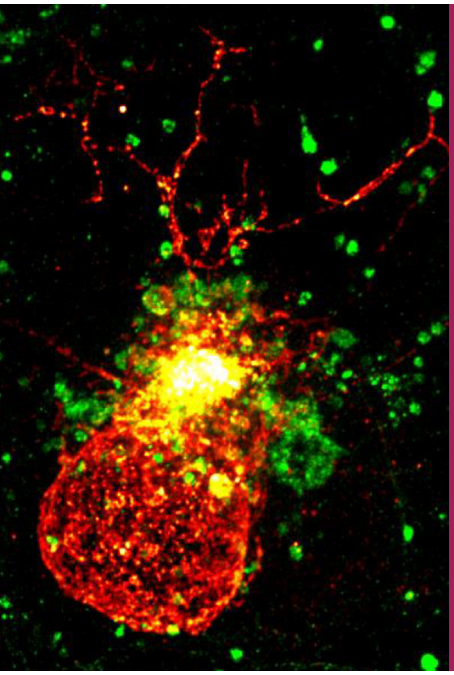
UNIVERSITY OF BERN
MCID
MULTIDISCIPLINARY CENTER FOR INFECTIOUS DISEASES

# THE SPREAD

A NEWSLETTER FROM THE MCID



ISSUE 12 | 25
NOVEMBER 2025

ARTICLES ON:



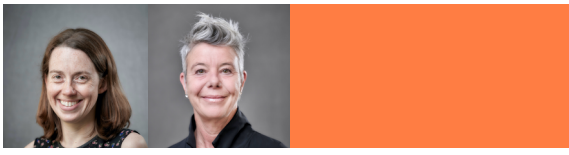
# News from the Center

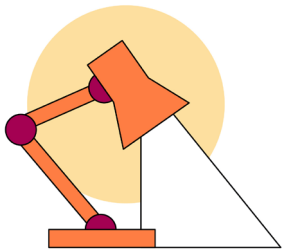
Dear readers,

welcome to the final issue of The SPREAD for 2025! Issue 12 brings to a close the showcasing of all 23 projects funded in the first phase of MCID project funding (calls launched in 2021 and 2022). At the time of writing, 43 publications linked to these projects have now been published; the new research tab of the MCID website is coming soon: check out publications and previous articles in The SPREAD, as well as the new MCID-funded projects beginning soon.

As the year draws to a close, the MCID has ended on a high note with the hosting of its Annual Event on 21st November. We welcomed more than 100 participants to a truly multidiscplinary event that featured not only infectious disease research from a range of disciplines, but also a lively panel discussion on the topic of science communication. Read more about the event:.

Yours sincerely,
Rebecca Limenitakis (MCID Managing Director)
Anita Hochuli (MCID Teaching and Outreach Coordinator)





# Shaping of Immune Function by Microbiota and Diet Across the Lifespan

Article by: PD Dr. Stephanie Ganal-Vonarburg, Department for Biomedical Research (DBMR), UVCM, Inselspital

## We Live in a World Full of Microbes

Wherever we look in nature - in water, soil, or even under extreme chemical conditions - microbes, especially bacteria, have adapted to thrive in these ecological niches. Remarkably, one of the densest microbial ecosystems exists within the mammalian intestine. These microorganisms form the so-called commensal microbiota - the collection of microbes that colonize our body surfaces, both inside and out, including the skin, airways, gastrointestinal tract, and urogenital tract. The microbiota comprises bacteria, viruses, fungi, and under certain conditions, even parasites. It is estimated that our bodies contain roughly as many microbial cells as human cells. Each of us carries a unique microbial signature. Microbiota composition is highly individualized, however not primarily determined by our genes, but rather by our environment: how we live, what we eat, and where in the world we reside.

## Why It Matters: The Function of the Microbiota

The microbiota harbors about 100 times more genes than the human genome, reflecting its vast metabolic potential. In the intestine, it fulfills two major functions. First, it assists in digestion. Microbes ferment otherwise indigestible dietary fibers, producing short-chain fatty acids which are key signaling molecules that regulate host immunity. They also synthesize essential metabolites such as vitamin K and several B vitamins. Second, the microbiota protects us from infection. It does so by occupying ecological niches, strengthening mucosal barriers, and providing crucial signals that guide the development, maturation, and regulation of the immune system.

PD Dr. Stephanie Ganal-Vonarburg

This interplay is most evident in the intestine, where microbes and immune cells coexist in proximity. The dense network of immune cells beneath the intestinal epithelium not only defends against invading microbes but is also shaped by them. Commensal microbes are essential for the maturation and fine-tuning of various immune cell types, including dendritic cells, innate lymphoid cells, T helper cells, regulatory T cells, and IgA-producing plasma cells. Beyond the gut, microbial signals can influence distant tissues such as the skin, lungs, and even the brain, through systemic immune and metabolic communication, often referred to as the gut-skin and gut-brain axes.

## How We Study Microbiota Function

To understand how commensal microbes shape physiology and immunity, researchers use germ-free or gnotobiotic mice, which are raised in sterile isolators (Figure 1) and colonized with defined microbial communities. These models allow precise investigation of host-microbe interactions under controlled conditions[1]. More refined tools include human microbiota-associated mice, colonized with microbial communities derived from healthy or diseased human donors, and reversible colonization systems, such as the use of the auxotrophic *E. coli* HA107, which enables time- and dose-restricted microbial exposure to study causal effects on host development[2].

## The Window of Opportunity

Microbial colonization begins at birth, when the newborn leaves the sterile environment of the uterus. Over the last decade, it has become clear that early-life events and environmental factors, such as mode of delivery, feeding (breast milk versus formula), introduction of solid foods, and geography (reflecting hygiene and sanitation practices), profoundly shape the developing microbiota and, in turn, the maturing immune system[3].

**Figure 1: Germ-free isolator in the Clean Mouse Facility Bern**

Recent work from our group has shown that even the maternal microbiota during pregnancy can beneficially influence immune development in the offspring[4]. Using the auxotrophic *E. coli* HA107 system that reversibly colonizes the murine intestine, we have developed a model in which germ-free mice are exclusively colonized during pregnancy, delivering germ-free pups. The offspring of such gestationally colonized dams showed enhanced innate immunity in the small intestine (alterations in type 3 innate lymphoid cell and mononuclear cell numbers) and enhanced epithelial barrier gene signature compared to offspring born to dams that had stayed germ-free throughout pregnancy. The period encompassing conception, fetal development, and early life is therefore referred to as the window of opportunity - a critical phase during which microbial and environmental cues can leave lasting imprints on health and disease risk[5].

## From Diet to Immunity

Diet is one of the strongest modulators of the microbiota throughout life. Nutrients and microbial metabolites derived from the maternal diet, breast-feeding and the transition from milk to solid food in infancy shape the composition of the microbial community and the immune networks it engages. Even later in life, dietary shifts remain among the few factors capable of altering the otherwise stable adult gut microbiota. Understanding these interactions provides a foundation for nutritional and microbial strategies to promote health across the lifespan. In our research, we model such interactions using purified, ingredient-defined diets that mimic distinct nutritional states (e.g. high-fat, high-sucrose, ketogenic, or protein- and micronutrient-deficient diets). Introducing a dietary change in colonized animals inevitably alters the composition of their commensal microbiota, making it difficult to distinguish between indirect microbial-mediated effects and direct dietary influences on the host immune system (Figure 2). Germ-free mouse models serve as a powerful tool to study the latter.

