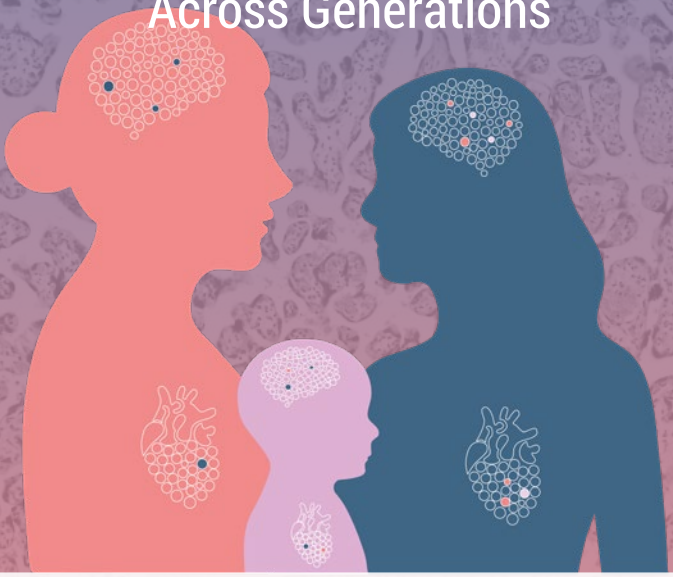
Program & Abstract Book

Multigenerational Me

The Science of Cellular Connection Across Generations



Public Symposium on Microchimerism
May 26, 2026
&
International Conference on Microchimerism
May 27-28, 2026

Medical University of Graz, Aula

Public Symposium on Microchimerism
May 26, 2026, Graz
&
International Conference on Microchimerism
May 27-28, 2026, Graz

Venue
Medical University of Graz
Neue Stiftingtalstraße 6
8010 Graz
Austria

Contact Conference
conference2026@microchimerism.info

Contact Industry Partners
Jacqueline Hirscher, ScienceEvents
conference2026@microchimerism.info

Website: microchimerism.info
LinkedIn: microchimerism community
#microchimerism2026

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Medical University of Graz

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Thomas Kroneis

Dear colleagues, friends, moms, kids and dads… dear Microchimerists

This International Conference and its preceding Public Symposium is inseparably linked with to our ongoing project on microchimerism. When drafting the project, we intended to raise microchimerism research to a next and holistic level. This meant to address microchimerism research as a biological phenomenon present since a 100 million years, level it up from a finding that coincidentally pops up in every tissue and organ and in every healthy and diseased condition and make it the main character in an equation allowing to interpret known associations with health and disease.

Likewise it was clear that the output of our approach must be greater than the number of researchers involved in the core project, that we need key opinion leaders in microchimerism research and beyond and that we need to get going a vivid discussion on the new and fascinating results that started to emerge from the microchimerism field switching from detection to functional description of microchimerism. As a consequence, we visualized a big conference on microchimerism research to meet as an Microchimerism Community and discuss how to move forward.

Interestingly, as we proceeded with the project, a thought got momentum that mirrored our responsibility towards the public, which is most often the financial driver of science.

We wanted our colleagues, friends, families and the public in general to know about this fascinating phenomenon which we think is present in all of us. But it was not only this fascination of fetal and maternal cells living inside of us that we thought would catch the people. We also embraced a psychological aspect of microchimerism reported in so many anecdotes by those who were made aware of microchimerism across the world and what I personally think is microchimerism's probably highest impact on our society, on human and women's health; namely its potential to support grief management after pregnancy loss (and even early loss of parents).

Thus, with the Public Symposium we will meet our mission and pleasure to introduce microchimerism to all of you who are eager for knowledge and interested in science, especially in this life-changing phenomenon, no matter if you are academic or not, attending school, college, university or apprenticeship, whether you

An overview of Graz

work in industry or a health care system or just like to philosophize. Let us guide you through the miracles of microchimerism and experience something that may never leave you and may even serve as comfort in times of sadness.

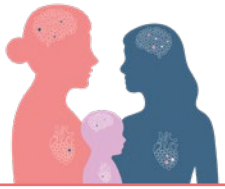
For those willing to dig into the science of microchimerism, the International Conference of Microchimerism will provide a delicately arranged bouquet of microchimerism data across its many shades. All these different aspects shall contribute their parts, allowing us to discuss the entire puzzle. We wish the talks to create the entrance for an vivid and fruitful exchange continued at the poster and the meet-the-speaker event, at the booths with the industry and in between scientists across all discipline backgrounds finally creating this sparkling atmosphere that will enable us as Microchimerism Community to thrive and take the next quantum leap.

May these events be informative and helpful, interesting and enlightening, provocative and demanding … and always of personal value.

Yours sincerely,

Thomas Kroneis
Confernce President

Public Symposium on Microchimerism
Program



1:15 pm Welcome coffe

1:45 pm Welcome Note, Thomas Kroneis

2:pm – 3:15 pm Session 1
What we know about Microchimerism today

- ▸ Diana W. Bianchi, USA, Microchimerism is underappreciated and it affects all of us
- ▸ J. Lee Nelson, USA, Microchimerism and the Interconnected Human Identity
- ▸ Katja Sallinger, Austria & Kristine Chua, USA, Interdisciplinary and Cross-disciplinary investigation of microchimerism

Q & A

3:15 pm - 3:45 pm Coffee Break

3:45 pm – 5:00 pm Session 2
What are the unknowns and potential applications of Microchimerism

- ▸ CANCELLED Markus Hengstschläger, Austria, Reconstructing the baby's genome from mom's blood – bioethical aspects of prenatal testing
- ▸ Amy Boddy, USA, Evolutionary perspectives on microchimerism
- ▸ Rachel Tompa, USA, Reflections on microchimerism after pregnancy loss
- ▸ Rachel Lewis, USA, Expecting the Unexpecting: Compassionate care for your pregnancy loss patients
- ▸ Anna Rath, Austria, Kathrin Kroneis, Austria, Breastfeeding and bonding

5:00 pm - 5:30 pm Coffee Break

5:30 pm – 7:00 pm Session 3
Hidden Guest including get together (until 9:30 pm)

- ▸ Lise Barnéoud, France, These cells that are not our own

Expert panel discussion

Summary & meeting with the speaker by wine and soup

Abstracts

2:00 pm – 3:15 pm Session 1
What we know about Microchimerism today

Microchimerism is underappreciated and it affects all of us

Diana W. Bianchi

NIH/NHGRI, USA

Sometimes the most important discoveries occur accidentally. In 1928 the British bacteriologist Sir Alexander Fleming returned from a vacation to find that a petri dish had become overgrown with mold, but around the mold growth there was no bacteria. He had discovered the antibiotic penicillin!
In 1994 we unexpectedly detected male chromosomal DNA in circulating stem cells from women carrying female fetuses. Initially attributed to laboratory contamination, subsequent studies by our team demonstrated that these cells originated from prior male pregnancies and could persist for decades in the mother, establishing a state of microchimerism. Further investigations revealed that these cells, which we called "pregnancy-associated progenitor cells" localize to damaged maternal tissues, differentiate into organ-specific cell types, and may contribute to tissue repair. Long-term epidemiologic data suggested a protective association between fetal microchimerism and reduced cancer mortality, supporting a functional role for these cells. More recent work has highlighted the immunological implications of bidirectional maternal–fetal cell trafficking, including roles in immune tolerance during pregnancy, tissue repair, and potentially autoimmune disease. Collectively, these findings support the concept of a multigenerational "microchiome" and suggest that microchimerism is a fundamental biological process contributing to maternal health, reproductive fitness, and immune development in the baby. We are eternally connected to generations that precede and supersede us.

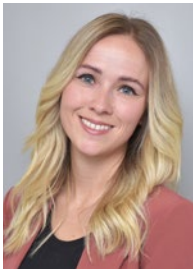
Microchimerism and the Interconnected Human Identity

J. Lee Nelson

Fred Hutch Cancer Center, Seattle, USA

Bi-directional exchange during pregnancy creates a long-term legacy of microchimerism (Mc), harboring a small number of genetically disparate cells and/or other biological material such as DNA. Maternal cells persist in her progeny into adult life and cells of fetal origin persist decades later in previously pregnant women. These naturally acquired cells carry a complement of genes that differ from the

individual who acquires them and have the capacity to affect an individual's health in a wide variety of ways. Mc has been implicated in a number of autoimmune diseases, both beneficial and detrimental to the individual, for example in rheumatoid arthritis and systemic sclerosis (scleroderma). Mc is thought to be beneficial against some types of cancer, but for others may fuel tumor growth, for example breast cancer and melanoma, respectively. Other studies point to the ability of Mc to contribute to tissue regeneration, including cardiac repair. Mc reaches the brain indicating a capacity to affect neurobiology and potentially development. A woman, who already harbors maternal Mc from when she was a fetus, can later acquire additional Mc sources from her own pregnancies and the capacity to influence reproduction has been explored in pregnancy complications such as preeclampsia and recurrent miscarriage. Of special interest is the role of transgenerational Mc in reproduction and in evolution. Additionally, naturally acquired Mc may influence transplantation success, for example inducing better tolerance in organ transplantation or decreased leukemia relapse after hematopoietic cell (bone marrow) transplantation. Our natural immigrants are with us for the long-term, often for better, sometimes for worse. The "Microchiome" has the potential to affect the health of an individual in multiple different ways which may also change with aging and be variable according to body location.



Interdisciplinary and Cross-disciplinary investigation of microchimerism

Katja Sallinger

Division of Cell Biology, Histology and Embryology, Gottfried Schatz Research Center, Medical University of Graz, Graz, Austria

Kristine Chua

University of Notre Dame, Notre Dame USA

This session explores how "bridging" disciplines can generate new questions about microchimerism, the presence of a small number of cells exchanged between two individuals, most commonly between mother and fetus during pregnancy. These cells can travel through the body and persist for decades, but scientists are still working to understand what they do and how they move in
the body. Early-career researchers Katja Sallinger (cancer research and data analysis) and Kristine Chua (evolutionary anthropology) will share how their fields approach the study of microchimerism and what new insights emerge when these disciplines are brought together. Together, they discuss how interdisciplinary collaboration can deepen our understanding of the role microchimerism may play in infection, pregnancy, and long-term health.

3:45 pm – 5:00 pm Session 2
What are the unknowns and potential applications of Microchimerism

Reconstructing the baby's genome from mom's blood – bioethical aspects of prenatal testing

Markus Hengstschläger

Vienna, Austria

Evolutionary perspectives on Microchimerism

Amy Boddy

Department of Anthropology, University of California Santa Barbara, USA

Fetal microchimerism has been associated with both positive and negative effects on maternal health. These mixed effects may stem from an evolutionary tension: mothers and their offspring have shared interests in some areas but conflicting interests in others, a dynamic known as maternal-fetal conflict. From an evolutionary perspective, fetal cells may function similarly to the placenta. Just as the placenta transfers resources from mother to baby during pregnancy, fetal cells that remain in maternal tissues after birth may continue to help transfer resources to the offspring. This resource transfer can benefit both mother and child, or it can represent the fetus „pushing" for more than what's optimal for the mother's health, creating conflict over how resources are allocated. Depending on the mother's specific circumstances and health needs, these fetal cells may help with maternal bodily maintenance (such as wound healing) or manipulate maternal physiology in ways that favor the offspring. We propose that fetal cells play important roles in sustaining maternal investment in offspring after birth by influencing key systems like milk production, body temperature regulation, and mother-infant bonding.

Expecting the Unexpecting: Compassionate care for your pregnancy loss patients

Rachel Lewis, USA

Pregnancy loss is a medical event that has no cure, and no treatment can mitigate the emotional devastation of loss. However, you have the power to offer compassionate care and ultimately prevent further trauma. In this talk, you will gain insight into what your pregnancy loss patients really need (hint: It's not making them feel better about

their loss); how, as a medical provider, you are specifically able to help them process their loss; and how you can teach them to advocate for their future medical needs with pregnancy loss as an important piece of their medical history.

Reflections on microchimerism after pregnancy loss

Rachel Tompa, USA

When I first learned about microchimerism, I'd been pregnant five times: three miscarriages before bearing two living children. I was working as a science writer at Fred Hutchinson Cancer Center, where J. Lee Nelson, MD — who has made seminal discoveries about this phenomenon of parent-child cell-sharing — is a faculty member. The idea that I had living cells remaining in my body from my two children fascinated me, but I was even more astonished when I came across a paper Nelson and her colleagues wrote titled Microchimerism in recurrent miscarriage. My diagnosis, for the three miscarriages I had before getting pregnant with my oldest child, was idiopathic recurrent miscarriage. Despite the many tests I was subjected to, my doctors didn't know why I kept losing pregnancies. That mystery was never solved, but here was a new mystery: Could I really have five different sets of cells from my five pregnancies persisting in my body? In this talk, I'll describe what we know about miscarriage and microchimerism, including the many remaining unknowns, and describe my struggle to understand what my body was going through during my period of loss.

