B, Theocharides APA, Tolnay M, Wicki A, Wollscheid B, Heinzelmann-Schwarz V.

Nat Commun. 2026 Jul 13;17(1):8624.
doi: 10.1038/s41467-026-74585-w.

Anti-PD1-IL18 immunoconjugate promotes effector T cell-mediated antitumor immunity

Oelgarth N, Martin K, Junker F, Serger C, Herr C, Buchi M, Gremlich L, Fusi I, Fürst J, Herzig P, Moosmann P, Heinzelmann-Schwarz V, Mertz KD, Rosenberg R, Schaeuble K, Carralot JP, Luu TT, Kreft B, Pattabiraman V, Zippelius A.

J Immunother Cancer. 2026 Jul 14;14(7):e015090.
doi: 10.1136/jitc-2026-015090.

Anti-CD20 B cell depletion is associated with elevated mucosal-originating circulating regulatory IgA B cells in multiple sclerosis

Neziraj T, Pössnecker E, Wang AA, Saary P, Schumacher AM, Ingelfinger F, Mathias A, Galli E, Häfelfinger M, Zuo M, Jones S, Pantazou V, Killestein J, Schädelin S, Benkert P, Granziera C, Pot C, Bischof A, Niess JH, Filipowicz Sinnreich M, Derfuss T, Teunissen CE, Kuhle J, Hafler DA, Du Pasquier R, Yasumizu Y, Gommerman JL, Pröbstel AK.

Sci Transl Med. 2026 Jul 22;18(859):eaee1580.
doi: 10.1126/scitranslmed.aee1580.

RYR1 mutations linked to malignant hyperthermia susceptibility are associated with phenotypic changes in human B-lymphocytes

Li H, Andrade PV, Ceren IH, Ruiz A, Silva AI, Farias MF, Zorzeto TS, Vainzof M, Zorzato F, Silva HCA, Treves S.

Front Immunol. 2026 Jul 27;17:1876709.
doi: 10.3389/fimmu.2026.1876709.

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Serum glial fibrillary acidic protein dynamics, disease progression, and therapy response in multiple sclerosis

Einsiedler M, Sandgren S, Schaedelin S, Ning K, Maleska Maceski A, Oechtering J, Cordano C, Melie-Garcia L, Gelfand JM, Cagol A, Henry RG, Finkener S, Lalive PH, Müller S, Pot C, Mathias A, Du Pasquier R, Hoepner R, Chan A, Disanto G, Zecca C, D'Souza M, Hemkens LG, Yaldizli Ö, Fischer-Barnicol B, Derfuss T, Roth P, Herwerth M, Gobbi C, Brassat D, Tackenberg B, Pedotti R, Wiendl H, Berger K, Hermesdorf M, Arrambide G, Piehl F, Zetterberg H, Cree BAC, Sormani MP, Kappos L, Hauser SL, Khalil M, Granziera C, Green AJ, Leppert D, Benkert P, Abdelhak A, Kuhle J; Expression, Proteomics, Imaging, Clinical (EPIC) study and the Swiss MS Cohort (SMSC) investigators; Hofer L, Hughes M, Genc N, Zadic A, Vilchez Gomez JF, Galbusera R, Galli E, Müller J, Uginet M, Kana V, Raposo C, Oksenberg J, Conen D.

JAMA Neurol. 2026 Aug 3:e262500.
doi: 10.1001/jamaneurol.2026.2500.

TCR γδ cell-specific STAT5 gain of function induces a druggable chronic human immune dysregulation

Meyer BJ, Loureiro JP, Nosi V, Hupfer R, Ghosh A, Poletti F, Jauch A, Hirsiger J, Berkemeier C, Heijnen I, Dirks J, Alborelli I, Menter T, Tzankov A, Hess C, Navarini AA, Berger CT, Mori L, De Libero G, Recher M.

J Hum Immun. 2026 Aug 10;2(5):e20250175.
doi: 10.70962/jhi.20250175.