A common approach in our laboratory is to apply dietary interventions within defined life stages (gestation, lactation, or adulthood) in parallel experiments with germ-free and colonized mice. This strategy enables us to dissect how diet and microbiota, individually and together, shape immune development and function over time.

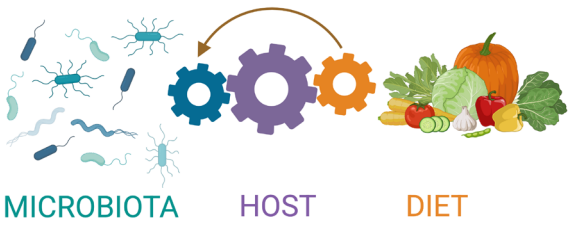



**Figure 2: Diet shapes host physiology both directly and indirectly by altering the composition of the intestinal microbiota.**

Recently, we demonstrated that a typical Western-style diet, high in fat and carbohydrates but low in fiber, plant-derived components, and microbial ligands such as lipopolysaccharide (LPS), profoundly impairs intestinal antibody responses[6]. Mice on this diet showed reduced IgA production in gut-associated lymphoid tissues and lower luminal IgA levels. Using high-throughput sequencing, we further revealed that diets rich in microbial ligands promote not only higher IgA production but also greater antibody diversity, a hallmark of intestinal immune fitness. Importantly, these effects were most pronounced during early life, leading to lasting antibody diversity, and occurred even in germ-free mice, indicating that early nutrition can shape gut immunity independently of microbial colonization.

## Translating Findings to Humans: Birth Cohorts Across Continents

To translate our mechanistic insights into the human context, we have established two complementary longitudinal birth cohorts, in Switzerland and Zimbabwe, designed to uncover how maternal transfer, microbial colonization, early-life nutrition, and environmental exposures shape infant immune and microbiota development across contrasting socio-economic settings.

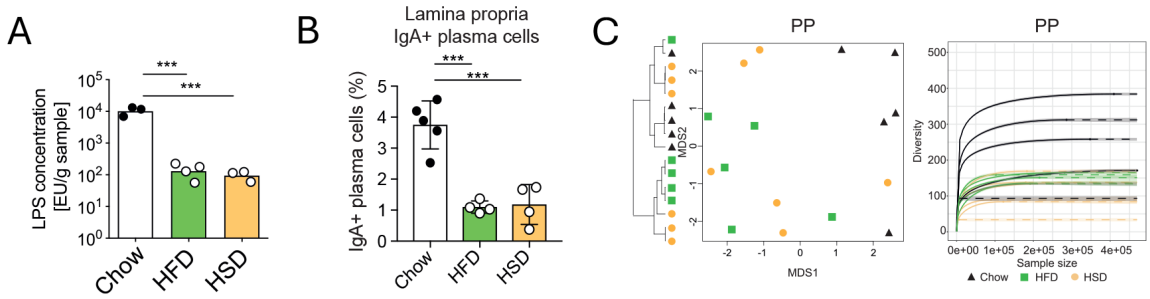



**Figure 3: (A) Purified high-fat and high-sucrose diets are low in LPS compared to chow diet. (B) Relative numbers of IgA+ plasma cells in mice fed chow or purified diets. (C) Immunoglobulin repertoire sequencing reveals a shift in the overall IgA repertoire (left) and lower diversity (right) in Peyer's patches (PP) of purified diet-fed mice.**

The University of Zimbabwe Birth Cohort Study-2 (UZBCS-2), supported by the University of Bern Global Health Initiative, has enrolled over 200 mother–infant pairs since 2025. It investigates how nutrition, hygiene, and sanitation affect microbiota–immune interactions through extensive longitudinal sampling of maternal and infant specimens from birth to ten years of age.

The Bern Birth Cohort (BeBiCo), established in 2020, follows a parallel design in a high-income European context, with over 180 enrolled mother–infant pairs and excellent retention (<10% dropout)[7].

## Concluding perspective

From birth to old age, our immune system develops and functions in continuous dialogue with the microbial world. Diet is deeply intertwined in this process. It shapes the microbial communities that colonize us, and these microbes, in turn, educate and fine-tune our immune defenses. Evidence from both experimental models and human studies has revealed that early-life nutrition and microbial exposure leave lasting imprints on immune competence and disease susceptibility. In the Ganal-Vonarburg lab, we seek to understand these interactions by an integrative approach that combines microbiology, immunology, nutrition, and systems biology, but also considers social, environmental, and economic dimensions, with the goal to find strategies that foster resilient immunity and lifelong health.

References

[1] Mooser C, Gomez de Aguero M, Ganal-Vonarburg SC (2018). Standardization in host-microbiota interaction studies: challenges, gnotobiology as a tool, and perspective. Curr Opin Microbiol.

[2] Hapfelmeier S, Lawson MA, Slack E, ..., Macpherson AJ (2010). Reversible microbial colonization of germ-free mice reveals the dynamics of IgA immune responses. Science.

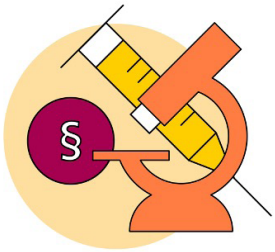
[3] Ganal-Vonarburg SC, Hornef MW, Macpherson AJ (2020). Microbial-host molecular exchange and its functional consequences in early mammalian life. Science.

[4] Gomez de Aguero M, Ganal-Vonarburg SC, Fuhrer T, ..., Macpherson AJ (2016). The maternal microbiota drives early postnatal innate immune development. Science.

[5] Torow N, Hand TW, Hornef MW (2023). Programmed and environmental determinants driving neonatal mucosal immune development. Immunity.

[6] Mooser C, Ronchi F, Limenitakis JP, ..., Ganal-Vonarburg SC (2025). Diet-derived LPS determines intestinal IgA induction and repertoire characteristics independently of the microbiota. Immunity.

[7] Cecchini L, Barmaz C, Cea MJC, ..., Ganal-Vonarburg SC (2023). The Bern Birth Cohort (BeBiCo) to study the development of the infant intestinal microbiota in a high-resource setting in Switzerland: rationale, design, and methods. BMC Pediatr.



# Investigating Cardiovascular and Respiratory Infection Risks Across Menopause Using Multi-modal Cohort Data

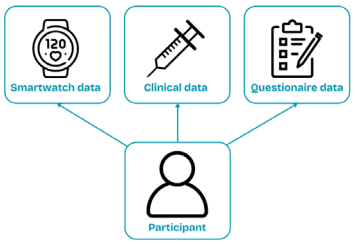
Article by: Antoine Faul, PhD student, Dr. Anja Mühlemann, Prof. Dr. David Ginsbourger, Institute of Mathematical Statistics and Actuarial Science, Prof. Dr. med. Petra Stute, Dr. med. Elena Pavicic, Dr. Dr. med. Danielle Bower, Gynaecological Endocrinology, Frauenklinik Inselspital Bern, Prof. Dr. Ben Spycher, Dr. Patric Wyss, Institute of Social and Preventive Medicine (ISPM)

Hormones exert wide-reaching effects on all organ systems of the body. During menopause, a woman's body produces substantially less oestrogen and progesterone, which are the primary female reproductive hormones. The impacts of this change in hormones leading up to and during menopause are diverse, affecting the likelihood of developing various diseases as well as manifestations in general and mental health and well-being. It is known that an increased risk of cardiovascular disease accompanies menopause. On the other hand, the role of hormones in modulating immune responses and vulnerability to infections is not fully understood.