Breastfeeding and bonding

Anna M. Rath

University of Applied Sciences, Graz, Austria &

Kathrin Kroneis

Intersections of Microchimerism and Midwifery Practice

Midwifery is a profession that provides continuous care throughout the reproductive phases, including pregnancy, birth, the postpartum period and lactation. This holistic approach integrates physiological support with emotional and psychosocial guidance for families. Key themes include the in-utero connection, maternal–infant bonding, and breastfeeding as a continuation of this bond. Another important theme is the emotional relationship that develops between mother and child.

While midwifery focuses on fostering and supporting these pro-

cesses, research on microchimerism explores how this connection manifests at a cellular level, examining the persistence and exchange of maternal and foetal cells across biological boundaries. This panel will explore the intersection between these two fields of study.
By combining cellular-level research with the clinical and emotional aspects of midwifery, the discussion will highlight the shared objective of both disciplines: to understand and support the profound biological and emotional bond between mother and child. We propose that integrating these two areas of study can generate new transdisciplinary knowledge, linking the microscopic with the deeply human aspects of birth and care.

Q&A

5:30 pm – 7:00 pm Session 3
Hidden Guest

These cells that are not our own

Lise Barnéoud, France

Following the success of her book Hidden Guests and in an effort to popularize science to the widest possible audience, French science journalist Lise Barnéoud will give a lecture on microchimerism. Starting from the discovery of these cellular interminglings, she will interweave science discoveries with human stories. This talk will offer a new perspective on our biology, on our immune system, and ultimately allows us to glimpse a new way of being in the world.
For this special event, the public will also be invited to participate and the lecture will continue with a discussion among some of the world's leading experts attending the conference.

Expert panel discussion incl. Q&A

This lively panel discussion will bring together several experts, with different perspectives. It will explore the latest researches, the unresolved questions, the challenges and future prospects for microchimerism. At the end, a question-and-answer session will give you a chance to engage directly with these experts.

Summary & meeting with the speaker by wine and soup (until 9 pm)

International Conference on Microchimerism

The interdisciplinary dimension of microchimerism research is the central interface between immunology, genetics, transplant medicine, cell biology and other related disciplines.

May 27 - 28, 2026
Medical University of Graz

Sessions

- Microchimerism Across the Lifespan: Concepts, Mechanisms, and Clinical Frontiers
- Microchimerism, an infection-sensitive system influencing immune protection, tolerance, and long-term disease susceptibility across generations
- (Micro)Chimerism & Treatment: Immune Tolerance, Transplantation, and Precision Therapeutics
- Microchimerism and Kinship: Evolutionary Conflict, Tolerance, and Cellular Competition
- Immunology, Transplantation & Diseases
- Microchimerism and Autoimmunity: Genetic Risk, Immune Recognition, and Familial Context
- Microchimerism in Reproductive Health: From Infertility and Placental Dysfunction to Cardiovascular Risk
- Microchimerism in Immune Tolerance and Neurodevelopment: Context-Dependent Persistence and Pathophysiology

Speakers

Diana W. Bianchi, USA
Amy Boddy, USA
Michael Eikmans, The Netherlands
David Haig, USA
Whitney Harrington, USA
Natalie Lambert, France
Dennis Lo, China - Keynote
J. Lee Nelson, USA
Henriette Svarre Nielsen, Denmark
Eitan Okun, Israel
Annetine Staff, Norway
Anne M. Stevens, USA
Sing Sing Way, USA

Program & registration

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International Conference on Microchimerism
DAY 1: Wednesday, May 27, 2026

8:30 am Welcome coffee

9:00 am Welcome note

- Andrea Kurz, Rector, Medical University of Graz
- Thomas Kroneis, Conference President

9:20 am Microchimerism Across the Lifespan: Concepts, Mechanisms, and Clinical Frontiers

Chairs: Diana W. Bianch, J. Lee Nelson,

- Diana W. Bianchi, USA, The Past, Present and Future of Microchimerism.
- J. Lee Nelson, USA, Microchimerism in AutoImmunity and AlloImmunity Over Time.
- Short talk: Kristine Joy Chua, USA, What is Microchimerism? Defining and refining its characteristics.

10:30 am Coffee break & Industry exhibition

11:00 am Microchimerism, an infection-sensitive system influencing immune protection, tolerance, and long-term disease susceptibility across generations

Chairs: Whitney Harrington, Frank Schildberg

- Whitney Harrington, USA, The intersection of maternal microchimerism and infectious disease.
- Short talk: Samantha de Freitas Cavalcante, Brasil, Gestational ZIKV exposure increases fetal microchimerism, including neural cells.
- Short talk: Isabel Graf, Germany, Placental extracellular vesicles indicate the vertical transfer of maternal microchimeric cells to the fetus in healthy and infection-affected pregnancies.
- Short talk: Gitte Lindved Petersen, Denmark, Maternal microchimerism at birth associates with reduced odds of non-malarial fever and respiratory tract infections in Tanzanian children.

12:15 pm Company talk sponsored by Evident

- Amin El-Heliebi, Austria, Spatial imaging technologies for the detection of rare cells and tracing variants to their tissue of origin.

12:30 pm Lunch & Poster session

Poster session

Chairs: Hyun-Dong Chang, Melissa Wilson

2:00 pm (Micro)Chimerism & Treatment: Immune Tolerance, Transplantation, and Precision Therapeutics

Chairs: Michael Eikmans, Katja Sallinger

- Michael Eikmans, The Netherlands, Consequences of pregnancy-derived microchimerism for outcome after transplantation
- Anne M. Stevens, USA, Strategies to test the role of microchimerism in autoimmune disease through antigen-specific therapeutic development
- Short talk: Blair Armistead, USA, A novel approach to investigate breastmilk T cell antigen-specific responses
- Short talk: Yoan Ghaffari, France, Is the skin the preferred target of twin microchimerism in mice in all cross combinations: allogeneic, congenic and semi-allogeneic?
- Short talk: Bernadette L. Bramreiter Austria, An optimized flow cytometry-based method for the isolation of potential microchimeric maternal cells in human mesenchymal stem cells using monoclonal HLA class I specific antibodies.

3:45 pm Coffee break & Industry exhibition

4:30 pm Microchimerism and Kinship: Evolutionary Conflict, Tolerance, and Cellular Competition

Chairs: Amy M. Boddy, Kristine Chua

- Amy M. Boddy, USA, Consequences of being microchimeric
- Tiffany Pan, USA, Whose fitness is it anyway? Maternal-origin microchimerism and the reach of selection
- Short talk: Ashley McDonough, USA, Detection and transcriptional profiling of microchimerism in the brain using snRNAseq data reveals prevalent microchimerism and diversity of maternal cell fates

5:30 pm Change location for Meet the Speaker event

Advance booking required.

DAY 2: Thursday, May 28, 2026

8:30 am Welcome coffee

9:00 am Keynote Lecture by Dennis Lo - Immunology, Transplantation & Diseases

Chair: Thomas Kroneis

- Y.M. Dennis Lo, China, Recent developments in cell-free DNA-based diagnostics

10:00 am Company talk sponsored by SomaScan

- Thomas Bauer, Sr. Executive Terrtory Account Manager, Germany, Proteomics and Beyond - Combining Protein- and Immuno-Profiling to reveal novel biological insights

10:15 am Coffee break & Industry exhibition

11:00 am Microchimerism and Autoimmunity: Genetic Risk, Immune Recognition, and Familial Context

Chairs: Nathalie C. Lambert , Henderson Cleaves

- Nathalie C. Lambert, France, Could Cells from Our Mother, Children, and/or Siblings Participate in Immune Reactions Mistakenly Labeled as Autoimmunity?
- Short talk: Haynes Heaton, USA, Cellector: A tool to detect foreign genotype cells in scRNAseq data with applications in leukemia and microchimerism.
- Short talk: Gitte L. Petersen Denmark, Circulating male origin microchimerism in Danish girls with and without type 1 diabetes
- Short talk: Tine Dreier Bille, Denmark, Predictors of maternal and male-origin microchimerism in peripheral blood of Danish youths.

12:15 pm Lunch & Poster session & Industry exhibition

Postersession

Chairs: Hyun-Dong Chang, Melissa Wilson

1:45 pm Microchimerism in Reproductive Health: From Infertility and Placental Dysfunction to Cardiovascular Risk

Chairs: Anne Cathrine Staff, Herbert Fluhr

- Henriette Svarre Nielsen, Denmark, Microchimerism in infertility and pregnancy loss
- Anne Cathrine Staff, Norway, Women's risk of cardiovascular disease after pregnancy complications: does a dysfunctional placenta and fetal microchimerism play a role?
- Ina A. Stelzer, Germany, Fetal Microchimeric Cell Retention Following Preeclampsia
- Short talk: Bernadette L. Bramreiter, Austria, Characterization of human amniotic fluid stem cells and their potential role in maternal microchimerism.

3:15 pm Coffee break & Industry exhibition

4:00 pm Microchimerism in Immune Tolerance and Neurodevelopment: Context-Dependent Persistence and Pathophysiology

Chairs: Thomas Kroneis, J. Lee Nelson

- Sing Sing Way, USA, Immune tolerance to mothers and maternal microchimeric cells
- Eitan Okun Israel, Microchimerism during Down Syndrome pregnancies causes maternal cognitive decline
- Short talk: Christopher Urbschat, Germany, Influenza-induced alterations of maternal microchimeric cell composition and function in brains of fetal mice
- Short talk: Giang Pham, USA, Complement-producing maternal microchimeric cells override infection susceptibility in complement-deficient murine offspring

5:20 pm - 5:40 pm Closing remarks, farewell and award ceremony

Abstracts

9:00 am - 9:20 am Welcome note by Andrea Kurz, Rector Medical University Graz, Thomas Kroneis, Conference President

9:20 am - 10:30 am Microchimerism Across the Lifespan: Concepts, Mechanisms, and Clinical Frontiers

Microchimerism challenges the classical view of biological individuality by demonstrating that genetically distinct cells and biological material can be exchanged bidirectionally—most prominently during pregnancy—and persist for decades. From early histopathological observations to modern molecular and genomic validation, the field has expanded into an interdisciplinary arena encompassing immunology, reproduction, transplantation biology, and evolutionary science. Individuals are increasingly recognized as cellular mosaics containing intact cells, cell-free nucleic acids, and extracellular vesicles derived from kin. As empirical evidence has grown, so too have conceptual debates. Efforts to refine definitions address questions of persistence, functional relevance, cellular versus subcellular components, and distinctions between naturally acquired and artificially introduced forms. Mechanistically, microchimerism might add a spin to the concept of autoimmunity towards alloimmunity, shaped by HLA relationships, age, and reproductive history. Associations with autoimmune disease modulation, malignancy risk, and transplantation outcomes underscore its translational relevance and position microchimerism as an evolutionarily embedded determinant of immune regulation and human health. This session introduces microchimerism, introduces its impacts on various other fields of research and is set up to provide a scaffold for discussion throughout the conference.

The Past, Present and Future of Microchimerism

Diana W. Bianchi

NIH/NHGRI, Washington D.C., USA

Originally studied 133 years ago by the pathologist Schmorl as a mechanism to understand eclampsia-related deaths during pregnancy, fetal cells in maternal blood have more recently garnered attention as a noninvasive source of fetal material for prenatal testing. In the 21st century, however, intact fetal cells have been replaced by circulating cell-free placental DNA for fetal aneuploidy screening. Instead, interest has pivoted to the ways in which fetal cells positively and negatively influence maternal health. At the same time, an increasing appreciation of the consequences of maternal cells in the developing fetus has occurred. In my introductory overview lecture, I will highlight the potential clinical and functional consequences of the bidirectional trafficking of intact cells between a pregnant woman and her fetus. Fetal

cells play a potential role in the pathogenesis of maternal disease and tissue repair. Maternal cells play an essential role in educating the fetal immune system and as a factor in transplant acceptance. Although these appear to be universal phenomena they have been largely underappreciated by the general scientific community. Future investigations in humans need to include complete pregnancy histories to understand maternal health and transplant successes or failures. Animal models are useful to understand the mechanisms underlying fetal wound healing and/or repair associated with maternal injury and inflammation. The lifelong consequences of the exchange of cells between a mother and her child are profound and have many applications in development, health, and disease. This intricate exchange of genetically foreign cells potentially creates a permanent connection that contributes to the survival of both individuals.