Rapid and long-lasting remodelling of the blood transcriptome following bariatric surgery

Roux J, Seyres D, Buxton JL, Handley D, Hofer G, Alsters SI, Herzig KH, Järvelin MR, Blakemore AI, Cavelti-Weder C.

Int J Obes (Lond). 2026 Aug 19.
doi: 10.1038/s41366-026-02196-y.

Hepatocellular carcinoma organoids enable drug discovery and combination therapy across heterogeneous tumors

Nuciforo S, Blukacz L, Heusler MA, Booij TH, Kübler L, Trulsson F, Boldanova T, Wang X, Ketterer S, Matter MS, Stirnimann CU, Wieland S, Heim MH.

Cell Rep. 2026 Aug 24;45(9):117895.
doi: 10.1016/j.celrep.2026.117895.

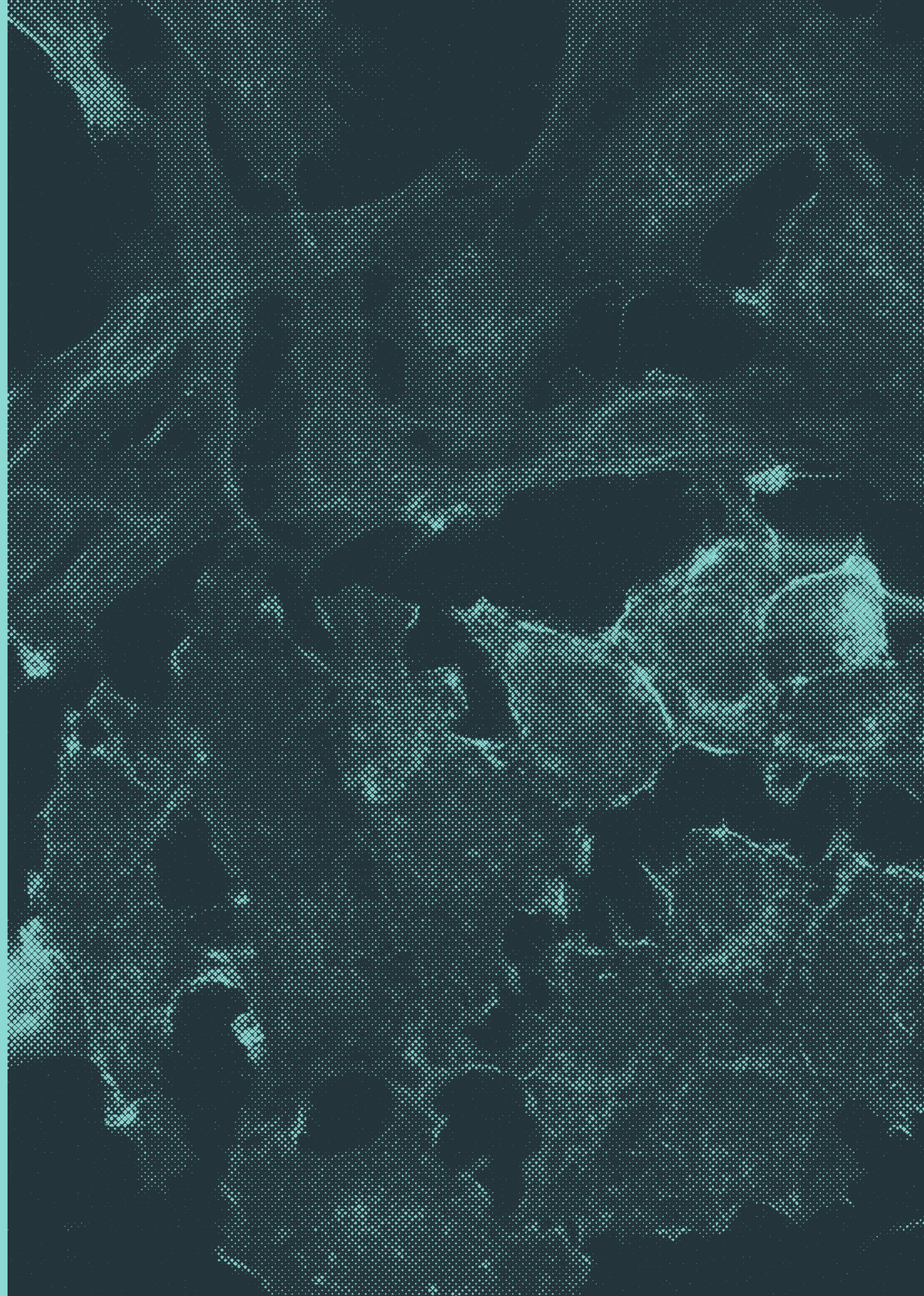
Bifunctional Phagocytic Synapse Enhancers Remodel the Tumor Microenvironment to Overcome Immunosuppression