**From left to right: Prof. Dr. Ben Spycher, Prof. Dr. med. Petra Stute, Prof. Dr. David Ginsbourger, Dr. Dr. med Danielle Bower, Dr. Anja Mühlemann, Antoine Faul, PhD student, Dr. med. Elena Pavicic, Dr. Patric Wyss**

Through a project funded by the Multidisciplinary Center for Infectious Disease at the University of Bern, a team spanning reproductive endocrinology at the Inselspital Frauenklinik, as well as the Institute of Mathematical Statistics and Actuarial Science and the Institute of Social and Preventive Medicine of the University of Bern is investigating cardiovascular risk and respiratory infection risk in perimenopausal and menopausal women. This multidisciplinary project combines prospectively collected clinical and questionnaire data, data from wearables, synthetic data modelling, and statistical approaches to tackle research challenges. These challenges notably pertain to handling missing data for clinical risk calculators, as well as to uncovering factors that underscore women's health in the transition to menopause and thereafter.

The project has involved two population-based studies: MenoFlu (still ongoing) and Frauenherzen (completed, a precursor study linked to a related Center for AI in Medicine project). Statistical methodology has been developed throughout the two projects with first applications to data[1] from a prior study (Cimbolic). In Frauenherzen, comprehensive health, lifestyle, and menopausal symptom data were prospectively collected from 253 women between 40 and 69 years of age. These data were supplemented with vital signs, blood metrics, and data from a wearable device worn continuously by each participant for one week.




We aimed to assess whether these data could be useful to surrogate missing input parameters of cardiovascular risk calculators. Our approaches highlight how probabilistically predicting blood metrics (such as cholesterol concentrations) and blood pressure, which typically require a visit to a physician, may be helpful to get provisional estimations of cardiovascular risk when these metrics are missing.

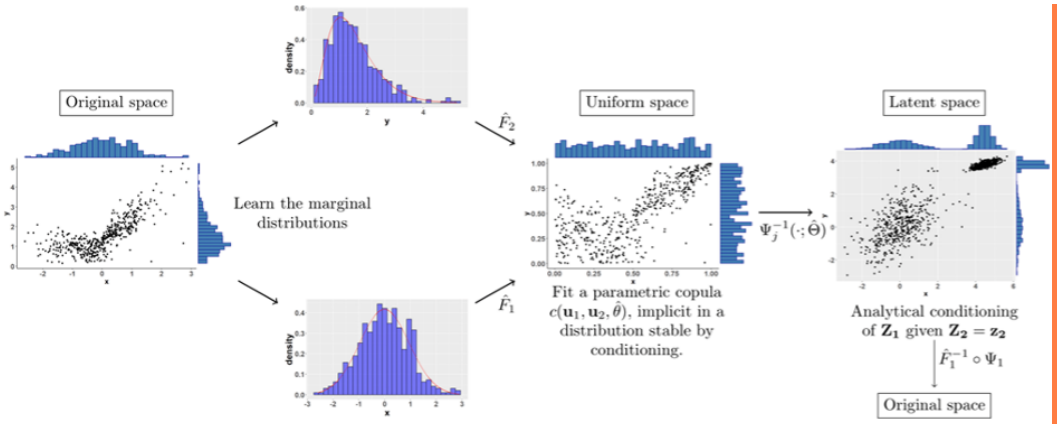



**Figure 1: Workflow of developed algorithms for sampling from conditional distributions**

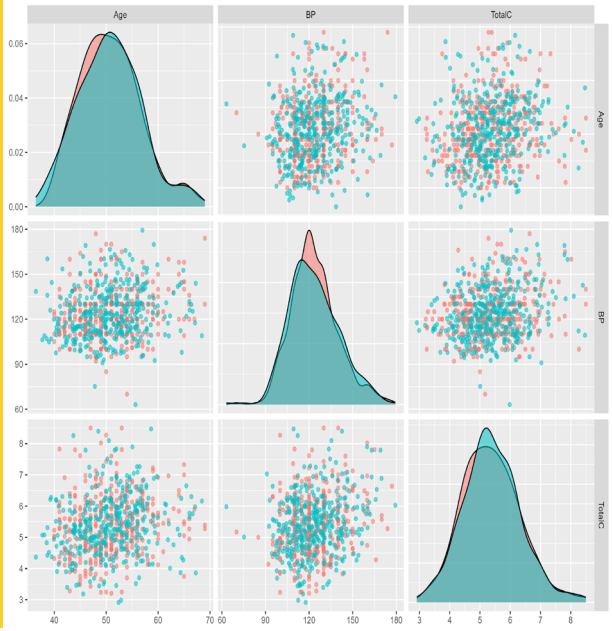



**Figure 2: Synthetic (blue) vs original data (red) from the CIMBOLIC dataset.**

The statistical methodology that is being used relies on learning joint distributions of all variables of interest on a complete data set to make predictions by computing conditional distributions. In Mühlemann et al.[2], an approach based on a Gaussian dependence structure (copula) was used. In the framework of his PhD in statistics within the MCID project, Antoine Faul generalises this approach to non-Gaussian distributions by leveraging distribution families stable under conditioning (Figure 1)[3]. The resulting workflow turns out to also lead to synthetic data generators with valuable properties. Figure 2 illustrates how a Gaussian Mixture Copula Model can be used to generate synthetic data mimicking marginal and dependency structures of a data set.

The second phase of the project examines the impacts of hormone replacement therapy in menopause on frequency and severity of respiratory tract infections (RTIs). Higher risks in post-menopausal women have been reported for both communicable and non-communicable diseases. However, it is unclear what role hormone levels play and whether hormone replacement therapy could alter the risk and severity of upper respiratory infections. To answer this question, more than 400 post-menopausal women between age 40 and 60 years were enrolled in a prospective cohort study that followed each participant for 6 months (Figure 3). The first cohort includes women who utilise systemic hormone replacement therapy, while the second cohort does not. Extensive medical background and behavioural information was collected, and participants documented life events and stressors (weekly diaries), menopausal symptoms, depressive symptoms and quality of life (monthly diaries) as well as daily symptoms and severity during respiratory infections. Additionally, vital data and blood values as well as wearable data were collected during the observation period.