Microchimerism in AutoImmunity and AlloImmunity Over Time

J. Lee Nelson

Department of Medicine, University of Washington, Seattle, WA, USA
Translational Science and Therapeutics Fred Hutchinson Cancer Center

Naturally acquired microchimerism (Mc) is creatively pleotropic and protean with beneficial and potentially detrimental consequences for an individual's health. Mc creates a venue for forward, reverse, and horizontal inheritance, with long-term persistence after bi-directional maternal-fetal exchange, exchange between twins, and among littermates in dogs and mice. HLA molecules function in multiple key roles to maintain an individual's health, discerning harmful infections, preventing harmful autoimmunity, and maintaining healthy alloimmunity. While mechanisms by which HLA molecules predispose or protect from an autoimmune disease are not fully understood, it is clear that age matters. The same HLA molecule can even predispose to different diseases at different lifespan times. Mc is of special interest because it is most often only HLA-haploidentical with the hosting individual. Mc is also time-dependent with potentially different impact as an individual and the Mc age (and whether Mc was acquired in utero or in adult life). Women acquire Mc during pregnancy and a woman's reproductive history clearly impacts numerous aspects of subsequent health, including autoimmunity, cardiovascular disease, and risk of some malignancies. An example of the time-dependence of parity is reduction of rheumatoid arthritis risk with protection diminishing as time elapses from a birth, vanishing by ten years. Time elapsed from a birth is also a factor in protection against some malignancies such as breast cancer. At present, Mc in healthy alloimmunity against malignancies is relatively underexplored, but clear benefit of donor-recipient HLA-disparity is well established in the setting of hematopoietic cell transplantation (HCT). Further intriguing is decreased leukemia relapse rate after HCT when cord blood is the donor product, implicating HLA-mismatched maternal Mc in cord blood. Looking to the future, Mc and HLA-relationships are there-

fore of particular interest to understand the interface of autoimmunity and healthy alloimmunity and for development of novel preventative and therapeutic interventions.

Acknowledgements
The author's work was supported by NIH grants HL-117737, AI-45659, the Washington Women's Foundation and the Wong Foundation.
Competing interests
JLN is a co-founder of Chimerocyte, Inc. that develops highly sensitive chimerism analysis technologies.
Chimerocyte, Inc. had no role in funding this research.

What is Microchimerism? Defining and refining its characteristics

Short talk: Kristine Joy Chua

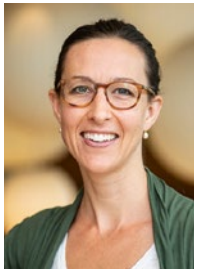
Department of Anthropology, University of Notre Dame, Notre Dame, USA
Rachel C. Quilang, Department of Immunology, Leiden University Medical Center, Leiden, Netherlands
Katja Sallinger, Department of Cell Biology, Histology & Embryology, Gottfried Schatz Research Center, Medical University of Graz, Graz, Austria
Melissa A. Wilson, National Human Genome Research Institute, National Institutes of Health Bethesda, Bethesda, USA
Thomas Kroneis, Department of Cell Biology, Histology & Embryology, Gottfried Schatz Research Center, Medical University of Graz, Graz, Austria
Amy M. Boddy, Department of Anthropology, University of California Santa Barbara, Santa Barbara, USA

Bidirectional cell exchange between mother-fetal dyads during pregnancy may result in a small amount of biological material, derived from another genetically distinct germline, that can persist in the host body for decades. Although this biological phenomenon has been documented for over half a century, its conceptual boundaries remain difficult to define. Many researchers refer to this as microchimerism. Yet, as this field expands, so too do ongoing disagreements and inconsistencies over what is and is not considered microchimerism. To address these existing debates, the Microchimerism, Human Health and Evolution Project launched a campaign to solicit perspectives from a range of experts working in microchimerism research, with the goal of working toward a shared consensus of how microchimerism is defined and which characteristics are required. One of the primary areas where clear disagreements arise stems from whether subcellular components, particularly extracellular vesicles and cross-decoration, should be included. Additional properties that have been cited are source of origin, potential for functionality, persistence versus transient, naturally versus artificially occurring, and amount of material. In this talk, we put forth a definition that accounts for these areas of debate, as well as edge cases that challenge rigid definitional boundaries. Our goal is not to adjudicate which per-

spectives are correct, but to work as a community to establish clear assumptions and decisions as to the characteristics and properties of microchimerism. Ultimately, we aim to work toward a standardized definition of microchimerism and provide greater clarity in the language used to discuss it. As microchimerism research continues flourishes, we hope that a clearer definition will help alleviate confusion and facilitate advancements in the field.

11:00 am – 12:15 pm Microchimerism, an infection-sensitive system influencing immune protection, tolerance, and long-term disease susceptibility across generations?

Maternal and fetal microchimerism emerge as dynamic mediators at the interface of infection, immune tolerance, and developmental programming. Vertical transfer of maternal immune cells during gestation—and potentially via breastfeeding—impacts the adaptive arm of the offspring's immune system linking maternal microchimerism at birth to enhanced vaccine responsiveness and reduced susceptibility to infectious diseases in early childhood. Interestingly, infectious challenges during pregnancy can reshape bidirectional cell trafficking, impact microchimeric subpopulations and shed light on immune recognition. Importantly, technical advances in isolation, transcriptional profiling, and functional expansion of microchimeric immune cells foster these analyses. And with placental extracellular vesicles showing infection-associated changes in vesicle cargo an interesting subcellular component lines up for biomarker status. This session covers microchimerism as an infection-sensitive system influencing immune protection, tolerance, and long-term disease susceptibility across generations.



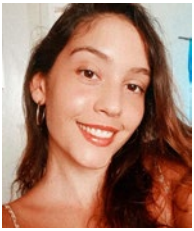
The intersection of maternal microchimerism and infectious disease

Whitney Harrington

University of Washington & Seattle Children's Research Institute, USA

Mothers transmit cells to their offspring in utero and likely via postnatal breastmilk exposure. The transmission of these cells is impacted by maternal immune stimuli in pregnancy including placental malaria, peripheral malaria, HIV, and potentially vaccination. Prior work has demonstrated that these cells are enriched for memory T cell populations, and we hypothesize that they may directly and indirectly impact fetal and postnatal immunity in the offspring. For example, we have found that maternal microchimerism at birth is associated with augmented response to Bacillus Calmette–Guérin (BCG) vaccination and reduced susceptibility to symptomatic malaria, respiratory infection, and non-malaria fever. To better understand the mechanism of this protection, we have recently developed an approach to isolate rare maternal

T cells from the offspring and investigate their transcriptional profile. Using this approach and others, we have identified Falciparum malaria-, cytomegalovirus- (CMV), and mycobacterium-specific T cells in select offspring, representing the direct transmission of cellular pathogen-specific immunity. In addition, we have recently developed an approach to massively expand maternal microchimeric T cells to better enable functional screening for potential antigen specificity. Further, we have identified an associated between the presence of maternal cells at birth and altered prenatal immune priming against malaria, suggesting that maternal microchimerism may also indirectly impact fetal immunity. These data suggest that maternal microchimerism plays an important and underappreciated role in providing "active" protection to the offspring against infection.



Gestational ZIKV exposure increases fetal microchimerism, including neural cells

Short talk: Samantha de Freitas Cavalcante

Samantha de Freitas Cavalcante[1,2]; Carolina Mendes Aguiar[3]; Fabio Barrozo Canto[4]; Selma Maria Bezerra Jerônimo[3]; Maria Bellio[1]; Eduardo Bouth Sequerra[5]

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Fetal microchimeric cells contribute to maternal immune tolerance to fetal antigens. Infectious challenges, however, may disrupt tolerance and elicit immune responses against the fetus. Congenital Zika virus (ZIKV) infection is linked to severe fetal neurological abnormalities, yet the contribution of maternal immune responses to the phenotypic variability observed in the Congenital Zika Syndrome (CZS) remains poorly understood. Here, we investigated whether congenital ZIKV infection alters the.
To assess fetal microchimerism, heterozygous β-actin–GFP males (B6-GFP) were crossed with wild-type C57BL/6 females, enabling detection of embryo-derived cells in maternal circulation. Maternal blood was collected at 5 days post-infection (dpi; injection in the amniotic fluid, 103 PFU) and analyzed by flow cytometry. ZIKV-infected dams showed increased levels of fetal microchimeric cells compared to controls, with neural stem cells detected exclusively in infected females. Also, the brains of GFP– embryos show an increase in the number of cells from their siblings.
By using a mouse with young neurons expressing TdTomato, we corroborated the above finding by showing that ZIKV infection leads to the detection TdTomato⁺ cells in the maternal blood. All TdTomato+ cells express CD14, suggesting that these neurons undergo phagocytosis.

Finally, analyzing BALB/c females crossed to B6-GFP males and intraperitoneally inoculated with ZIKV (10^6 PFU), we observed increased fetal cells (H-2b/H-2d double positive cells) in the circulation and in secondary lymphoid organs.
Moreover, analyzing BALB/c females crossed to B6-GFP males and intraperitoneally inoculated with ZIKV (10^6 PFU), we observed that fetal cells (H-2b/H-2d double positive) seem to accumulate mainly in the spleen of the dam. Together, these findings indicate that congenital ZIKV infection reshapes fetal microchimerism by increasing permeability between the embryonic nervous system and the mother, which potentially impacts on maternal immune tolerance towards fetal antigens during infection.

Placental extracellular vesicles indicate the vertical transfer of maternal microchimeric cells to the fetus in healthy and infection-affected pregnancies

Isabel Graf

Isabel Graf[1,2], Christopher Urbschat[1,2], Bente Siebels[3], Christian Müller[4], Anke Diemert[5], Petra Arck[1,2]

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Background: During pregnancy, maternal (immune) cells are vertically transferred to the fetus. These cells can persist in the offspring, then referred to as maternal microchimeric cells (MMc). There is mounting evidence that MMc can impact the child's susceptibility to diseases later in life. However, little is known about driving factors and mechanisms of MMc trafficking. This study investigates placental extracellular vesicles (EVs) as messenger of MMc transfer in healthy and infection-affected pregnancies.
Methods: The proteome of serum-derived EVs from healthy and SARS-CoV-2-infected pregnant women was analyzed by imaging flow cytometry and mass spectrometry and correlated with MMc frequencies in cord blood. Murine models were used to validate proteins modulating MMc trafficking.
Results: Maternal SARS-CoV-2-infection led to decreased MMc transfer rates. This was associated with an increased placental EV secretion, along with an altered protein cargo, including downregulation of PSME1, an immunoproteasome component. In a murine model we proof that PSME1 is associated with MMc.
Conclusion: Placental EVs were identified as biomarkers indicating trafficking and regulation of MMc in response to adverse conditions.

Maternal microchimerism at birth associates with reduced odds of non-malarial fever and respiratory tract infections in Tanzanian children

Gitte L. Petersen

Gitte L. Petersen[1,2], Paul T. Edlefsen[3], Xiaohong Li[4], Robert Morrison5, Edward Kabyemela[6], J. Lee Nelson[7,8], Patrick E. Duffy5, Michal Fried[5], Whitney E. Harrington[9,10,11]

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Presenting author: Gitte Lindved Petersen, MSc, PhD

The presence of maternal cells in infant cord blood, a phenomenon known as maternal microchimerism, has been previously associated with malaria and respiratory infections in early childhood suggesting a role in immunological responses to infections. Here, we assess the association between cord blood maternal microchimerism and non-malarial infections in Tanzanian children.

We conducted a secondary analysis using a nested birth cohort of 52 children from Muheza, Tanzania, with previously measured cord blood maternal microchimerism and longitudinal records on infections in the first four years of life. The associations between maternal microchimerism and lower and upper respiratory tract infections, diarrhea, and non-malarial fever were estimated using generalized estimating equation models.

In total, 29% of the 52 children in the study screened positive for cord blood maternal microchimerism. Detected versus non-detected maternal microchimerism was associated with 58% lower odds of non-malarial fever (fully adjusted odds ratio (OR): 0.42

[95% CI: 0.18-0.98]) and 28% lower odds of RTI (OR: 0.72 [95% CI: 0.53-0.96]). Lower and upper respiratory tract infections contributed equally to the observed association with any respiratory tract infections (ORs respectively: 0.81 [95% CI: 0.50-1.31] and 0.71 [95% CI: 0.50-1.01]). We did not find any association between maternal microchimerism and odds of diarrhea (OR: 1.63 [95% CI: 0.85-3.13]).
Detectable cord blood maternal microchimerism was associated with lower odds of non-malarial fever and respiratory infections in Tanzanian infants. These findings emphasize that MMc may play an underrecognized role in protection from infection during early childhood.

12:15 pm - 12:30 pm Company talk by Evident

Spatial imaging technologies for the detection of rare cells and tracing variants to their tissue of origin

Amin El-Heliebi

Medical University Graz

The detection of microchimerism remains a significant analytical challenge due to the extreme rarity and low abundance of microchimeric cells in blood and tissue samples. Recent advances in spatial and imaging technologies now enable the simultaneous assessment of multiple molecular markers, thereby substantially improving specificity and sensitivity. Emerging approaches such as open source in situ sequencing further expand these capabilities by allowing not only the characterization of gene expression profiles, but also the detection of single nucleotide variants (SNVs), including single nucleotide polymorphisms (SNPs) and somatic mutations. In this presentation, I will discuss the application of a novel multicolor imaging workflow based on the VS200 Scanner from Evident. I will highlight how this platform can be utilized for the detection of single nucleotide variants as well as the identification of rare circulating cells in blood samples, providing new opportunities for microchimerism research and diagnostics.