Sabatino V, Tang C, Acúrcio RC, Coelho AR, Müller FM, Fontes M, Khaw WT, Labão-Almeida C, Hicks DR, Yang W, Vasco Vidal A, Sarkar I, Shome D, Cabeza-Cabrerizo M, Rohm TV, Fleisch S, Gerber A, Hogan SA, Gerster F, Kaymak D, Pantelyushin S, Vom Berg J, Li L, Reijmers RM, Fior R, Baker D, Florindo HF, Hutter G, Bernardes GJL.

Cancer Res. 2026 Sep 4.
doi: 10.1158/0008-5472.CAN-25-5578.

We acknowledge Andrea Banfi for his contribution and dedication to the publication list.

DBM
Basel
Department of
Biomedicine



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The DBM Congratulates

Awards since May 2026

We extend our heartfelt congratulations to the following DBM members for their remarkable awards and achievements since May 2026.

Maurizio Cortada, **Soledad Levano**, and **Daniel Bodmer** won the first prize for their poster presentation, "Hair cell loss – cause or consequence of hearing loss?" at the Swiss ENT Society (SGORL) annual spring meeting.

Sandra Kessler has been awarded an SNF mobility grant, for her project, "Neurodevelopmental dysregulation as a driver of cellular plasticity and immune evasion in glioblastoma."

The Novo Nordisk Foundation will support **Ivan Martin** and **Sébastien Pigeot** in their three-year project, "Ossigel: a human engineered tissue-graft for skeletal regeneration."

Ivan Martin and **Andrea Barbero** received an Innosuisse grant for their work on "CARMA: Cartilage Manufacturing Automation in closed system."

Benjamin Thiele was successful in applying to the Propatient Forschungsstiftung for funding his research project "Neue Biomarker für das Monitoring des Glioblastoms."

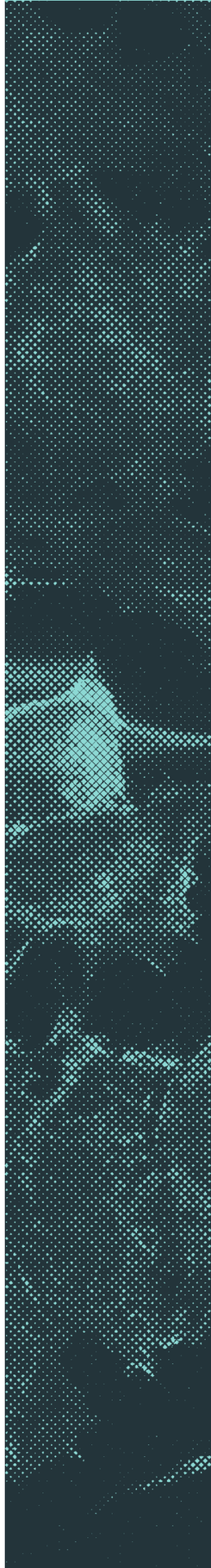
The Geistlich-Stucki Stiftung is supporting **Benjamin Thiele** and his work, titled "Reprogramming the Glioblastoma Myeloid Landscape: Siglec-Anchored Combination Immunotherapy Guided by Spatial Biomarkers and Brain-Penetrant Delivery."