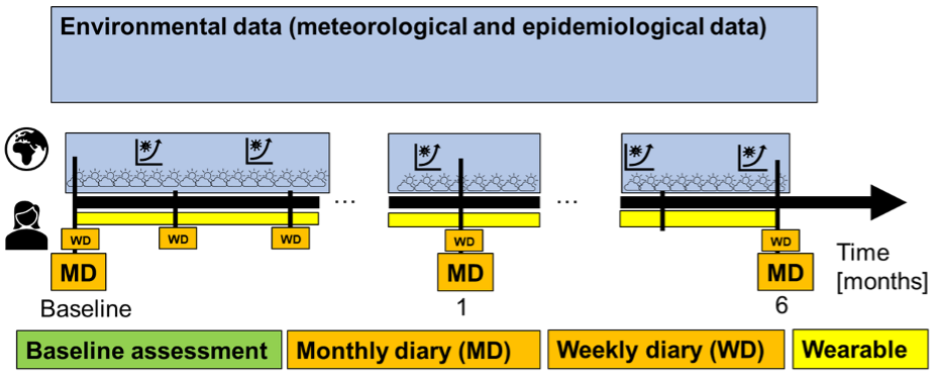



**Figure 3: Design of the longitudinal prospective study.**

These data are being analysed to examine factors associated with the frequency and severity of respiratory infections and to assess whether hormone replacement therapy affects them. Furthermore, evaluation of the wearable data leading up to the onset of respiratory infections will seek to identify patterns in the physiological data from the wearables that presage the development of a respiratory infection before symptoms emerge. Identification of such patterns could serve as an early warning system for a respiratory infection before an individual realises that they are infected. The system would facilitate population health management to reduce transmission of infections, particularly for vulnerable populations and during future pandemics.

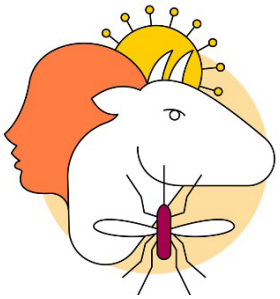
The work presented in this article would not have been possible without the contributions of many further colleagues, including Dario Allenbach, Elena Anderhalden, Claus Bachert, Silvia Biesa, Danae Bodmer, Flavia Brun, Adriana Carrie, Jann Caviezel, Fabienne Durand, Christina Giese, Audrey Grötzinger, Gunar Günther, Elena Haller, Conny Hartmann, Elin Hurschler, Rowan Iskandar, Ahmed Khattab, Kerstin Khattab, Valerija Krbanjevic, Serena Lozza-Fiacco, Irene Marcu, Eleftheria Michalopoulou, Manuela Moraru, Yana Müller, Mauricio Nogueiradislich, Enea Oertle, Janna Pape, Julia Perreten, Dominik Redzic, Natascha Rieben, Arlena Rusli, Sabine Stampfli, Philip Stange, Lara Stein, Miriam Strasser, Susanne Theis, Karla Theurer, Maria Trachsel, Laura Vogel, Linus Walker, Susanna Weidlinger.

References

[1] Faul A, Stange P, Mühlemann A, ..., Stute P (2024). Menobalance: Cardiovascular risk factors from the CIMBOLIC study. Dataset on Zenodo.

[2] Mühlemann A, Stange P, Faul A, et al. (2025). Comparing imputation approaches to handle systematically missing inputs in risk calculators. PLOS Digital Health.

[3] Faul A, Ginsbourger D, & Spycher B (2025). Easy Conditioning far beyond Gaussian. arXiv preprint.



# When Two Infections Meet: How Viruses Can Shape Malaria

Article by: Prof. Dr. Volker Heussler, Mathi Funk, PhD student, Dr. Reto Calderari, Institute of Cell Biology (IZB)

When we speak about infectious diseases, we often imagine one pathogen acting alone: one virus, one bacterium, one parasite. However, in reality, people are frequently infected not just once, but several times over the course of their lives, and sometimes even at the same time. These co-infections, when two or more pathogens are present in the same individual, are increasingly recognized as an important but often overlooked part of global health.

## Co-infections at the Population Level

Co-infections are common in regions where multiple infectious diseases are present and where exposure occurs repeatedly across a lifetime. For example, in many parts of sub-Saharan Africa, malaria is highly endemic, but so are chronic viral infections such as hepatitis B virus (HBV), Epstein-Barr virus (EBV), cytomegalovirus (CMV), and HIV[1, 2, 3]. Because infections do not happen in isolation, the health outcomes that we observe in populations are shaped by these overlapping exposures.

A well-studied example is the relationship between malaria and EBV. EBV infects nearly all humans, usually early in life, and then persists silently in immune cells. However, in regions with intense malaria transmission, repeated malaria episodes reduce the immune system's ability to control EBV. As a result, EBV levels in the body rise, and this contributes to the development of endemic Burkitt lymphoma, a childhood cancer[4]. This example shows how one infection does not need to cause disease directly; it can instead alter the immune environment, enabling another pathogen to do harm.

Although the malaria–EBV interaction is now well established, we know surprisingly little about how malaria interacts with other common viruses, including HBV and CMV, despite their high prevalence in the same populations. This is a major gap in our understanding of malaria severity across different regions, ages, and health conditions. Our research focuses on this gap: how viral infections can change the course of malaria, and which biological processes are affected. To understand this, it helps to follow malaria's life cycle inside the body, beginning in the liver and then moving to the blood.

## The *Plasmodium* Liver Stage: Where Infection Begins

When a person is bitten by an infected mosquito, malaria parasites travel directly to the liver. There, each parasite enters a liver cell and develops quietly for several days, producing thousands of new parasites. This stage is clinically silent, but it is crucial: if liverstage development is disrupted, the infection will not progress to the bloodstream. We initially aimed to study co-infection of malaria with hepatitis B virus, because HBV also infects the liver. However, working with HBV requires biosafety level 3, and delays in access to suitable laboratory space made it impossible to begin these experiments on schedule.

Prof. Dr. Volker Heussler

To keep the research moving safely, we turned to adeno-associated virus (AAV), which is widely used in gene therapy research and can be handled under standard biosafety level 2 conditions. AAV does not cause disease, but it can infect a variety of cells and modifies their internal environment including the autophagy machinery, a cellular recycling system[5]. This made it a useful model to test whether the presence of a virus-like infection in the liver changes malaria parasite development.

In his Master's thesis, Mathi Funk examined the effect of AAV infection on the growth of *Plasmodium berghei* liverstage parasites in cultured liver cells[6]. He found that the number of parasites entering liver cells did not change in the presence of AAV, suggesting that parasite invasion was unaffected. However, the parasites that developed in AAV-transduced cells were significantly smaller, indicating slower or impaired growth (Figure 1). Interestingly, this reduction in parasite size did not appear to be caused by changes in the cell's autophagy pathway known to play a role in parasite development[7, 8, 9, 10]. This suggests that virus-induced changes in the liver can influence malaria development through unexpected and previously overlooked mechanisms.

These results highlight that co-infections can shape parasite development from the very beginning of infection, long before symptoms appear.