12:30 pm Lunch & Poster session & Industry exhibition

2:00 pm - 3:45 pm (Micro)Chimerism & Treatment: Immune Tolerance, Transplantation, and Precision Therapeutics

Pregnancy-derived microchimerism exerts durable immunological and clinical consequences that extend from early development to transplantation and autoimmunity. Bidirectional trafficking of maternal and fetal cells establishes antigen-specific tolerance or sensitization shaped by HLA disparity, with implications for transplant compatibility, graft-versus-leukemia effects, and longterm immune regulation. Mechanistic frameworks linking non-inherited maternal antigens and inherited paternal antigens to immune education underscore how gestational exposure can promote either tolerance or pathogenic alloimmunity. In parallel, emerging antigen-specific therapeutic tools like CAR T cells provide experimental avenues to directly test the contribution of rare chimeric populations to autoimmune disease. Technological innovation is central to these advances: high-resolution single-cell transcriptomics and novel bioinformatic tools now enable detection of exceedingly rare foreign-genotype cells across blood, tissues, and transplant samples, revealing diverse cellular fates and lifelong persistence. Expansion and characterization of microchimeric populations further suggests developmental routes for durable engraftment and biological functionality. This interdisciplinary session highlights microchimerism as a biologically embedded determinant of immunity, disease risk, and therapeutic opportunity and provides insight into fascinating technological developments.

Consequences of pregnancy-derived microchimerism for outcome after transplantation

Michael Eikmans

Leiden University Medical Center, The Netherlands

Microchimerism can cause immune recognition between mother and fetus. Differences in human leukocyte antigen (HLA) genes between two hosts is a main driver of immune reactions. The child inherits one set of HLA genes from the mother and one from the father. The mother may develop immunity by forming antibodies and effector- and regulatory T cells, which are specific against the inherited paternal antigens (IPA) of the fetus. Vice versa, T cells from the fetus may show reactivity to non-inherited maternal antigens (NIMA).

Likewise, exposure of a recipient to a transplanted organ or cells from a genetically different individual leads to immune reactions. Previous studies have emphasized the possible consequence of microchimerism for outcome of transplantation performed later in life. For instance, immune recognition by the pregnant woman may lead to HLA antibody development, forming a hurdle when a transplant is given containing HLA antigens to which those antibodies are directed to. Alternatively, maternal ex-

posure during pregnancy in the womb leads to immune tolerance. If a patient, having developed tolerance toward the NIMA, is offered a donor kidney containing a mismatched antigen that is the same as the NIMA, there is no significant negative impact on transplant outcome.

In this lecture I focus on maternal microchimerism (mMC) in the fetus. Umbilical cord blood (UCB) from the newborn can be used as a source for cell transplantation in patients suffering from leukemia. Clinical studies provided indirect evidence that mMC cells in fetal blood mediate graft-versus-leukemia effects in the recipient after UCB transplantation. Attempts for enriching these cells are discussed along with questions including: which cell types are these chimeric cells? How are these chimeric cells maintained and not cleared by the host's immune system? Why would these cells not directly attack host cells, but would exert alloreactivity in a recipient?

Strategies to test the role of microchimerism in autoimmune disease through antigen-specific therapeutic development

Anne M. Stevens

Executive Medical Director, Century Therapeutics
Adjunct Clinical Professor, Pediatric Rheumatology, Stanford University
Retired Professor, Pediatric Rheumatology, University of Washington
Attending Physician, Pediatric Rheumatology, Renown Regional Medical Center, Reno, NV

Maternal and fetal microchimerism (MMc, FMc) derived during pregnancy have been associated with various aspects of health and disease. In the context of interactive genetic backgrounds, both FMc and MMc have been implicated in the triggering and perpetuation of chronic autoimmune diseases. Functional studies in vitro and in mouse models support a role for loss of allogeneic T cell regulation of fetal-maternal tolerance contributing to chronic inflammation. Novel strategies to target cells with an antigen-specific CAR T cell or bispecific large molecule therapeutics opens up a pathway toward treatment of autoimmunity via depletion of small numbers of pathogenic chimeric cells expressing non-inherited maternal or fetal antigens. Innovative therapeutic development approaches targeting antigen-specific T and B lymphocytes will be discussed with relevance to applying technologies to definitively test roles of MMc and FMc in the pathogenesis of autoimmune disease.

A novel approach to investigate breastmilk T cell antigen-specific responses

Short talk: Blair Armistead

Yonghou Jiang[1], Jennifer E. Stolarczuk[2], Sharon Kung[2], John Houck[1], Victoria L. Campbell[3], David M. Koelle[3,4,5], Alisa Kachikis[2], Whitney E. Harrington[1,7,8], Blair Armistead[1,8]

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Breastmilk plays a pivotal role in infant health and development, providing both nutritional and immunological benefits. In addition to protective antibodies, breastmilk contains immune cells, including T cells, which have an unknown role in human infant immunity. However, multiple studies in animals have shown that milk-derived T cells can traffic to peripheral organs of nursing offspring as a form of maternal microchimerism (MMc) and provide protection from infection. Further study is needed to understand the potential of breastmilk T cells to exert pathogen-specific effector functions at the maternal-infant interface. To-date, direct ex vivo functional analyses and T cell receptor sequencing of breastmilk T cells have been limited by low T cell frequency. To overcome this barrier, we optimized a method to expand breastmilk T cells in culture using cell sorting and mitotic stimulation. With this approach, we generated expanded breastmilk T cell (EBM T cell) cultures from n=4 lactating women, resulting in a ~1,000-fold increase in T cells from the original sample. Because each donor reported receipt of SARS-CoV-2 (SARS2) mRNA vaccination and/or history of SARS2 infection, we used SARS2 Spike as a model for detecting antigen-specific responses in EBM T cell cultures. In EBM T cell cultures from 2 of 2 donors tested, we identified SARS2 Spike-specific CD8+ T cells using an HLA-matched, Spike-loaded tetramer. To assess antigen-specific functional responses, we stimulated EBM T cells from one donor with a SARS2 Spike peptide pool, using autologous irradiated PBMC for antigen presenting function. Spike-stimulated EBM T cells contained CD4+ and CD8+ T cells expressing activation-induced markers, suggesting that their antigen-specific functionality was preserved. Together, our findings show that our novel approach is a

valuable platform for investigating pathogen-specific responses in breastmilk T cells, with relevance to the study of breastmilk-derived MMc in infants.

Is the skin the preferred target of twin microchimerism in mice in all cross combinations: allogeneic, congenic and semi-allogeneic?

Short talk: Yoan Ghaffari

Yoan GHAFFARI[1], Chahinez ARIF[1], Mathilde GIASSI[1], Marielle MARTIN[1], Isabelle AUGER[1], Catherine DUEZ[1] and Nathalie LAMBERT[1].
INSERM U1097 ARTHEMIS Aix Marseille University, France

During pregnancy, bidirectional cell exchange—especially between twins—represents a significant but often overlooked source of microchimerism. This mouse study investigates two key questions: What are the preferred niches for littermate microchimeric cells (LMc), and what factors influence the quantity of cells transferred between twins?

Previous work by the team showed that an embryo's location in the uterine horn affects the amount of LMc it receives. Here, we examine how genetic relationships (allogeneic, semi-allogeneic, congenic) influence the passage and persistence of LMc from embryonic to adult stages. We use crosses where the father is heterozygous for the TdTomato fluorescence gene, allowing tracking of TdTomato+ LMc cells in offspring that did not inherit the gene. Crosses are as follows : C57BL6 X C57BL6-Tom+ (congeneic), DBA/2 X C57BL6-Tom+ (allogeneic), and D2BL6 X C57BL6-Tom+ mice, with respectively 6, 17, and 22 offspring dissected at embryonic, young adult, or older adult stages. About 20 tissues per offspring are tested for TdTomato presence via quantitative PCR, and organ sections are preserved for microscopy.

Preliminary results show that, as previously observed in semi-allogeneic crosses, the skin is the preferred niche for LMc in allogeneic crosses with respectively 4/4 embryos, and 5/5 of young adults. Congenic crosses have not yet been tested. Further analysis is needed to characterize these skin-resident cells. These findings echo earlier lab work identifying cells from a vanished twin in a 40-year-old man with scleroderma-like syndrome, suggesting that twin microchimerism may play a role in skin-targeted autoimmune diseases such as scleroderma.

An optimized flow cytometry-based method for the isolation of potential microchimeric maternal cells in human mesenchymal stem cells using monoclonal HLA class I specific antibodies

Short talk: Bernadette L. Bramreiter

Bernadette L. Bramreiter[1], Rachel C. Quilang[2], Carin van der Keur[2], Anne Wagenmakers[2], Katja Sallinger[1], Emiel Slaats[1], Julia Schönberger[1], Katharina Schuch[1], Hyun-Dong Chang[3,4], Dave Roelen[2], Thomas Kroneis[1], Michael Eikmans[2]

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Objectives

Microchimerism (MC) is defined as the presence of a small population of genetically distinct cells originating from another individual. During pregnancy, cells are trafficking across the feto-maternal interface in both directions, resulting in maternal and fetal MC (mMC, fMC). Microchimeric cells have been reported to exhibit stem and progenitor cell-like properties, suggesting that stem cell compartments may contribute to MC persistence. However, the identity and trafficking pathways of the cells remain poorly understood. This study aimed to develop and optimize a sex-unbiased flow cytometry-based method to isolate viable potential mMC cells from human fetal mesenchymal stem cell (MSC) populations using monoclonal HLA class I-specific antibodies (HuMoAbs).

Methods

HuMoAbs of IgG and IgM isotypes were generated at LUMC from transformed B lymphocytes. Thirteen HuMoAbs targeting HLA-A and HLA-B (e.g. A3 and B8) were selected and validated for the separation of maternal and fetal cells. To improve population discrimination and detection sensitivity, maternal specific HuMoAbs were separately conjugated to PE or FITC and used simultaneously for staining. PE/FITC double-positive cells were considered of maternal origin.

4:30 pm – 5:30 pm Microchimerism and Kinship: Evolutionary Conflict, Tolerance, and Cellular Competition

Microchimerism situates evolutionary cooperation and conflict within the bodies of mammals, embedding kin-selected interests into maternal and offspring tissues. Cellular exchange during gestation introduces genetically distinct lineages whose fitness interests may align or diverge depending on relatedness, timing, and repro-

ductive context. Evolutionary models predict that offspring-derived cells residing in maternal tissues could influence resource allocation, interbirth intervals, and maternal physiology in ways that enhance their own genetic success, while maternal cells introduced during fetal immune development may shape tolerance with fewer long-term costs. This temporal asymmetry suggests distinct immunological and fitness consequences for fetal versus maternal microchimerism. Emerging evidence that newly acquired fetal microchimeric populations displace older ones challenges assumptions of cumulative tolerance and reframes persistence as a dynamic arena of cellular competition. Mathematical modeling further conceptualizes displacement as an adaptive strategy shaped by conflicting selective pressures between mothers and sequential offspring. This session covers microchimerism as an evolutionary mechanism balancing reproductive tolerance, immunological surveillance, and intergenerational conflict.

Consequences of being microchimeric

Amy M. Boddy

Department of Anthropology, University of California Santa Barbara, USA

Internal gestation creates a biological paradox: placental mammals must maintain immunological self-identity while hosting genetically distinct offspring. This challenge extends beyond pregnancy, cellular exchange during gestation (microchimerism) persists for decades, with fetal cells detectable in mothers and maternal cells in offspring throughout life. Mammalian evolution has navigated this paradox through adaptive compromises that facilitate internal gestation while potentially increasing disease vulnerability.

Microchimerism transferred during pregnancy may represent an evolved mechanism for acquired immunological tolerance. Gradual cellular exchange could facilitate tolerance to non-self antigens (e.g., paternal alleles in mothers, non-inherited maternal antigens in offspring) where large scale exposure could trigger rejection. However, this tolerance comes at a cost: the immunological tolerance required for gestation may constrain HLA diversity in mammals. Recent work suggests mammals are more vulnerable to cancer than other species, suggesting a fundamental trade-off between reproductive tolerance and immunological surveillance.

But do the costs and consequences of microchimerism fall equally on mothers and offspring? Building on Medawar's pioneering work on acquired tolerance, fetal and maternal microchimerism have fundamentally different evolutionary dynamics. Fetal cells colonize a mature maternal immune system, potentially triggering chronic low-grade immunological conflict, while maternal cells are introduced during fetal immune development when self-versus-non-self boundaries are still being established. This temporal asymmetry predicts greater immunological conflict from fetal microchimerism than maternal microchimerism and suggests greater costs on maternal health.