This year's recipients of the Dora Seif Prize for Cancer Research are **Judith Zaugg** and **Heinz Läubli**! Every two years, the Dora Seif Foundation awards its Cancer Research Prize for "the best work that contributes to improving the early diagnosis and treatment of cancer." The award recognizes outstanding achievements in basic and clinical cancer research.

Petya Apostolova and **Karen Dixon** were both awarded European Research Council Starting Grants. These awards recognize the excellence of their research and will allow them to explore new ideas in the coming years.

The ISREC Foundation awarded two TANDEM grants to collaborative cancer research projects involving DBM investigators and external partners. One grant went to **Petya Apostolova** together with **Nina Cabezas-Wallschweid** from ETH Zurich; the second was awarded to **Kirsten Mertz**, **Alfred Zippelius**, and **Karin Schäuble** together with **Grégory Verdeil** from the University of Lausanne. The projects address significant challenges in cancer treatment, such as overcoming immune evasion in acute myeloid leukemia and determining why T cells lose their anti-tumor function in solid tumors.

David Bargiela received an SNSF Ambizione Grant for his project, "Metabolic determinants of treatment-driven tumour adaptation in glioblastoma multiforme (GBM)."



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New Krebsliga Schweiz Funding

Personalized Treatment for Liver Cancer
Markus Heim
Start: 01.07.2026 | End: 30.06.2028

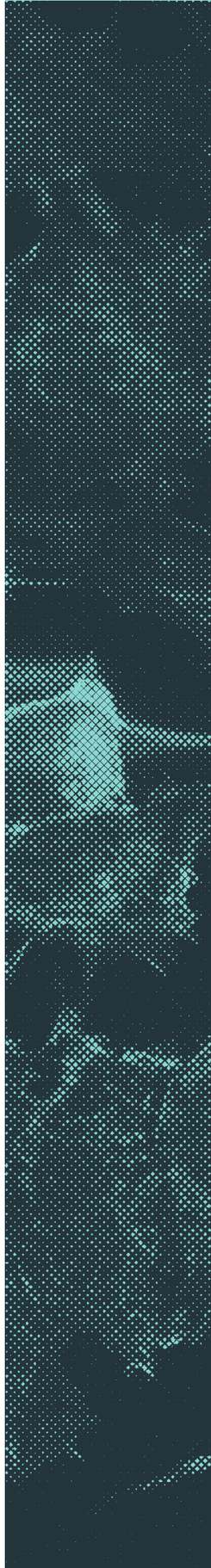
Heterogeneous bone marrow niches shape leukemic persistence and relapse in acute myeloid leukemia
Judith Zaugg
Start: 01.07.2026 | End: 30.06.2029

Fellowship: SPARC-OCS – Systematic Precision Analysis of Rare Cancers – Ovarian Carcinosarcoma: Identifying drivers of aggressiveness and potential therapeutic targets
Ruth Stefanie Eller
Start: 01.09.2026 | End: 31.08.2028

Patient-centered discovery of EBV-specific T cell receptors in EBV associated malignancies for precision immunotherapy
Nina Khanna
Start: 01.11.2026 | End: 31.10.2030

PhD Defenses since May 2026

02.06.2026	Medical-Biological Research	Dennis Strobbe
04.06.2026	Medical-Biological Research	Peymanehsadat Zahiroddini
16.06.2026	Molecular Biology	Raphael Schmid
22.06.2026	Medical-Biological Research	Johanna Nimmerfroh
16.07.2026	Cognitive Neuroscience	Ehssan Amini
13.08.2026	Molecular & Biological Sciences	Gangyu Zhang



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Athena's Journey Summer Apéro

August 20, 2026

This year's Athena's Journey Summer Apéro was yet another charming get-together. Almost 100 people gathered together in the Kollegienhaus to attend a lively panel discussion titled "Connecting Research and Innovation: Women at the Interface of Academia and Industry" featuring Carolyn King, Luisa Deberle, and Katyayanee Neopane. After the discussion, everybody was invited to join the apéro and given the opportunity to connect with the speakers, women in science, supporters, past Athena's Journey and TWIST speakers, and other members of the growing community. The whole event created a warm and very active atmosphere that encouraged personal exchanges and new connections.