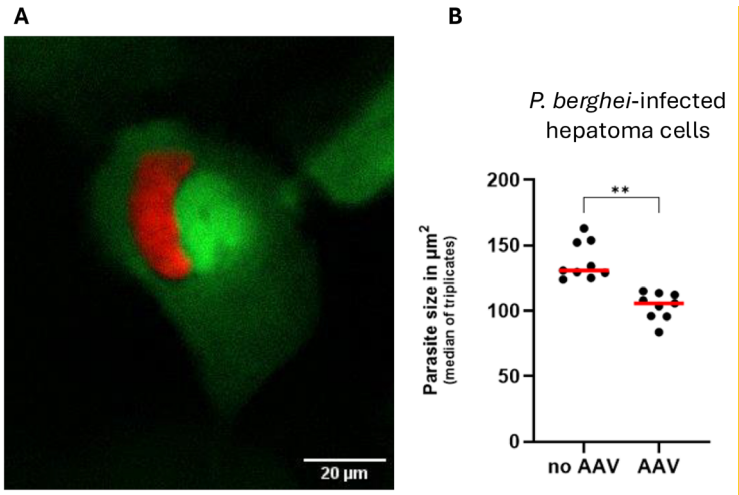



**Figure 1 Co-infection studies in Huh7 hepatoma cells. (A) Hepatoma cells were infected with *P. berghei* mCherry-expressing sporozoites. Two hours post-infection, cells were transduced with AAV for one hour. Images were acquired 48 hours post-infection. Parasite is visible in red and the presence of recombinant GFP-expressing AAV is indicated by green fluorescence. (B) Quantification of the median parasite size from individual infections in Huh7 cells, compared to the control condition (no AAV transduction).**

### The Blood Stage: Filtering Parasites in the Spleen

After leaving the liver, malaria parasites enter the bloodstream, where they invade red blood cells (RBCs). This is the stage that causes fever, chills, and potentially dangerous complications. A critical organ during this phase is the spleen. The spleen filters the blood and removes damaged or infected RBCs (Figure 2A). It also organizes immune cells that help control malaria.

If the structure of the spleen is altered, its ability to filter the blood is compromised. When this happens, malaria-infected RBCs can circulate for longer, and more mature parasite stages, which are normally removed efficiently, remain in the bloodstream. This can potentially increase parasite load and severity of disease (Figure 2B).

Some viruses, including EBV and CMV in humans, are known to subtly alter spleen structure and immune cell organization over time[11, 4]. However, these changes are difficult to study directly in people, and population-level data are still limited. To understand how spleen disruption affects malaria, we need an experimental system in which spleen architecture can be altered in a controlled and reversible way.

While much of our current understanding of viral–malaria co-infections comes from epidemiological studies, we still lack controlled experimental systems that can dissect the underlying cellular and immunological interactions. Working with highly infectious human viruses such as HBV or CMV requires biosafety level 3 facilities, which limits experimental flexibility.

To address this, we are now focusing on the development of safe and versatile *in vitro* co-infection models that can be handled under biosafety level 2 conditions. One promising direction is to follow up the AAV systems as surrogates to mimic viral modulation of host cells. Such models can help reveal how viral infection changes the architecture and function of cells that are essential for controlling malaria parasites.

Ultimately, these in vitro systems will complement animal models and epidemiological data, allowing us to link cellular changes induced by viral infection to altered malaria outcomes at the organism and population level. They also provide a safe and scalable platform for Swiss laboratories, bridging molecular biology, immunology, and public-health research within the MCID network.

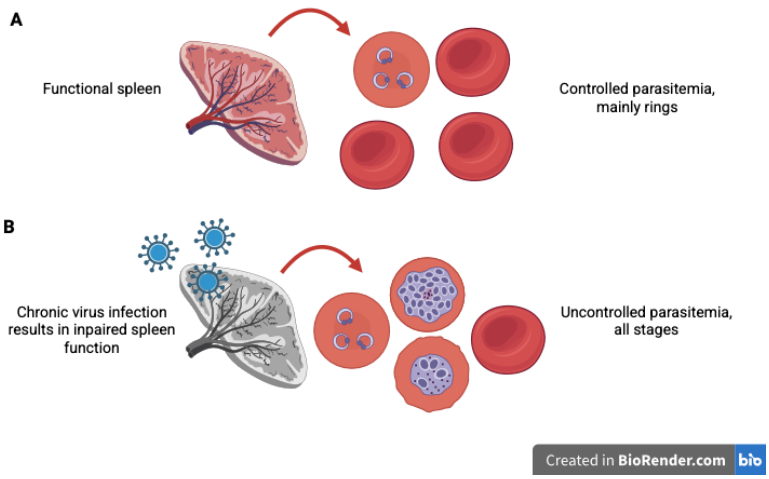



**Figure 2: Mono-infection and co-infection during the blood stage of malaria. (A) During early blood stage malaria infection, infected red blood cells (RBCs) are efficiently filtered by a functional spleen, allowing primarily ring-stage parasites to circulate. (B) In co-infected individuals with a viral infection that impairs splenic architecture and function, higher parasitemia levels and circulation of all parasite stages are expected due to reduced splenic clearance.**

References

[1] Kotepui KU & Kotepui M (2020). Prevalence of and risk factors for Plasmodium spp. co-infection with hepatitis B virus: a systematic review and meta-analysis. Malar. J.

[2] Kotepui M, Kotepui KU, Milanez GDJ, Masangkay FR (2020). Prevalence of and risk factors for severe malaria caused by Plasmodium and dengue virus co-infection: a systematic review and meta-analysis. Infect. Dis. Poverty.

[3] Mahittikorn A et al. (2021). A meta-analysis on the prevalence and characteristics of severe malaria in patients with Plasmodium spp. and HIV co-infection. Sci. Rep.

[4] Moormann AM & Bailey JA (2016). Malaria - how this parasitic infection aids and abets EBV-associated Burkitt lymphomagenesis. Curr. Opin. Virol.

[5] Hösel M et al. (2017). Autophagy determines efficiency of liver-directed gene therapy with adeno-associated viral vectors. Hepatology.

[6] Funk M (2025). Host cell in double trouble: The effect of AAV transduction of host cells on Plasmodium berghei development in the liver stage (Master's thesis).

[7] Agop-Nersesian C, Niklaus L, Wacker R, Heussler VT (2018). Host cell cytosolic immune response during Plasmodium liver stage development. FEMS Microbiol. Rev.

[8] Bindschedler A, Schmuckli-Maurer J, Buchser S, ..., Heussler VT (2024). LC3B labeling of the parasitophorous vacuole membrane of *Plasmodium berghei* liver stage parasites depends on the V-ATPase and ATG16L1. Mol. Microbiol.

[9] Bindschedler A, Wacker R, Egli J, ..., Heussler VT (2021). *Plasmodium berghei* sporozoites in nonreplicative vacuole are eliminated by a PI3P -mediated autophagy-independent pathway. Cell. Microbiol.

[10] Schmuckli-Maurer J, Bindschedler AF, Wacker R, ..., Heussler VT (2024). *Plasmodium berghei* liver stage parasites exploit host GABARAP proteins for TFEB activation. Commun. Biol.

[11] Benedict CA et al. (2006). Specific Remodeling of Splenic Architecture by Cytomegalovirus. PLoS Pathog.