Whose fitness is it anyway? Maternal-origin microchimerism and the reach of selection

Tiffany D. Pan

Biodemography Lab Director, Center for Studies in Demography and Ecology
Affiliate Assistant Professor, Department of Anthropology
University of Washington, USA

Detection and transcriptional profiling of microchimerism in the brain using snRNAseq data reveals prevalent microchimerism and diversity of maternal cell fates

Ashley McDonough

Ashley McDonough[1], Sami B Kanaan[2], Reza Behboudi[4], Coline Gentil[3], Francesca Urselli[2], Dan Geraghty[2], Dan Eisenberg[5], Haynes Heaton[4], Jeff Ojemann[6,7], Jonathan R Weinstein[1,6], J Lee Nelson[2,8]
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Bi-directional maternal-fetal exchange during pregnancy creates long-term persistence of microchimerism (Mc) including in the human brain. However, Mc studies in human brain are limited due to difficulty in obtaining brain tissue concurrent with material for genotyping from related individuals that would allow for identification of microchimerism. To address this gap of knowledge, we obtained surgically resected brain tissue from medication refractory epilepsy patients (aged 28d to 18 years) and buccal swabs from family members. Employing a panel of highly sensitive and specific quantitative PCR assays targeting polymorphisms, mainly in HLA loci, we found a striking prevalence and quantity of maternal-origin Mc (MMc) in resected brain tissue. We next performed single nuclei RNA sequencing (snRNAseq) on tissues from a subset of these patients. We collaborated with colleagues to develop a bioinformatics software for finding rare cells in a disproportionate mixture of two allogeneic entities based on polymorphisms across scRNAseq data to identify Mc. Using this orthogonal method, Mc was identified in pediatric brain and showed strong transcriptional similarity to patient indigenous cells and thus localize alongside patient cells using stan-

dard UMAP methodology for clustering of similar cell types. We observed diverse fates acquired by microchimeric cells, including multiple neuronal subtypes, microglia, oligodendrocytes, astrocytes, and endothelial cells. Finally, we applied our methods to publicly available snRNAseq data from individuals with healthy brain development across the lifespan (gestational tissues up to 93 years). Mc was prevalent, detected in most individuals, suggesting persistence in the brain throughout life. Our work provides compelling evidence of the long-term persistence of Mc in human brain, with unprecedented detail into the transcriptional profile and identity of these cells, as provided by novel bioinformatic approaches.

6:45 pm Meet the speaker - networking event

The Meet the speaker – networking event will not take place at the university.

Thursday, 28.05.2026, Day 2

9:00 am – 10:00 am Keynote Lecture by Dennis Lo - Immunology, Transplantation & Diseases

Recent developments in cell-free DNA-based diagnostics

Y.M. Dennis Lo

Department of Chemical Pathology, The Chinese University of Hong Kong, Shatin, New Territories, Hong Kong SAR, China

Cell-free DNA in bodily fluids such as plasma offers exciting diagnostics opportunities, such as for noninvasive prenatal testing, cancer liquid biopsy and transplantation monitoring. Recent understanding of the characteristics of cell-free DNA, such as its end characteristics (including end motifs and jagged ends) and the existence of long cell-free DNA (up to kilobases in length) has offered new research and diagnostic opportunities. In this lecture, I shall review some of these recent developments, hoping that it would also have implications to research into chimerism.

10:00 am - 10:15 am Company talk by SomaScan

Proteomics and Beyond
Combining Protein- and Immuno-Profiling to reveal novel biological insights

Thomas Bauer

Sr. Executive Terrtory Account Managere (Germany)

Biomarkers are measurable indicators of normal biological function or indicate pathogenic processes or specify biological changes. They are mostly used as diagnostic, prognostic, or predictive tool. Typically, Biomarkers today are related to the protein field where Slow Off-Rate Modified Aptamers (SOMAmer) can be used for Protein-Profiling, reliably detecting up to 11.000 Protein targets at the same time in minimal sample volume, highly specific, precise, and reproducible.
An additional valuable information is to gather information about the Immuno-Profile by multiplex detection of Antibodies, precisely Autoantibodies using fully expressed, correctly folded proteins (KREX). The humoral immune system surveys the body for danger and marks potentially harmful proteins with antibodies. This includes aberrantly expressed proteins that often appear in diseases like Cancer, Autoimmunity, Infectious disease, or Neurology but also play a key role in the Microchimerism context.

11:00am - 12:15 pm Microchimerism and Autoimmunity: Genetic Risk, Immune Recognition, and Familial Context

Microchimerism questions the concept of autoimmunity adding an alloimmunity aspect to it as persistent, genetically distinct cells from mothers, offspring, and siblings also contribute to our immune networks. With epidemiologic data scientists try to understand the link of fetal- and male-origin microchimerism with diseases such as rheumatoid arthritis and type 1 diabetes that often reveal dose-response pattern suggesting that these rare cells may function as genetic risk modifiers or biomarkers, particularly within specific familial HLA constellations. Experimental models point to microchimeric cells from distinct sources as contributing to autoantibody production and inflammatory pathology. At the population level, efforts to identify predictors of maternal and male-origin microchimerism investigate the complexity of the associations with host and maternal factors. This session covers genetic epidemiology, immunopathogenesis, and family-based analyses to reexamine autoimmune disease through the lens of intergenerational cellular exchange.

Could Cells from Our Mother, Children, and/or Siblings Participate in Immune Reactions Mistakenly Labeled as Autoimmunity?

Nathalie C. Lambert

INSERM UMRs 1097 Arthritides, Microchimerism and Inflammations (ARTHEMIS), Aix Marseille University, Marseille, France

The idea that semi-allogeneic cells resulting from feto-maternal exchanges might play a role in autoimmune diseases was first suggested in 1893, when a German pathologist, Georg Schmorl, discovered fetal placental cells in the lungs of mothers who had died of eclampsia. A century later, in 1996, J. Lee Nelson revisited this hypothesis, asking whether autoimmune diseases might in fact be semi-alloimmune in nature. She sparked interest by demonstrating that the peripheral blood of women with scleroderma more frequently and abundantly contained male cells—presumed to be of fetal origin—than that of age- and pregnancy-matched controls. This phenomenon, known as fetal microchimerism (Mc), became the focus of extensive correlation studies linking Mc levels to various autoimmune conditions.

But does the mere presence of these cells in higher quantities among patients truly establish their causal role in disease? Could other cells arising from placental exchanges participate also ? Do maternal, fetal and sibling microchimeric cells exert pathogenic effects only within specific genetic contexts in families?

In a cohort of patients with rheumatoid arthritis, we have recently identified specific genetic patterns across three generations that may influence the levels of microchimerism. We, along with others, have shown that microchimerism can represent a genetic risk factor for developing the disease in hosts who do not carry a known predisposition. Finally, using a mouse model, we have analyzed the ability of different microchimeric sources to produce autoantibodies and to contribute to pathogenesis. Does this represent a worrying biological phenomenon—or, conversely, an exciting therapeutic opportunity?

Cellector: A tool to detect foreign genotype cells in scRNAseq data with applications in leukemia and microchimerism.

Haynes Heaton

Haynes Heaton[1,†,], Reza Behboudi[1,†], Colin Ward[1], Minindu Weerakoon[1], Sami Kanaan[2], Skylar Reichle[1], Nathan Hunter[1], Scott Furlan[2,3]*

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The existence of rare, genetically distinct cells can occur in various samples such as transplant patient samples, naturally occurring microchimerism between maternal and fetal tissues, and cancer samples with sufficient mutational burden. Computational methods for detecting these foreign cells are vital to studying these biological conditions. An application that is of particular interest is that of leukemia patients post hematopoietic cell transplant (HCT). In many leukemias, a primary therapy is HCT, after which, the primary genotype of the bone marrow and blood cells should be of donor origin. If cells exist that are of the patient's genotype and the cell type lineage of the particular leukemia, this is known as measurable residual disease (MRD). If the MRD is high enough, this may represent a relapse of the patient's leukemia. Furthermore, accurately estimating the MRD is important for driving clinical decision making for these patients. Using high throughput single cell RNAseq (scRNA-seq) such as drop-seq1, 10x Genomics2, Seq-Well3, InDrops4 among others, one can use the expressed genetic variants in the RNAseq reads to detect microchimeric cells. Unlike multiplexed single cell experiments, one cannot biochemically tag these cells for demultiplexing5,6. Tools made for demultiplexing cells by genotype such as souporcell7,8, vireo9, and scSplit10 rely on clustering based systems that don't perform well with highly skewed cluster sizes, which microchimerism has by definition. Other tools such as Demuxlet11 require knowledge of the genotypes up front which may be costly, not possible, or unavailable. Here we present Cellector, a computational method for identifying rare foreign genotype cells in single cell RNAseq (scRNAseq) datasets. Cellector uses a sparse beta-binomial anomaly detection method to identify cells with different genotypes than the majority of the sample. We show cellector accurately detects microchimeric cells down to an exceedingly low percentage of these cells present (0.05% or lower) even when the cells come from related individuals which represent the most common donors for HCT.
Cellector is freely available under an MIT open-source license at https://github.com/wheaton5/cellector.

Circulating male origin microchimerism in Danish girls with and without type 1 diabetes

Gitte L. Petersen

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Presenting author: Gitte Lindved Petersen, MSc, PhD

Male origin microchimerism (MOMc) has been associated with autoimmune disease in women. While typically attributed to prior male pregnancy, MOMc is not confined to parous women as 14% of nulliparous Danish girls have detectable MOMc in their circulation. We investigated whether MOMc is associated with type 1 diabetes (T1D) in Danish girls.

Our study population includes 34 female cases with T1D sampled from the Danish Registry of Childhood and Adolescent Diabetes and 262 female controls without T1D from the Lolland Falster Health Study. DNA purified from buffy coat was screened for the multi copy Y chromosome specific gene DYS14 using quantitative PCR, yielding binary indicators of MOMc presence and estimated cell quantities in genomic equivalents (GE)/100,000 GEs screened. The MOMc levels were categorized as 0, >0 to <1, and ≥1 GEs/100,000 GEs. T1D-associated genetic variants and covariates from nationwide registers are being obtained through ongoing linkage at Statistics Denmark.

MOMc was detected in 50% of girls with T1D (17/34) and 21% of controls (54/262). Among girls with vs. without T1D, 24% vs. 15% carried >0 to <1 MOMc GEs, and 27% vs. 5% carried ≥1 MOMc GEs. Crude logistic regression showed that the MOMc-positive girls had an odds ratio (OR) of T1D of 3.85 (95% confidence interval (CI): 1.84–8.09) compared with MOMc-negative girls. Compared to MOMc-negative girls, those with >0 to <1 MOMc GEs had an OR of T1D of 2.39 (95% CI: 0.92-5.76), and those with ≥1 MOMc GEs had an OR of 8.47 (95% CI: 3.11-22.68).

Detectable MOMc in peripheral blood was associated with higher odds of T1D in girls, with evidence of a dose-response pattern. If this is not due to confounding or selection bias, circulating MOMc may represent a potential biomarker of T1D. Ongoing analyses will assess the roles of T1D-associated genetic risk and other covariates.

Predictors of maternal and male-origin microchimerism in peripheral blood of Danish youths.

Tine Dreier Bille

Tine Dreier Bille[a],, Flemming Pociota, Randi Jepsen[b], Christina Ellervik[c,d,e], Sofie Dolores Holm Müller[f], Marianne Antonius Jakobsen[f], Gitte Lindved Petersen[a,e]*

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Identifying predictors of maternal (MMc) and male-origin microchimerism (MOMc) is key to understanding potential links to health outcomes. We examined predictors of MMc and MOMc in peripheral blood of Danish youths aged 15-20 years and assessed their associations.

We included 501 mother-offspring pairs from the Lolland-Falster Health Study (2016-2020). Survey, biochemical, and nationwide register data were linked via personal identification numbers. Information covered maternal factors (maternal age, educational level, spontaneous abortions, parity, smoking, hypertension, preeclampsia, accreta, gestational diabetes, autoimmune disease, asthma, caesarean section, preterm birth, and birth weight), and youth factors (baseline age, sex, asthma, tobacco use, and biochemical measurements (iron, creatinine, glucose, haemoglobin, lipids, thyrotropin, thyroxin, triiodothyronine, and albumin)). DNA purified from buffy coat was analyzed for MMc in offspring via autosomal indels. Females were also screened for MOMc using the Y-chromosome marker DYS14. Predictors were explored using Random Forest, followed by logistic regression to assess associations.

MMc was detected in 17.4% of youths (80/461) and MOMc in 20.5% of the female youths (54/263). For MMc, Random Forest ranked youth factors as stronger predictors than maternal factors, with asthma notably influential, though the odds ratio was 2.51 [95% CI 0.95–6.22]. For MOMc, maternal smoking and educational level were most important. The odds ratio for smoking before vs. during pregnancy was 2.35 [95% CI 1.05-5.25], while medium vs. primary education yielded an odds ratio of 0.84 [95% CI 0.49-1.43]. No other predictors or associations were identified.

Overall, youth factors, particularly asthma, predicted MMc more accurately than maternal factors. All the potential MOMc predictors tested were inconsistent and uncertain. Despite the breadth of the available data, we identified no robust predictors or associations for MMc or MOMc.