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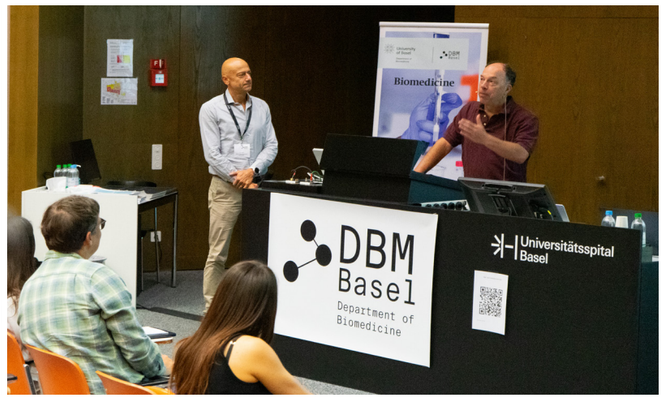
DBM Summer Symposium

September 9, 2026

The Summer Symposium marks another highlight of the academic calendar. And what a program we had for 2026! Keynote speaker Stephen Quake, from the Department of Biosystems Science and Engineering, talked about "Medical Innovations from the Genome Revolution". This was followed by four scientific sessions, including nearly twenty talks, and an award ceremony. The presenters at the sessions ranged from PhD students and postdocs to last year's awardee. The whole morning represented the diverse biomedical interests of the DBM. We are very proud of all our speakers. The DBM Paper Prize winners, Hailey Kim and Mika Schneider, are featured in this issue. And everybody present will vividly remember our Best Presentation awardee: Dorssa Akbari and her talk about, "Rare germline SAMHD1 SAM-domain mutation associated with autosomal dominant type I interferonopathy".



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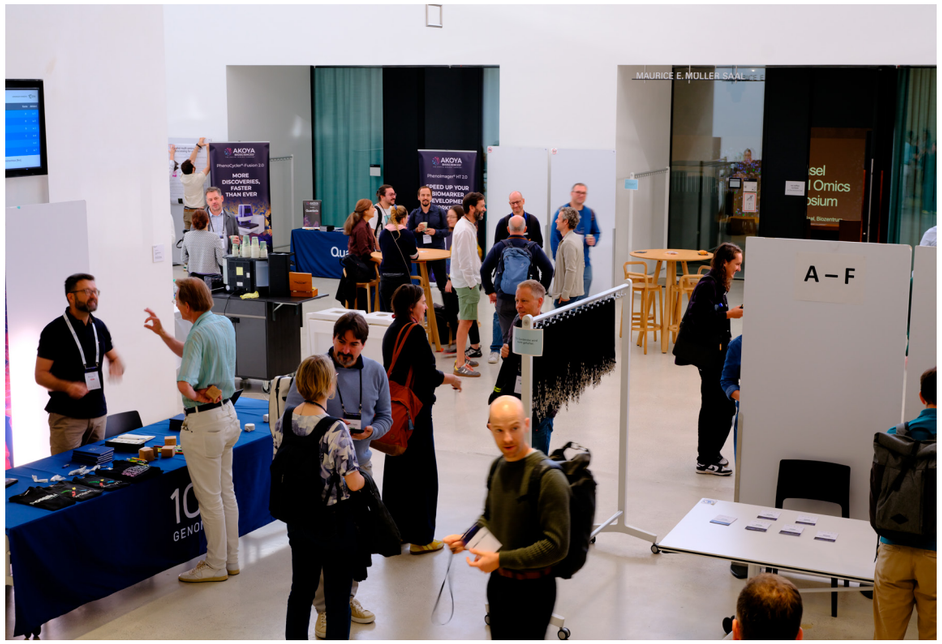
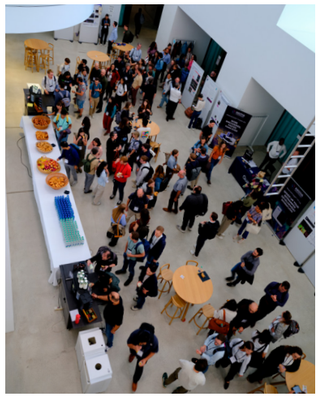
Spatial OMICS Symposium