THE SPREAD
A NEWSLETTER FROM THE MCID

# Mothers‘ experiences of breast-feeding during the COVID-19 pandemic in Switzerland

Article by: Dr. Jessica Laine Carmeli, Institute for Social and Preventive Medicine (ISPM), Independent lactation consultant and postpartum health support, Fribourg

I gave birth to my first child a few weeks before the COVID-19 pandemic. Not fully knowing the consequences if she or I were to contract this potentially deadly virus, and fueled by a fear of being hospitalized and separated, I froze liters of breastmilk as an emergency supply. This provided some relief from my foreboding and the volatile time that surrounded postpartum life. As a perinatal epidemiologist, I also noticed significant impacts on others' experiences of birth and postpartum. My personal experiences, combined with a research aim to understand barriers and facilitators of breastfeeding in Switzerland, led me to conduct a study of other mothers‘ breast-feeding experiences during the COVID-19 pandemic. Fortunately, I received funding from the MCID to pursue this research.

Most mothers (pandemics or not) do not achieve their breastfeeding goals or public health recommendations for breastfeeding. According to the most recent Swiss Infant Feeding Study, despite high rates of breastfeeding initiation, 74% of babies stop being breastfed at around 5 or 6 months or stop being breastfed exclusively[1]. Beyond this, very little data exists about the experiences of breastfeeding mothers, and even less about how experiences were affected by the COVID-19 pandemic in Switzerland.

I hypothesized that barriers to breastfeeding existed at multiple levels and were not solely rooted in intrapersonal aspects of knowledge deficits or decision-making (e.g., knowing whether breastfeeding was beneficial), but that higher levels (e.g., political/structural factors) also played a central role during the COVID-19 pandemic. Using a qualitative research framework, mothers and key informants, including those involved in perinatal and postnatal care, were interviewed.

**The first bottle of pumped milk, I didn't even have proper storage bags because I wasn't planning on pumping. I saved this bottle in my freezer until the end of this study to remind me of the importance of this research.**

Women's experiences breastfeeding during the pandemic varied, though there were some common threads. Barriers to breastfeeding existed at multiple levels, and while some women mentioned facilitators of breastfeeding, none viewed the pandemic as something that made their breastfeeding experience easier or better.

The greatest burdens to breastfeeding initiation were related to changes in hospital procedures regarding birth and immediate postpartum care. Women mentioned that challenging birth experiences because of the pandemic influenced their breastfeeding. Additionally, mothers were not routinely referred to a certified lactation consultant in the hospital, and at dismissal, little to no contact information for support was provided. Postpartum services were limited, cancelled, or conducted online.



UNIVERSITY OF BERN
MCID MULTIDISCIPLINARY CENTER FOR INFECTIOUS DISEASES

Many women expressed frustrations in getting immediate support services, especially when they faced breastfeeding challenges, such as concerns about their baby's health (e.g., weight gain), or if they were experiencing personal crises (e.g., mastitis). Midwives expressed they felt that managing crises, such as the spread and management of the disease, took urgency over quality and continuity of care for new mothers.

*"I didn't have a pleasant birth experience, and starting breastfeeding was very challenging. The hospital staff were not very helpful, and I felt ignored and dismissed. I didn't feel confident that my baby was getting enough milk, breastfeeding was painful, and I felt horrible from the way my birth went. I was released early, and when I asked for a lactation consultant at home, they said postpartum visits were on hold."*
*-Angela, 1st-time mom who gave birth in the canton of Bern in March 2020*

*"We were all in crisis, trying to minimise the spread of disease and take care of COVID-infected mothers. Our workloads increased, and protocols were continuously changing. Postpartum care was probably the least of our worries. If a new mother seemed to be fine, we could shift our limited time and focus on others. Postpartum home visits were shortened or cancelled. Looking back, I think this limited mothers' ability to ask for or receive help, especially when they really needed it. I think most mothers were not fine."*
*-Marie, a hospital and home-visit midwife in the canton of Vaud*

Many mothers discussed barriers to breastfeeding from existing social norms (e.g., supplementation with formula), insufficient maternity leave policies, and a lack of support, information, and regulations surrounding breastfeeding. For example, there was a lack of support for breastfeeding mothers who wished to breastfeed their children when they were out of the home (e.g., daycare), and a lack of training of health professionals, including pediatricians, on how to support breastfeeding mothers.

*"Before giving birth, I thought I would breastfeed for as long as I wanted to, but when my maternity leave was over, I was confused about how to transition my breastfed child into daycare. My baby wasn't taking pumped milk from a bottle, and the daycare wouldn't allow me to nurse him at lunch because of COVID measures. They also said that they wouldn't take pumped milk after the first year anyway. I panicked, and my pediatrician suggested I supplement with formula."*
*-Alina, 1st-time mother who gave birth in the canton of Bern in March 2020*

*"My baby had a fever and wasn't drinking a lot of breastmilk. My pediatrician said if he didn't drink more in the next 24 hours, to try formula supplementation, formula was the solution."*
*-Julia, 2nd-time mother who gave birth in the canton of Vaud in May 2020*

Knowledge of the benefits of breastfeeding was also challenged by potential COVID-19 transmission risks and perceived vaccine risks vs benefits.

*"When I got COVID, I was so exhausted and sick that breastfeeding became a huge challenge, my milk supply dropped, and I had no family support for the care of my other children. Despite what I knew about the low risk of passing the virus to my baby, I feared that my milk was going to make her sick. I stopped breastfeeding when I got sick. This is something I still regret years later."*
*-Lara, 3rd-time mother who gave birth in the canton of Bern in December 2020*

Many mothers expressed stress from measures associated with the management of the pandemic. Aspects such as isolation and changes in familial and social support complicated their breastfeeding experiences, where adequate support was not received from family members due to the need for social distancing and pandemic restrictions. Furthermore, mothers of multiple children lacked care for older children, including during the 10-week period that schools were closed in the first wave of the pandemic and later when schools were reopened.

As demonstrated by Leonie's should statement below, many mothers expressed "shoulds" and internalised notions rooted in patriarchal motherhood, including that their (in)actions reflected whether they were good or bad mothers. Several viewed their breastfeeding bodies as a site of care that conflicted with their personal goals for breastfeeding, where they felt pressure to be a good mother, and that if they didn't protect their baby via their breastmilk, they were bad mothers. Furthermore, mothers mentioned feeling like bad mothers for not being able to handle the stress that came from the pandemic.

*"With my first two kids, my parents would take care of them two times a week, while I worked, but with COVID, we wanted to protect their health because they were older. They didn't even meet their third grandchild until she was 8 months old. Taking care of three children on my own was a huge stressor, especially when schools were closed during the first lockdown. Breastfeeding my baby became too much of a burden with all that I had to manage, so I supplemented with formula and then quit breastfeeding. I know that I should have continued, but it was what I needed to do."*
*-Leonie, 3rd-time mother who gave birth in the canton of Vaud in February 2020*

**Dr. Jessica Laine Carmeli: „Two years after the pandemic began, breastfeeding in public places meant that I wore a mask for protection."**

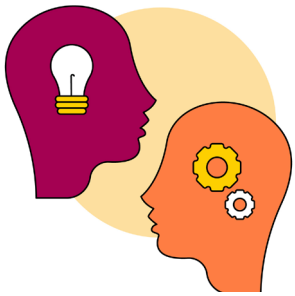
Five years after I frantically pumped and stored my breastmilk, now as a mother of two, I know the many challenges that mothers stated above from mothering during a pandemic because I also lived them. But hearing the many voices of others, I was able to understand the multifaceted, multilayered, and complex challenges for addressing breastfeeding barriers during pandemics. This study has identified breastfeeding experiences of mothers were subpar during the COVID-19 pandemic. This is not surprising given that Switzerland ranks low in support for breastfeeding mothers before the pandemic. According to the World Breastfeeding Trends Initiative, which audits breastfeeding programmes and policies, Switzerland scored 48 points / 100 points in 2019[2]. The need for vast changes to support breastfeeding in Switzerland, during non-pandemic and pandemic times, is dire.