12:15 pm Lunch & Poster session & Industry exhibition

1:45 pm – 3:15 pm Microchimerism in Reproductive Health: From Infertility and Placental Dysfunction to Cardiovascular Risk

Microchimerism intersects with reproductive health and long-term maternal disease risk, linking placental biology, immune regulation, and vascular pathology. Evidence of microchimeric cells in endometrium and menstrual blood has prompted hypotheses that dysregulated maternal–fetal cell trafficking may contribute to infertility and pregnancy loss. Placental dysfunction—central to complications such as preeclampsia and fetal growth restriction—is associated with altered release of inflammatory mediators and increased fetal microchimerism in maternal circulation. Persistence of fetal-origin cells months to years postpartum, correlations with inflammatory proteomic signatures, and variation by fetal sex support a model in which microchimerism participates in sustained immune activation that may influence later cardiovascular risk. Advances in detection technologies, including digital PCR, high-dimensional proteomics, and highly sensitive HLA-specific flow cytometry capable of isolating rare viable maternal cells from fetal stem cell compartments, are refining mechanistic insight into trafficking and retention. This session covers microchimerism as a potential mediator connecting placental stress, reproductive outcomes, and women's long-term cardiometabolic health.

Microchimerism in infertility and pregnancy loss

Henriette Svarre Nielsen

University of Copenhagen, Denmark

This talk will focus on microchimerism in infertility and pregnancy loss. The talk will give an overview of the presence of microchimerism in the endometrium and menstrual blood. The research leading to the hypothesis that microchimerism could play a role in infertility and pregnancy loss will be summarized and studies exploring the hypothesis will be presented.

Women's risk of cardiovascular disease after pregnancy complications: does a dysfunctional placenta and fetal microchimerism play a role?

Anne Cathrine Staff

University of Oslo and Oslo University Hospital, Oslo, Norway

Several common obstetric complications are associated with increased risk of future maternal cardiovascular disease (CVD). The risk is especially high after severe and repeated pregnancy

complications. The mechanisms for the associations are not clear, but likely involve a synergy of preexisting risk factors (for the obstetric adverse outcome and cardiovascular disease) and risk factors mediated by the pregnancy complication. Common to many of these obstetric complications (e.g. preeclampsia and other hypertensive disorders of pregnancy, fetal growth restriction, preterm birth and gestational diabetes mellitus) is that the placenta is dysfunctional.
In preeclampsia, this placental dysfunction is closely linked to cellular syncytiotrophoblast stress, with an ensuing dysregulated release of proinflammatory and antiangiogenic proteins into maternal circulation. Preeclampsia is also associated with increased presence and quantity of long-lived fetal-origin cells in maternal circulation, termed fetal microchimerism. Our studies from human pregnancy have shown that the levels of fetal microchimeric cells in the mother correlates with placental dysfunction, as well as with severe maternal hypertension. Our human data also support a role for fetal-maternal histocompatibility in fetal microchimerism dynamics, both during pregnancy and postpartum.
The presentation will lay out the limited epidemiological background for linking fetal microchimerism to long-term maternal CVD. It will discuss how fetal microchimerism could potentially drive vascular inflammation in women and thereby contribute to premature maternal cardiovascular disease. Future and ongoing projects to improve the understanding of the role of fetal microchimerism in female cardiovascular health will be discussed.

Fetal Microchimeric Cell Retention Following Preeclampsia

Ina A. Stelzer

Ina A. Stelzer[1,2], Joshua Gillard[1], Maximilian Sabayev[1], Agnes Wieczorek[3], Dorien Feyaerts[1], Oshra Sedan[4], Petra C. Arck[3], Brice Gaudilliere[1], Mark Hlatky[4], Virginia D. Winn[5]**
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Introduction: Fetal microchimerism (FMc), the acquisition and long-term persistence of intact fetal cells in the mother, is increased in the blood of pregnancies with preeclampsia (PE) and may affect immune function and contribute to the higher risk of developing cardiovascular diseases after PE. The aim of this study was to measure levels of FMc in the postpartum period in participants with and without PE, and to examine the association of FMc with the plasma proteome.
Methods: To screen for FMc in maternal blood, we performed PCR in paired cord- and maternal-blood derived DNA to identify deletion-insertion polymorphisms (DIP) that

were present only in the fetal DNA (i.e., informative DIPs). After informed consent, we applied digital droplet PCR to maternal postpartum PBMC-derived DNA from 8 subjects with PE (incl. 2 twin pregnancies) and 11 with normotensive pregnancies, and screened for informative DIPs. FMc was quantified as genomic equivalents (fetal cells, gEq) per 1×106 maternal cells, and compared between groups using Mann-Whitney test. We analyzed the maternal postpartum plasma proteome for 7,000 proteins using an aptamer-based platform (SomaLogic), and performed gene set enrichment on the proteins most highly correlated with FMc levels.

Results: FMc cells were detectable in 79% (15/19) of samples up to three years after delivery. There was no significant difference in FMc levels between those with a history of PE compared to controls in this small sample, or according to gravida or maternal age. Interestingly, FMc levels were significantly lower in subjects who had been pregnant with a female fetus (n=11) than with a male fetus (n=10): mean 40 vs. 111 gEq/1×106 maternal cells, p=0.02. This difference appeared to be more pronounced in subjects with a history of PE, but there were relatively few such subjects. Higher FMc levels were significantly associated with higher levels of inflammatory plasma proteins, including toll-like-receptor pathway-associated proteins, and the enriched gene sets 'TLR7/8 Cascade', 'MyD88', 'TLR2 Cascade'.
Conclusion: In this pilot study, FMc cells were retained in maternal blood for months to years post-delivery, and FMc levels varied according to fetal sex. FMc levels were positively correlated with an inflammatory proteomic plasma environment. Maternal immune profiles of this and follow-up cohorts will determine whether PE-associated immune dysregulation persists postpartum alongside FMc levels.

Characterization of human amniotic fluid stem cells and their potential role in maternal microchimerism

Bernadette L. Bramreiter

Bernadette L. Bramreiter[1], Katja Sallinger[1], Emiel Slaats[1], Julia Schönberger[1], Katharina Schuch[1], Philipp Klaritsch[2], Herbert Fluhr[2], Thomas Kroneis[1]

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[2] Department of Obstetrics and Gynaecology, Medical University of Graz, Graz, Austria

Objective

Microchimerism (MC) is defined as the presence of a small population of genetically distinct cells within a host. During pregnancy, bidirectional cell trafficking across the feto-maternal interface results in maternal and fetal microchimerism. Since microchimeric cells represent cell types derived from all three germ layers, we assume cells with stem cell-like properties to be responsible for the establishment of lifelong MC. However, the cellular routes and mechanisms remain unclear. We hypothesize that maternal cells reach the fetus via ingestion of fetally-derived amniotic fluid (AF) and

subsequent transmigration into fetal tissues, potentially through the gastrointestinal tract, and that these cells represent a subpopulation of amniotic fluid stem cells (AFSCs).

Methods

Stem cells were isolated from AF using CD117-targeting microbeads and characterized using a 16-marker multicolor flow cytometry panel designed to identify AFSCs and distinguish them from other progenitor and contaminating populations. Markers included CD27, CD34, CD44, CD45, CD73, CD90, CD105, CD117, HLA-ABC, HLA-DR, SSEA-3, SSEA-4, Tra-1-60, Tra-1-81, and OCT3/4. Pluripotency was assessed by trilineage differentiation into early germ layer intermediates. Potential maternal microchimeric cells were identified in male pregnancies using XIST and RPS4Y1-specific padlock probes and/or X Y-FISH.

Results

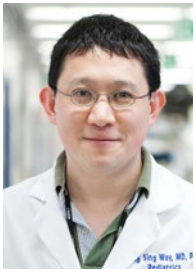
AFSCs expressed pluripotency-associated markers, including OCT3/4 and CD117, and lacked lineage-specific markers such as CD34 and CD45. Upon differentiation, AFSCs generated early germ layer intermediates expressing lineage specific morphology and markers such as FOXA2 (endoderm), CD144 (mesoderm), and Nestin (ectoderm). Maternal cells were detected in three of four AFSC samples.

Conclusion

The presence of maternal cells within the AFSC compartment supports a potential role for feto-maternal cell trafficking in the AF and suggests AFSCs as a potential source of maternal microchimeric cells.

4:00 pm -5:20 pm Microchimerism in Immune Tolerance and Neurodevelopment: Context-Dependent Persistence and Pathophysiology

Microchimerism shapes immune tolerance, neurodevelopment, and tissue-specific persistence across the lifespan. Enhanced tolerance to maternal antigens, observed epidemiologically and recapitulated in animal models, parallels the durable presence of maternal cells that promote regulatory T cell differentiation, resilience against fetal loss, and cross-generational immune calibration. New mouse models enabling microchimeric lineage-specific depletion providing a tool set to clarify how rare maternal and fetal cell populations interact and potentially compensate for genetic deficiencies. At the same time, altered microchimeric dynamics under pathological conditions reveal context-dependent consequences. Transfer of precursor molecules links fetal microchimerism to maternal cognitive decline, while prenatal influenza reshapes maternal cell composition and inflammatory phenotype in the fetal brain, with implications for neurodevelopment. Twin-derived microchimerism further points at tissue-selective niches suggesting organ-specific persistence influenced by genetic relatedness. This session discusses microchimerism as a dynamic system of immune tolerance, cellular competition, and context-sensitive vulnerability affecting both maternal and offspring health.

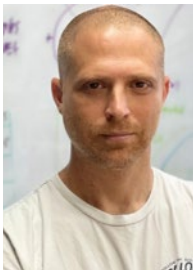


Immune tolerance to mothers and maternal microchimeric cells

Sing Sing Way

Cincinnati Children's Hospital, USA

Enhanced tolerance of individuals to their mothers compared with their fathers is consistently shown in human epidemiological studies. This parallels the vertical transfer and long-term persistence of maternal cells that establish microchimerism in offspring. My presentation will discuss shared maternal tolerance phenotypes and presence of maternal microchimeric cells in other mammalian species, including rodents, and ongoing use of animal models to investigate what microchimeric cells do and how their work. These include differentiation of CD4+ T cells into immune-suppressive regulatory T cells, cross generational resiliency against fetal wastage, differentiation distinctions primed by maternal compared with fetal microchimeric cells, and the remarkable interplay between these two types of exceptionally rare cells. Additional considerations will include whether phenotypically wildtype microchimeric cells can replace missing proteins in the context of autosomal recessive disorders and new adaptable platforms for experimental manipulation of microchimeric cells, including conditional depletion based on cell-lineage, to gain further insights on what microchimeric cells do and how their work.



Microchimerism during Down Syndrome pregnancies causes maternal cognitive decline

Eitan Okun

Head, the Paul Feder laboratory for Alzheimer's disease research
The Mina and Everard Goodman Faculty of Life Sciences
Bar Ilan University, Ramat-Gan, Israel

Down Syndrome (DS), caused by trisomy of chromosome 21, is the most common genetic form of intellectual disability, affecting 1 in 750-1000 live births worldwide. Individuals with DS face an increased risk of Alzheimer's disease (AD) due to the life-long over-expression of the amyloid precursor protein (APP) gene encoded on chromosome 21. Pregnancies involving a DS-affected fetus increase the mother's risk of late-onset AD (LOAD) almost five-fold compared to pregnancies with a fetus with other intellectual disabilities through mechanisms that remain unknown. We report that fetomaternal transfer of human APP (hAPP) impairs maternal cognitive function dose-dependently in a mechanism that involved microchimerism. Maternal vaccination targeting APP before pregnancy mitigated short-term memory loss. Our findings provide mechanistic insights into the increased LOAD risk in mothers of DS individuals and suggest possible preventive measures.

Influenza-induced alterations of maternal microchimeric cell composition and function in brains of fetal mice

Christopher Urbschat

Christopher Urbschat[1,2], Terry Ko[3], Szymon Rawiak[1,2], Hyojung Paik[3], Petra Arck[1,2,4]

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Fetal development is highly dependent on maternal mediators, e.g. maternal microchimeric cells (MMc), that can be transplacentaly transferred to the fetus during pregnancy. MMc seed to fetal organs during development and can reside in those niches for a lifetime where they e.g. steer fetal hematopoiesis and brain development. Maternal immune activation (MIA) due to infections can alter MMc composition and phenotype. To investigate the role of MIA-associated MMc during fetal brain development, we utilized a prenatal influenza A infection model.

Therefore, we infected WT C57BL/6 mice allogenicaly mated to Balb/c males with 102 P.F.U influenza during gestation. Nine days post infection we harvested fetal brains and performed high-dimensional flow cytometric analysis and single cell sequencing of MMc and fetal cells. On gestational day 18.5 flow cytometric analysis revealed that total numbers of MMc per 106 were significantly increased in fetuses of mothers who were infect with influenza. Although the composition of MMc was not affected and fetuses of both groups showed similar frequencies of cell types, MMc of prenatally infected fetuses exhibited higher expression levels of inflammatory cytokines like IL-6 and INFg.