September 11, 2026

Basel welcomed the Spatial Omics community, building on a journey that started three years ago with the Microscopy Network Basel. Similar to last year's major success, the 3rd Basel Spatial Omics Symposium was sold out quickly. Keynote speaker Mats Nilsson from SciLifeLab, Sweden, opened the program with his talk on "Spatial transcriptomics by in situ sequencing". After that, there were ten oral presentations plus poster presentations and panel discussions. The speakers came from Basel institutions, including the Biozentrum, DBM, D-BSSE, FMI, IOB, USB, Novartis, and Roche. This whole day of vibrant presentations and lively discussions was only possible due to the support of the following sponsors: 10X Genomics and Illumina, ariadne.ai, AKOYA, Element Biosciences, bio-techne Spatial, Leica, Novogene, Takara, Zeiss, and the Nikon BioImaging Lab. The relentless work and energy of the cross-institutional organizing committee, led by Sheida Hadji Rasouliha (Biozentrum), Ewelina Bartoszek (DBM) and Judith Zaugg (DBM), also made this event a big success. Spatial Omics vibrated.





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Third DBM Core Facilities Retreat: Strengthening Collaboration and Communication

September 14, 2026

For the third time, the DBM Core Facilities held a joint retreat to strengthen collaboration, exchange ideas, and plan for the future. The program focused on improving internal collaboration and user support with M365, raising the profile of the Core Facilities, and planning the next Core Facility User Meeting. Andrea Ottolini-Voellmy gave the welcome speech and led through the first informative session: exploring opportunities for enhanced teamwork using the new M365 tool and focusing on improving internal cooperation and streamlining user support. Xiomara Banholzer joined the retreat to discuss new promotional materials, including individual roll-ups featuring professional photographs of each facility. Christophe Kunz provided an update on the new DBM building and the future locations of the Core Facilities. After lunch, the team participated in Basel Tourism's guided city walk "Basel for Insiders." The tour featured curious, unbelievable, and amusing facts found in chronicles, archives, and eyewitness reports. Thank you to everyone who contributed to the retreat, especially to Claudia Vogt, who organized the day.