References

[1] Dratva J, Gross K, Späth A, Zemp, Stutz E (2014). Swiss Infant Feeding Study: A national study on infant feeding and health in the child's first year.

[2] Initiative TWBT. The World Breastfeeding Trends, Initiative (WBTi), Report for Switzerland (2020).

THE SPREAD
A NEWSLETTER FROM THE MCID



# Bright and earlier: meet two young MCID researchers

Interview with:
Selina Wegmüller, Institute of Philosophy (PHILO)
and
Marta Zimoch, Institute of Virology and Immunology (IVI), Mittelhäusern and Vetsuisse

**Can you describe briefly the project you are working on and what you aim to achieve?**

**Marta:** Our MCID-funded project focuses on the Wesselsbron virus (WSLV), a neglected mosquito-borne flavivirus endemic to sub-Saharan Africa that affects livestock and occasionally humans. However, information about its transmission routes, disease characteristics, and prevalence remains limited. Our objectives are to examine virus transmission in sheep, study the disease profile with an emphasis on liver pathology, and establish a mouse model to investigate pathogenesis. To achieve these goals, we are using reverse genetics to recreate field strains affecting humans and animals in endemic regions. These strains, previously unavailable as isolates in Europe, make our project both timely and directly applicable.

**Selina:** The regulation of animal research remains a topic of frequent debate in Switzerland. Since the 1980s, various popular initiatives have emerged, proposing stricter regulations such as a complete ban on animal experimentation. Each associated referendum has generated a public discourse on animal research, involving various ethical arguments and reflecting shifting sentiments of the population. Our research project (NRP 79, SNSF) at the MCID Ethics and Policy Lab examines the ethical dimensions and the policy narratives of the societal debate on animal testing in Switzerland over time. My part of the project focuses on the ethical dimensions and analyses the arguments of the debate. To share this knowledge, we will create an interactive website that makes our results accessible to everyone interested and will provide the various stakeholders and the public with the means to use more solid arguments instead of moral intuitions without an argumentative basis.

**What excites you about multidisciplinary research?**

**Selina:** I find it very exciting that our project is being approached from two disciplines, as this allows for a more comprehensive perspective on the research question. That's what I find so fascinating about the multidisciplinary approach: it opens up new perspectives, brings new knowledge and ideas that can also be useful for one's own work. It's also highly enriching when a question is examined from two different fields, because ideally, this allows for a deeper understanding than if only one discipline were involved. I'm not sure that every research question necessarily benefits from an interdisciplinary approach, but in our case, I believe it's particularly advantageous for analysing the debate on animal testing in Switzerland.

Selina Wegmüller, PhD student

**Marta:** What excites me most is looking at the same problem through the eyes of people trained in completely different ways. It keeps me grounded and pushes me to reflect on how I work, my habits, my priorities, and how I collaborate with others.

**What do you see as being the main challenges in multidisciplinary research?**

**Marta:** To me, one of the trickiest parts of multidisciplinary research is setting shared priorities. Different fields often bring different goals and ways of working, and communication can be complicated. Finding outcomes that satisfy everyone takes time and flexibility, but when it clicks, I think it's super rewarding.



UNIVERSITY OF BERN
MCID MULTIDISCIPLINARY CENTER FOR INFECTIOUS DISEASES

**Selina:** Presumably, one challenge in multidisciplinary research is communication, which is closely related to the differences in methods and approaches between disciplines. But I believe that these very difficulties are what make interdisciplinary research valuable. It is an art to connect different ways of thinking and create something meaningful from them. Ideally, this challenge can be incredibly exciting and stimulating.

**How does your project contribute to preparedness for infectious disease threats?**

Marta Zimoch, PhD student

**Marta:** Our work enhances preparedness on multiple levels. In the laboratory, we have developed methods that can be applied in future studies of WSLV and used to support diagnostics in the event of new outbreaks. For clinicians, and within a One Health framework, improved understanding of the disease makes it easier to recognize symptoms and to include WSLV in potential diagnostic panels. Importantly, our transmission studies demonstrate that the virus can be spread through milk, which should serve as a guideline for those working closely with animals in endemic regions. This is particularly relevant given the prevalence of raw milk consumption in endemic areas, as well as in other regions, including Europe, where this trend is increasing.

**How did your education lead you to choose a multidisciplinary project for your PhD thesis?**

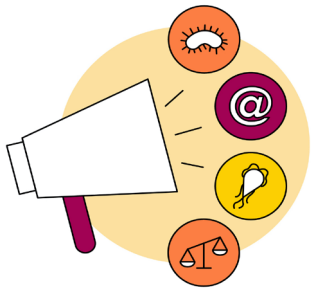
**Marta:** I studied biotechnology, and what hooked me was its core idea: thinking beyond the lab bench, whether in health, business, or somewhere in between. I still get the most energy from projects that let me connect the dots, especially when they push me outside my usual bubble.

**Selina:** I had some difficulty deciding what to study during my bachelor's degree, which led me to explore several fields for a few semesters. In the end, I chose philosophy because it represented in a way, what I was looking for – a form of interdisciplinarity. I was fascinated by the openness to different topics and by the ability to engage with various approaches and analyse them through logic. For that reason, a multidisciplinary project was really the only option that made sense for my PhD.

**Short biography of the two young researchers:**

**Marta Zimoch** is a PhD student at the Institute of Virology and Immunology (IVI), working on a project led by Prof. Dr. Charaf Benarafa and Dr. Obdulio García Nicolás. Her research focuses on the neglected zoonotic Wesselsbron virus (WSLV), and the project has already published findings in PLOS Pathogens showing that WSLV can spread in sheep through milk, without mosquitoes shared (first author publication 🔍). Before starting her PhD, Marta earned her MSc in Biotechnology from the University of Wroclaw, Poland.

**Selina Wegmüller** earned a Master's degree in Philosophy and German Studies at the University of Bern in 2020. During her studies, she worked as a research assistant at the ISPM and the former CTU. After graduation, she worked as a Junior Project Manager and participated in two very exciting MCID projects: the BEready cohort and the project Early Detection for Early Action: Integrating Multiple Data Sources for Monitoring the SARS-CoV-2 Epidemic in Near Real-Time. Through this work, Selina came into contact with the MCID Ethics and Policy Lab and applied for the open PhD position (NRP 79, SNSF). Her first paper, An analysis of ethical arguments in the public debate on animal testing in Switzerland, will be submitted this month. Finally, an interactive website with an accompanying outreach event is planned. The next steps include conducting a web-based stakeholder mapping and interviews, which will lead to a publication on stakeholders' ethical arguments regarding animal testing, followed by another paper on the normative assessment of these arguments.