Further, scRNA-seq. showed distinct cell cycle associated gene expression patterns between MMc and fetal cells. Whereas fetal brain cells showed increased expression of genes related S-phase, MMc exhibit stronger expression of G2-phase associated genes. Cell-cell communication analysis revealed a strong communication between fetal and maternal microglia cells, with the latter being of a more inflammatory phenotype in response to the prenatal influenza infection.

Taken together our data revealed that MIA leads to an increase of MMc being transferred to the fetus which are also of a more inflammatory phenotype and might have the potential to skew fetal brain development, resulting in long-lasting negative effects for offspring´s cognitive function.

Complement-producing maternal microchimeric cells override infection susceptibility in complement-deficient murine offspring

Giang Pham

Giang Pham, Raymond E. Diep, Lucien H. Turner, David B. Haslam, and Sing Sing Way
Division of Infectious Diseases, Center for Inflammation and Tolerance, Cincinnati Children's Hospital Medical Center, University of Cincinnati School of Medicine, Cincinnati, USA.

Long-term persistence of vertically transferred maternal cells occurs ubiquitously in mammalian offspring. The presence of these exceptionally rare maternal microchimeric cells (MMcs), with ensuing immunological tolerance to noninherited maternal antigen (NIMA), is associated with a variety of remarkable phenotypes including serological resistance to noninherited maternal HLA sensitization, improved long-term survival of NIMA-matched renal allografts, neonatal heart block, type 1 diabetes, and cross-generational reproductive fitness with expanded accumulation of NIMA-specific Tregs. Here, we considered whether MMcs may confer other physiological benefits beyond these immunological features linked with antigenicity. A provocative consideration is whether phenotypically wildtype (WT) MMcs can reduce disease severity in autosomal recessive disorders caused by defective or missing proteins. Given a shared susceptibility to infection caused by complement deficiency in humans and mice and enriched MMcs in the liver, where C3 and other complement components are produced, this hypothesis was investigated by evaluating complement levels and infection susceptibility of C3 NIMA mice (C3−/− mice born to C3+/− mothers) compared with genetically identical C3−/− mice born to complement-deficient mothers, along with C3+/− littermate controls. We found complement-producing MMcs were responsible for the above-background C3 levels and reduced infection susceptibility in complement-deficient offspring.
Beyond complement deficiency, our results suggesting clinical phenotypes associated with missing or defective proteins in autosomal recessive disorders can be altered by functionally WT MMcs open up fundamental new ways for explaining why individuals with the same gene defect in many autosomal recessive disorders, including cystic fibrosis and sickle cell anemia, have widely varied disease severity. In turn, these protective benefits associated with complement-producing MMcs highlight the importance of further investigating how these cells work, including their cellular identity and phenotype heterogeneity, since expanding their accumulation beyond natural microchimeric levels represents an innovative approach for therapeutically reducing the severity of common genetic disorders.

5:20 pm Closing Remarks

Poster Abstracts

Maternal vaccination promoted B cell microchimerism associates with high levels of protection against infection in offspring

William Horsnell

Alisha Chetty[1], Émer Hickey[2], Sirisha Naidoo[1], Matthew Darby[1], Peter Cook[1], Kai Toellner[3], Adam Cunningham[4], William Horsnell[1,2]
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Transfer of antibody-mediated immunity from mother to offspring protects infants from microbial exposure that would otherwise progress to life-threatening infection during early life. This maternal–offspring immune relationship has been successfully leveraged through maternal vaccination to close critical windows of susceptibility to severe childhood infections. Such maternal immune transfer is particularly important for protection against non-typhoidal Salmonella (NTS) in young infants.
We present pre-clinical data demonstrating that maternal vaccination against NTS provides offspring with protective cellular B-cell immunity. Offspring fostered on vaccinated mothers display enhanced B-cell maturation within the bone marrow, and bone marrow from these offspring secretes vaccine-specific antibodies capable of conferring protection against infection.
Importantly, this ability to secrete vaccine-specific antibody is associated with increased detection of maternally acquired B cells within the offspring bone marrow. Depletion of maternally acquired cells from the bone marrow abolishes its capacity to produce protective antibody, demonstrating a functional requirement for these cells in antibody-mediated protection.
Together, these findings show that offspring can acquire from their mothers the capacity to secrete antibodies directed against antigens experienced by the mother. This capacity is associated with elevated levels of B-cell maternal microchimerism, identifying MMc B cells as a previously unrecognised contributor to the protective antibody-mediated immunity transferred from vaccinated mothers to their offspring.

Dissecting the Role of Maternal Microchimerism in Offspring Immunity Following Candida albicans Exposure

Emer Hickey

Émer Hickey[1], Siphamandla Ngwenya[1],Orlando Ross[1], Phuong Tuyen Nguyen[1], Kazuki Sumiya[1], Peter Cook[1], Kai Toellner[3], Adam Cunningham[4], William Horsnell[1,2]
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Maternal immune experience can shape offspring immune development, yet the mechanisms underlying this intergenerational effect are incompletely understood. Here we address the role of maternal exposure to the ubiquitous fungal pathobiont Candida albicans in shaping offspring immunity.
Using a murine model, we show pre-conceptional exposure to Candida albicans induces lasting immune changes in both mothers and their offspring via nursing. Here, prior to infection, 10-day-old naïve offspring nursed by C. albicans–exposed dams already display enlarged spleens and altered intestinal length, indicating early-life immune changes. Following a subsequent systemic C. albicans challenge, these offspring display persistent splenomegaly, an impaired ability to control infection, evidenced by increased fungal burden in the kidney, and exacerbated clinical symptoms. Together, these surprising findings demonstrate that maternal C. albicans exposure can have negative consequences for offspring immunity that are evident before and during infection. Ongoing work aims to dissect the mechanisms underlying this intergenerational immune imprinting, with a particular focus on whether the postnatal transfer of maternal microchimeric cells contributes to the disrupted immune and infection outcomes observed in the offspring.

A novel approach to investigate breastmilk T cell antigen-specific responses

Blair Armistead

Yonghou Jiang[1], Jennifer E. Stolarczuk[2], Sharon Kung[2], John Houck[1], Victoria L. Campbell[3], David M. Koelle[3,4,5], Alisa Kachikis[2], Whitney E. Harrington[1,7,8], Blair Armistead[1,8]

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Breastmilk plays a pivotal role in infant health and development, providing both nutritional and immunological benefits. In addition to protective antibodies, breastmilk contains immune cells, including T cells, which have an unknown role in human

infant immunity. However, multiple studies in animals have shown that milk-derived T cells can traffic to peripheral organs of nursing offspring as a form of maternal microchimerism (MMc) and provide protection from infection. Further study is needed to understand the potential of breastmilk T cells to exert pathogen-specific effector functions at the maternal-infant interface. To-date, direct ex vivo functional analyses and T cell receptor sequencing of breastmilk T cells have been limited by low T cell frequency. To overcome this barrier, we optimized a method to expand breastmilk T cells in culture using cell sorting and mitotic stimulation. With this approach, we generated expanded breastmilk T cell (EBM T cell) cultures from n=4 lactating women, resulting in a ~1,000-fold increase in T cells from the original sample. Because each donor reported receipt of SARS-CoV-2 (SARS2) mRNA vaccination and/or history of SARS2 infection, we used SARS2 Spike as a model for detecting antigen-specific responses in EBM T cell cultures. In EBM T cell cultures from 2 of 2 donors tested, we identified SARS2 Spike-specific CD8+ T cells using an HLA-matched, Spike-loaded tetramer. To assess antigen-specific functional responses, we stimulated EBM T cells from one donor with a SARS2 Spike peptide pool, using autologous irradiated PBMC for antigen presenting function. Spike-stimulated EBM T cells contained CD4+ and CD8+ T cells expressing activation-induced markers, suggesting that their antigen-specific functionality was preserved. Together, our findings show that our novel approach is a valuable platform for investigating pathogen-specific responses in breastmilk T cells, with relevance to the study of breastmilk-derived MMc in infants.

Maternal microchimerism in biliary atresia: What is the role of maternal cells?

Toshihiro Muraji

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Background and Aim

Biliary atresia (BA) is a neonatal cholestatic liver disease and the leading indication for pediatric liver transplantation. Its etiopathogenesis is recognized as immune mediated, involving $CD4^+$ T cell activation toward Th17, suppression of Treg function as well as activation of $CD8^+$ T cells with macrophage derived TNF α, producing Th1 cytokines. Maternal microchimerism in BA was first reported by Suskind in 2004. Since then, we have explored the hypothesis that maternal engrafted lymphocytes may induce a graft versus host disease (GvHD)–like immune insult. The aim of this presentation is to introduce these findings to the microchimerism research community and to discuss potential approaches to elucidate the etiopathogenesis of BA.

Histopathological Findings

Marked lymph node enlargement at the porta hepatis is not constantly associated with reactive germinal centers, suggesting a limited role for infection (Bove, Pediatr

Dev Pathol, 2018). Portal tracts show dense lymphocytic infiltration, especially M2 macrophage and bile duct disruption, whereas in hepatic lobules, M1 macrophages infiltration with hepatocellular apoptosis and multinucleated giant cells. These features closely resemble of GvHD.

HLA Haplotype Frequency and BA Incidence

A significant HLA A compatibility between BA patients and their mothers has been reported. Across 10 populations (Japan, South Korea, Taiwan, the Philippines, Māori in New Zealand, the UK, France, Germany, Norway, and Sweden), the frequency of each population's most prevalent HLA haplotype correlated significantly with its incidence of BA ($p = 0.0126$) (Muraji, Human Immunology, 2018).

Placental Chronic Villitis in Dizygotic Twins with BA

A case of villitis of unknown etiology in the placenta of the BA affected twin in a discordant dizygotic pair suggests that maternofetal GvHD may contribute to the etiopathogenesis of BA (Kosaka, Pediatrics Int, 2022).

Characterization of microchimeric cells of maternal origin in umbilical cord blood by HLA-targeted spectral flow cytometry

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Background and Objective

Maternal microchimerism is the transmission of small quantities of maternal cells to the fetus. It has been found that maternal microchimeric cells can home to fetal bone marrow and lymph nodes where they exert influence on the fetal immune system. This can lead to the development of regulatory T cells that suppress fetal anti-maternal immunity and persist at least until early adulthood. However, due to the low frequency of maternal microchimeric cells in cord blood it can be challenging to identify and isolate enough cells for further functional analysis. The objective of this study was to detect and characterize maternal microchimeric cells in cord blood samples.

Methods

DNA was extracted from thirty cord blood and maternal PBMC dyad samples. These were HLA typed and screened by qPCR to target the unique maternal HLA alleles present in the cord blood. Eighteen samples had HLA class II targets, while a non-HLA polymorphism was used for 12 samples.

Results

Before subjecting the cord bloods to cell sorting, artificial mixtures of maternal PBMCs were spiked into a bulk background of fetal cord blood in ratios of 50%, 10%, 1%, and 0.1%. This was first performed to optimize antibody titrations and determine anti-HLA cross-reactivity among the different HLA combinations of the actual mater-

nal and fetal samples. Once determined, maternal and fetal samples were matched to a recombinant or hybridoma derived human monoclonal anti-HLA antibody unique to that sample. Using a 14-colour antibody panel for spectral flow cytometer sorting, maternal cells were sorted from of fetal cord blood by targeting the unique HLA antibody and the composition of sorted cells characterized.

Conclusion

Using both qPCR and flow cytometric cell sorting, this methodology enables reliable detection of maternal microchimeric cells in fetal cord blood allowing for further functional studies with these rare cells, which may play a role in influencing the fetal/neonatal immune system.

Do women after pregnancy loss benefit from knowing about fetal microchimerism?

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The frequency of pregnancy loss is unknown and probably may elude exact determination. Recent data from 2021 in Germany yielded 39,762 clinically recorded cases of pregnancy loss while the German Federal Statistical Office reported 795,492 live births and 3,420 still births. These data do not include spontaneous or medication-induced extramural pregnancy loss. However, international estimates claim 20% of all diagnosed pregnancies to face pregnancy loss. Based on the data of live births in the EU, pregnancy loss would affect his would concern 890,000 pregnancies (women) Pregnancy loss is associated with an extraordinary stress situation for women. The psychological disorders include depression, anxiety, post-traumatic stress which may become pathological in case the grief reaction is either severe or long-lasting. This causes not only personal healthcare issues and also stress in the patients' environment (e.g., families) but also economic concerns of unknown scale. Therapeutic procedures aiming at affective and behavioral responses (prenatal genetic testing, palliative care, birthing options, sedative, seeing/holding stillborn baby, memory creation, web-based and social support) have been suggested but none of them turned out to be the treatment of choice.