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Explore the Lab

20.10.2026

DBM Kids Day

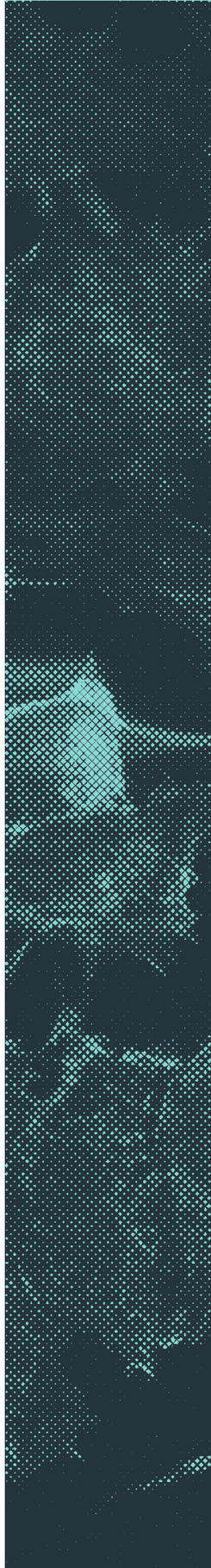
12.11.2026

DBM Xmas Dinner

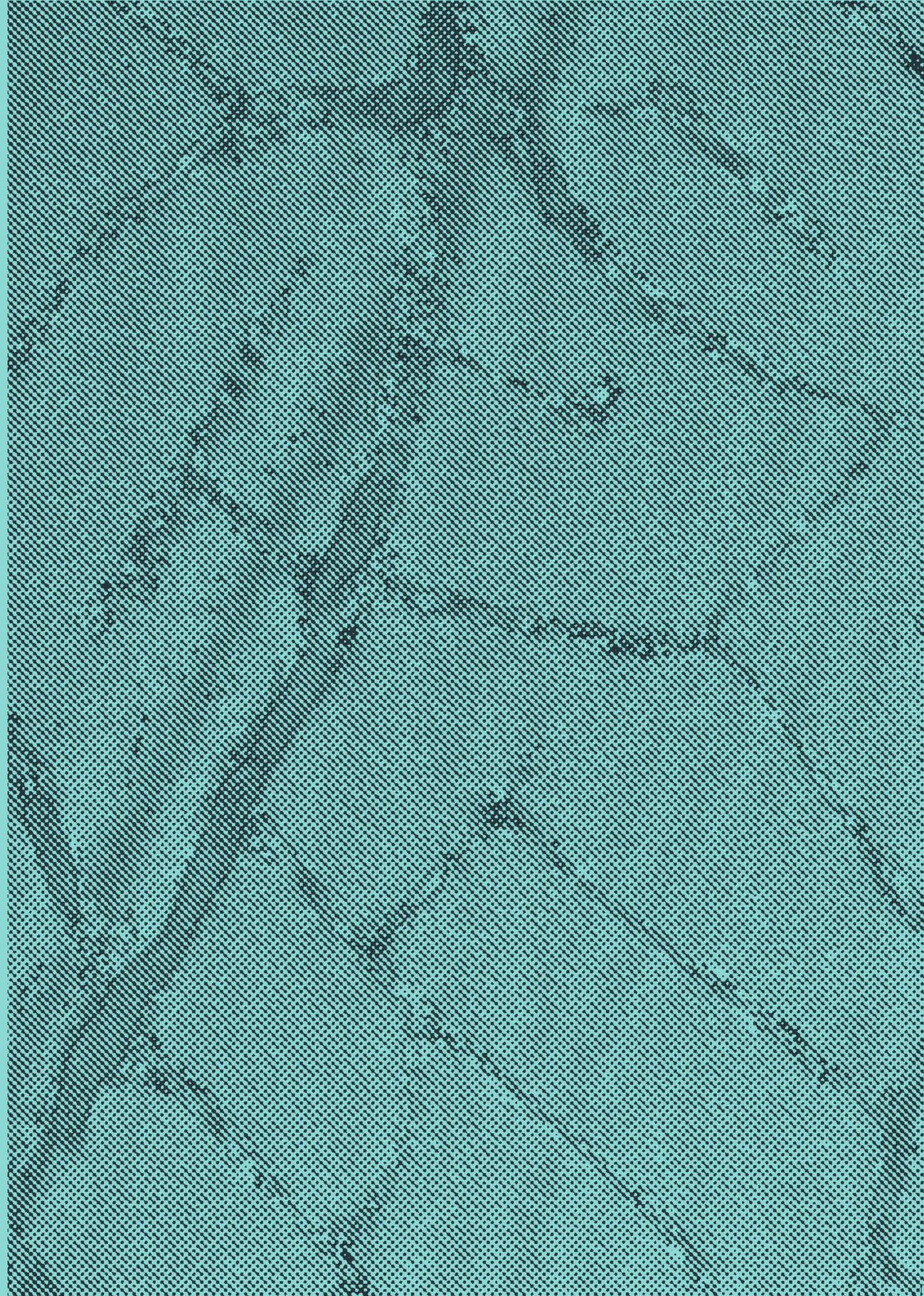
03.12.2026

DBM Research Day

28.01.2027



DBM
Basel
Department of
Biomedicine



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We are delighted to have you among us. We would like to extend our warmest welcome and good wishes!