# Events and opportunities

**Seminar by Prof. Dr. Marcel Salathé, 5th December**
Title: "Development of AI in Health". Date: 5th December, 12.15. Location: Hochschulstrasse 4, room 205. Seminar co-organised by MCID, Institute for Infectious Diseases (IFIK) and Swiss Biorisk Alliance

**Seminar by Dr. Elisabeth Bik, 5th December**
Public lecture on Scientific Integrity, title: "Errors and Misconduct in Biomedical Research Images". Date: 5th December, 16.00. Location: Hochschulstrasse 4, Aula 2nd floor.

**Open Science courses and workshops, Open Science University of Bern**
Courses on Open Access publishing, Research Data Management, the BORIS research data and projects repository portal and advice on compliance with funding agency publishing requirements. See here for more information:

**Public Health Sciences (PHS) Course Program**
Training program focusing on strengthening research competence of PhD students in methodologies employed to generate scientific evidence for the public health field. More information and course content (single course registration possible)

# Highlighted publications

**Stephen Leib and collaborators on three-dimensional mapping of tick-borne encephalitis virus (TBEV) in the brain**
Stephen Leib and collaborators have developed a green fluorescent protein (GFP) reporter for TBEV, allowing high-resolution 3D-mapping of TBEV in the mouse brain, revelaing new insights into virus tropism (MCID-funded)

**Tim Rollenkse and team on the efficient expression and purification of IgA**
Tim Rollenkse and team have developed a scalable and efficient method for production and purification of mouse dimeric monoclonal IgA antibodies, a key step in deepening understanding of IgA antibodies in mucosal immunity (MCID-funded)

**Céline Honegger, MCID-PhD student, on government blame deflection during crises**
Céline Honegger provides insights into how government communication shapes perceived policy legitimacy and how elections affect government credit-claiming/blame-shifting patterns, with a focus on the Covid-19 pandemic & (MCID-funded)

**Annika Frahsa and team on community participation in public health research during the Covid-19 pandemic**
Annika Frahsa and team have conducted a scoping review on participatory approaches used during the Covid-19 pandemic, providing key recommendations to allow more equitable and effective community health interventions (MCID-funded)

**Ronald Dijkman and team on high-throughput whole-genome sequencing of influenza A virus**
Ronald Dijkman and team have developed an optimized methodology for whole-genome sequencing of influenza virus A, with improved throughput and sensitivity that allows genomic surveillance at the human-animal interface (MCID-funded)

**Britta Lundström-Stadelmann and colleagues on screening of a compound library against *Echinococcus* parasites**
Britta Lundström-Stadelmann and team screened an open-access compound library composed of 400 drug-like molecules (Pandemic Response Box), revealing one compound as a potential drug candidate for alveolar echinococcosis treatment

**Jörg Jores and collaborators on the design of an engineerable bacterial chassis for antigen and drug delivery**
Jörg Jores and team have genetically engineered a cell-wall deficient *Mycoplasma* bacterial species to stably incorporate foreign DNA, a key step in adaption to use as a drug delivery system and live vaccine chassis

# MCID @ The UniBE “Nacht der Forschung” (Night of Research) 2025

On 6th September, the MCID took part in the University of Bern’s Nacht der Forschung, a vibrant evening of science that welcomed more than 10,000 visitors. Guests had the chance to explore diverse exhibitions showcasing the cutting-edge research happening across the university.

As well as showcasing the BEready Cohort and Ethics and Policy Lab, the MCID station included a Pandemic Control Centre, where visitors could follow a real-time virtual outbreak as it unfolded, posters on MCID-funded research projects, hands-on activities including quizzes, interactive posters and a handwash station, and the now legendary “Splat the Bacteria” challenge. Thank you to all our volunteers, the event organisers and all who visited the station!

# MCID Annual Event 2025


The MCD Annual Event 2025 took place on 21st November with more than 100 participants (see here for the event program: ).

The day opened with a keynote lecture from Dr. Eva Veronesi on the threat and control of vector-borne diseases. The morning continued with a series of short oral presentations on a wide-range of topics linked to infectious diseases, with topics including classroom exposure to respiratory viruses, tick-borne encephalitis, and the experiences of breastfeeding mothers during the COVID-19 pandemic.

During the lunch break, participants explored more than 25 posters on wide-ranging aspects of infectious disease research. These included many MCID research projects, an impressive display of the centre’s diverse scientific portfolio.

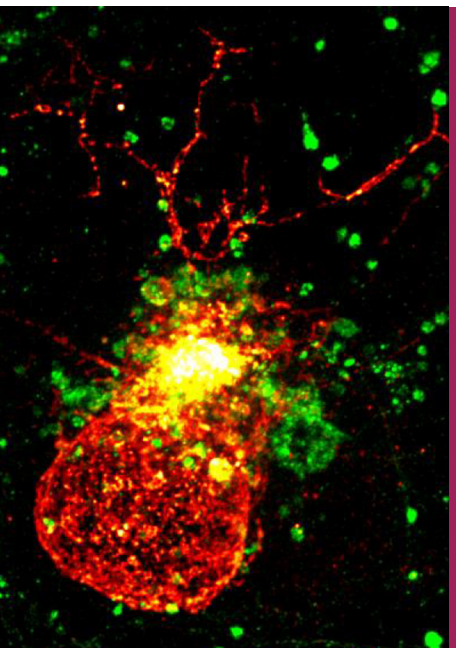
The afternoon featured a trio of keynote speakers, with invited talks by Prof. Tobias Rohrbach, Dr. Alina Zumbrunn and Dr. Philipp Markolin on the role of journalism in the spread of conspiracy theories during the COVID-19 pandemic, public attitudes influenced by affective polarisation with a focus on vaccination and an exploration of the rise of an anti-science ecosystem that prioritises emotional narratives and conspiracy myths over evidence-based information.

The final session focussed on “Bridging the divide: Science communication in a polarised world”. MCID co-chairs Prof. Carmen Faso and Prof. Volker Thiel moderated a panel discussion with the afternoon‘s speakers, joined by Dr. Theres Lüthi. The panel tackled a range of timely questions: how much fake news can, or should society tolerate? How has the role of journalists evolved during the pandemic? Should all scientists be expected to engage publicly?

A truly multidisciplinary day! Thank you to all involved.

IMPRINT

THE SPREAD
A NEWSLETTER FROM THE MCID



The Multidisciplinary Center for Infectious Diseases (MCID) is a strategic center of the University of Bern, Switzerland, founded through the generous support of the Vinetum Foundation

Register here to receive future copies of the newsletter by email and visit the MCID website to read more about MCID activities and news

Image (front and back cover) Courtesy of Kodzo Atchou, Heussler lab

## CONTACT

University of Bern
Hallerstrasse 6
Multidisciplinary Center for Infectious Diseases
3012 Bern
Switzerland

Website: www.mcid.unibe.ch
Email: info.mcid@unibe.ch
Bluesky: @mcid-unibe
Linkedin: mcidbern

UNIVERSITY OF BERN
MCID
MULTIDISCIPLINARY CENTER FOR INFECTIOUS DISEASES