Fetal microchimerism (i.e., the presence of fetal traits in maternal tissue) has been detected as early as six weeks of gestation, is increased after pregnancy loss, and likely to occur also in case of pregnancy loss. Anecdotes from healthcare providers and pedagogues (midwifes, psychotherapist, teacher) report positive response of individuals experiencing pregnancy (and parental) loss when confronted with the fact that they might harbor fetal (and maternal) cells.

In this work we present the strategy and the current progress of our project on developing a tool based on microchimerism to support bereavement after pregnancy loss.

Delineating maternal invading cells: Presence and distribution of maternal invading cells across fetal villous tissue layers in samples diagnosed with villitis of unknown etiology

Katja Sallinger

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Microchimerism – the presence of non-self cells in an individuum of the same species – is believed to be an evolutionary conserved phenomenon that establishes due to cellular trafficking during pregnancy, presumably via a transplacental route (Ref). Attempts to investigate the transplacental trafficking includes screening for maternal cells entering male placental villi tissue sections or placental perfusion models using pre-labelled maternal blood cells. While both approaches come with a certain bias and shortcomings, invading maternal cells are also routinely detected in placental pathologies including villitis of unknown etiology (VUE). But since we currently lack biomarkers for pre-screening, pathologists need to work through the entire organ, relying on random sampling and subsequent histological analysis to detect VUE. An analysis to obtain the baseline of lesions in term placentae from physiological pregnancies revealed >75% of them to contain a placental lesion. We thus speculate placental lesions including maternal cells invading fetal villous tissues are highly frequent across all physiological pregnancies. Furthermore, we hypothesize cellular trafficking establishing microchimerism resembles the processes involved in VUE when maternal cells transmigrate into fetal villi.
In the presented work we aimed at tracing maternal cells invading fetal villous tissues in VUE gaining access to the fetal circulation and, thus, may play a role in establishing microchimerism. Additionally, we sought to develop a workflow allowing to overcome the drawbacks in pre-analytical screening using a 3D/2D multimodal approach including lightsheet microscopy, spatial transcriptomics and proteomics.

Fetal microchimerism in female stroke patients

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Cardiovascular disease including ischemic stroke (CVD), is a leading cause of mor-

bidity and mortality in women. Epidemiological studies link preeclampsia and other conditions with placental dysfunction to increased later CVD risk, but underlying mechanisms are not fully understood. During pregnancy, fetal cells transfer to the mother and a subset persists for decades post-partum, termed fetal microchimerism (MC). Increased quantity of fetal cells in maternal circulation has been observed during preeclampsia. We hypothesized that the long-term presence of these cells promotes low-grade vascular inflammation and atherosclerosis, increasing stroke risk. In this study, we examined occurrence of male MC, measured as Y-chromosomal DNA, in two female stroke cohorts. First, digital droplet PCR (ddPCR) targeting SRY- and TSPY genes was employed to detect circulating male DNA in elderly women with ischemic stroke (n=80) and healthy controls (n=38); in a subset (n=33) longitudinal blood samples up to 8 months post-stroke were analyzed. In a second cohort of women undergoing carotid or inguinal endarterectomy, tissue-bound male MC was assessed by ddPCR on snap-frozen specimens (n=20) and fluorescence in situ hybridization with dual Y-chromosome probes on formalin-fixed tissue (n=20). Paired blood samples for circulating male MC analysis were available for a subset (n=12+12). Preliminary data show male DNA in 16,25% (13/80) of acute stroke patients and in 15,8% (6/38) of healthy controls.

Generating a spatial map of microchimerism in whole mice

Katharina Schuch

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Background and aims
Microchimerism is the presence of a small number of cells within an individuum that originate from another individuum – commonly due to bidirectional exchange of maternal and fetal cells during pregnancy. Microchimerism has been reported in numerous tissues individually, but its distribution in the whole organism remains to be elucidated. We aim to map microchimeric cells on sections of whole mouse pups and feed a 3D mouse Microchiome Atlas.
Methods
Serial sections of whole fresh frozen wildtype pups with tdTomato+/- microchimeric cells were briefly fixed, DAPI stained and assessed for tdTomato fluorescence. Thereafter, padlock probe-based in situ hybridization (ISH) was performed on the same slides to validate tdTomato based on its transcripts. Images were analyzed using QuPath.
Results
Fully intact sagittal sections of whole mouse pups were generated using adhesive films and contained up to 1.2 million cells per section. Preliminary results indicate

on average less than 10 microchimeric cells per 100 000 cells for both tdTomato-fluorescent cells and cells with ISH signals, respectively, although only a fraction of these are positive in both assays.

Conclusion

We present a workflow for assessing the spatial distribution of microchimerism and highlight the need to cross-validate potential cells-of-interest using multiple assays.

Optimized pre-analytics for spatial detection of microchimeric cells

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Background and aims

Microchimerism (MC) describes the long-term persistence of genetically foreign cells within an individual that are commonly acquired during pregnancy. The quantity of microchimeric cells has been reported to be in the range of approximately 1 in 10^4 down to 1 in 10^9 cells, depending on detection method and tissue. The spatial distribution of MC in whole individuals has not been investigated before. Our aim was to optimize pre-analytical processes to allow the spatial detection and validation of these rare cells on sections of whole mice.

Methods

We used transgenic mice with expression of eGFP or tdTomato under the CAG promotor. RNA sensitivity to 4% formaldehyde fixation was assessed on serial cryosections for fixation times ranging from 15 min up to 48 h as is common practice for histological staining. The effect of storage on RNA detection was assessed with sections of the two halves of a pair of kidneys, stored at -20°C or -80°C respectively for 2 weeks up to 1 year. RNA was detected using padlock probe-based in situ hybridization (ISH), and signal quantity of multiple highly expressed transcripts was compared over time.

Results

We show that the transgenes were expressed in all tissues, but expression varied greatly depending on the individual. We detected the highest number of RNA signals with a maximum of 1 h fixation, standard 24-48 h of fixation resulted in over 90% decrease in signals. Storage durations of up to 13-26 weeks resulted in comparable signal yield at both -20 and -80°C, longer storage resulted in reduced RNA signals.

Conclusion

Even the CAG promoter, widely regarded as one of the strongest promoters for ubiquitous transgene expression, does not ensure uniform expression across all cells, and results from respective mouse models should be interpreted with appropriate caution. Spatial detection of RNA can be improved significantly by keeping fixation time short and processing fresh frozen samples within several months.

In Situ Padlock Probes Targeting mRNA Variants for the spatial detection of microchimerism and maternal-fetal interactions

Emiel Slaats

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Maternal microchimerism (mMC) is the long-term persistence of a small population of maternal derived cells in the offspring after birth. To better understand the functional consequences of mMC in humans, as well as the mechanisms driving their transplacental migration during pregnancy, it is crucial to detect and characterize these cells within the spatial context of the host tissues. Historically, the spatial detection of mMC in the offspring has relied on XY FISH, which requires a sex mismatch between mother and child and thus inherently introducing a sex bias.
To overcome this issue, we developed a novel approach for microchimerism detection in tissues using spatial transcriptomics-based techniques. We designed a panel of padlock probes targeting twelve common (frequency within the population above 20%) biallelic single nucleotide polymorphisms (SNPs) expressed in the mRNA as well as HLA-A and HLA-C allele-specific transcripts. By combinatorial use of assays targeting allele mismatches between mother and child, we show how an individual specific signal can be generated within single cells at their native locations in the tissue. When combined with cell-type marker detection, this approach enables in-depth exploration of maternal microchimeric cells within their spatial and cellular tissue context in a sex unbiased manner.

On the origin of mast cells in cutaneous graft-versus-host disease

Thomas Kroneis/ A.R. Teufelberger

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Allogeneic hematopoietic stem cell transplantation (allo-HCT) is a curative treatment option for various hematologic diseases. It induces artificial chimerism in the recipient (host) through engraftment of donor-derived hematopoietic stem cells and resulting immune cells. Cutaneous graft-versus-host disease (cGvHD) is a potential complication after allo-HCT, in which the engrafted immune cells attack the host's skin tissue. cGvHD can present acutely (less than 100 days after allo-HCT) or chronically

(more than 100 days after allo-HCT). Mouse models of acute and chronic cGvHD have shown that mast cells play a protective, disease-ameliorating role in acute cGvHD, but increase disease severity in chronic cGvHD. It is not yet known whether skin mast cells in cGvHD patients originate from the host or the graft, there is a difference of origin between acute and chronic cGvHD, or their protective role is linked with their origin.
To investigate the origin of mast cells, we used a combination of immunofluorescence staining for mast cells and XY-chromosomal fluorescence in situ hybridization on lesional skin samples from patients who received sex-mismatched allo-HCT and developed acute or chronic cGvHD.
We found that in acute cGvHD patients, the majority of skin mast cells originated from the host, while higher frequencies of engrafted mast cells were found in chronic cGvHD patients' samples.
Our study demonstrates an association suggesting that the dual function of mast cells in acute versus chronic cGvHD may be related to their origin: protective when host-derived and combative when graft-derived. In the future, this association will need to be confirmed in functional studies in human cGvHD.

Microchimerism in biliary atresia: What is the role of maternal cells?

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Background

Biliary atresia (BA) is a devastating pediatric liver disease characterized by progressive destruction of the bile ducts. The disease mechanism involves increased CD8+/CD4+T cells with upregulation of Th17 and downregulation of Treg cell under IL-6. Postoperative cholangitis is a major risk factor for poor outcomes. Microchimerism has been reported in BA, yet its clinical significance remains unclear. Maternal chimeric (Mc) DNA was detected in buffy coat (10–328 gEq per 10^6 host cells) of 7/12

BA patients and was significantly associated with the presence of portal hypertension (Masuya R, Kanaan SB, et al, Front Pediatr. 2022). This study aimed to examine whether Mc DNA in buffy coat correlate with serum C reactive protein (CRP), driven by IL-6.

Methods

Eight BA patients treated at our institution were included. Mc DNA in buffy coat was quantified using the same qPCR assay described by Masuya et al. We compared the serum CRP levels between the two groups, Mc DNA present (Mc group) and absent (non-Mc group) in buffy coat.

Results

Five patients were classified into the Mc group and three into the non-Mc group. Median CRP levels showed no significant difference between the two groups (0.04 mg/dl vs. 0.09 mg/dl, p=0.76). However, two patients demonstrated notable temporal changes. In Patient 1, the initial CRP level was 0.08 mg/dl with undetectable Mc DNA in the buffy coat, but when the patient developed cholangitis and the CRP level increased to 1.47 mg/dL, Mc DNA also increased to 9.8 gEq per 10^6 host cells. In Patient 2, Mc DNA was undetectable when CRP was <0.02 mg/dL, but at follow up, CRP increased to 0.04 mg/dL and Mc DNA increased to 847 gEq per 10^6 host cells.

Conclusion

This study showed no significant correlation between Mc DNA levels in buffy coat and CRP levels in BA. Nevertheless, two patients exhibited synchronous increases in Mc DNA and CRP. This suggests that immune cells such as maternal lymphocytes might lead the inflammation in BA.

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Art & Science

Tom Lohner on LinkedIn: "The painting "Microchimerism" is a cosmic event on canvas, that dissolves the boundaries between science, myth, and human connection. Serving as the lead visual for the International Symposium at the Medical University of Graz in May 2026, it invites the viewer to explore the invisible, cellular threads that bind us inextricably across generations.

The canvas is dominated by a central mother figure radiating an aura of unshakable strength. Floating before her is an infant, a delicate being of light born from a protective halo of maternal energy. The mother shields her child with oversized, levitating hands that stand like two mighty rocks against the tide. Integrated into these painted hands are carefully selected collage elements: superhero figures cut from original 1975 comic books. This choice is an homage to "ancient superpowers“, extraordinary abilities such as telepathy and intuition that were long dismissed as mere fantasy, but which science is now beginning to identify as the biological reality of microchimerism. They serve as proof that the power of creation (and love in Tom’s opinion) are among the strongest forces in the universe. To her right, her second daughter floats upside down, defying the laws of gravity and linear time. This compositional stroke serves as a reminder of life's unpredictability and the countless dimensions in which we exist and remain connected. The figures are linked by DNA double-helixes and energetic currents, bridging the gap between the mystical atmosphere as well as the scientific research made today ;-). ...

"Microchimerism" is an invitation to reflect on the nature of deep human bonding, the power of love, and the fascinating discoveries science makes every day. It is an homage to the invisible threads that connect us and the infinite possibilities slumbering within."

More about Tom Lohner
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Fun fact Tom's brother Andy, whom he has been working with creatively since young age, created the graphical layout around the artwork.

More about Andy G. Lohner
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Photo credit next page
Painting:Tom Lohner , Microchimerism/Acrylic on canvas, 110 x 80 cm
Grafik by Andy Lohner

INTERNATIONAL CONFERENCE & PUBLIC SYMPOSIUM ON MICROCHIMERISM

AULA MED UNI GRAZ

PUBLIC SYMPOSIUM
MAY 26TH, 2026

INTERNATIONAL CONFERENCE
MAY 27-28, 2026