Andrieu Matthieu
Cancer- and Immunobiology (Wymann)

Bajrami Driton
Cardiac Surgery and Engineering (Marsano / Baraki)

Bassi Dario
Pediatric Immunology (Holländer)

Bauer Denise
Molecular Neuroscience (Papassotiropoulos)

Beha Ardita
Embryology and Stem Cell Biology (Taylor)

Bentires-Alj Noa Chemsi
Blood Cancer Biology and Immunotherapy (Apostolova)

Berger Thomas Klaus
Cellular Neurophysiology (Bischofberger)

Blättler Noémie
Infection Immunology (King)

Blàzquez Butler Luisa
Developmental Genetics (Zuniga)

Bösiger Vivien
Skin Biology (Navarini)

Bouitbir Abdoullah
Tumor Heterogeneity Metastasis and Resistance (Bentires-Alj)

Bournon Pauline
Brain and Sound (Rinaldi)

Brait Sarah
Cancer- and Immunobiology (Wymann)

Braschler Nora
Pulmonary Infection Biology (Boeck)

Bratz Solano Jessica
Cardiac Surgery and Engineering (Marsano / Baraki)

Brückner Nadine
Reproductive Biology and Health (von Versen)

Buss Samantha
Translational Immuno-Oncology (Binder)

Celano Franco
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Chatelain Nina
Inner Ear Research (Bodmer)

Cottrell Oliver
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De Pascalis Francesca
Childhood Leukemia (Schwaller)

De Sa Godinho Maria
Regenerative Angiogenesis (Banfi)

Del Valle Lezier Margot
Infection Immunology (King)

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Cartilage Engineering (Barbero)

Dominguez Pazos Marta Lucia
Psychopharmacology Research (Liechti)

Doulberis Michael
Gastroenterology (Niess)

Dübi Marion
Pediatric Immunology (Holländer)

Dyson Francesca Maria
Ovarian Cancer Research (Heinzelmann / Jacob)

El Kholtei Jakob
Molecular and Computational Hematology-Immunology (Zaugg)

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Epple Raja
Cartilage Engineering (Barbero)

Erupathil Julie
Cartilage Engineering (Barbero)

Esposito Cinzia
Tumor Heterogeneity Metastasis and Resistance (Bentires-Alj)

Floryan Marie Anna
Tumor Heterogeneity Metastasis and Resistance (Bentires-Alj)

Fong Arwen
Skin Biology (Navarini)

Freitas Pontífice Encarnação de Oliveira Mariana
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Gallo Simone
Immune Cell Biology (Borsa)

Garg Yashraj
Cognitive Neuroscience (de Quervain)

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DBM-Zentrale Dienste Pestalozzistrasse (Vogt)

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Khalid Mina
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Miko Eszter
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Nachit Marine
Nephrology (Cippà)

Negretto Axel
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Nikolic Stefan
Infection Biology (Khanna)

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Rajeeth Krithika
Tissue Engineering (Martin)

Rajesh Aditi
Tissue Engineering (Martin)

Raspanti Mario
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Reddmann Dana
Systems Pharmacology (Zampieri)

Revin Jennifer
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Rostami Iman
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Salz Lucia
Cancer Immunotherapy (Läubli)

Schmid Luana
Nephrology (Cippà)

Schmidt-Barbo Paul
Molecular Neuroscience (Papassotiropoulos)

Schulz Kristina
Clinical Immunology (Trendelenburg)

Schütz Simone
DBM-Zentrale Dienste Hebelstrasse (Ottolini-Voellmy)

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Brain Tumor Immunotherapy and Biology (Hutter / Mariani)

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Brain Ischemia and Regeneration (Guzman)

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Metastasis Biology (Sznurkowska)

Tesfamariam Yonas Mehari
Experimental Neuroimmunology (Pröbstel)

Trachsel Daniel
Psychopharmacology Research (Liechti)

Volkmer Ann-Kathrin
Lehre Fachbereich Anatomie

Walter Richard
Clinical Neuroimmunology (Derfuss / Kuhle)

Weiss Philipp
Tissue Engineering (Martin)

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Wilde Levin
Tissue Engineering (Martin)

Yan Yan
Cancer Neuroimmunology (Dixon)

Zhuk Nikita
DBM-IT (Dörflinger)

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Thank You!

The DBM newsletter team would like to thank all the contributors for their work. We hope you enjoyed reading the newsletter.

Please feel free to submit your ideas and input for our next issue.

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September 2026

Universität Basel

Department of Biomedicine

DBM Basel

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September 2026