Bone Marrow Adipose Tissue:
From Molecular Biology to Pathology

Bone Marrow Adiposity Society
3RD SUMMER SCHOOL, 2025

24-26 September, virtual event

Final program and abstract book

www.bma-society.org

@BMA_Society

Organizing Committee:
Next Generation BMAS

Drenka Trivanovic
Tânia Amorim
Adriana Roque
Souad Daamouch
Tiange Feng
Young Eun Park
Izabela Podgorski
Maxime Bedez

BMAS
SUMMER SCHOOL 2025
A VIRTUAL MEETING

WELCOME MESSAGE

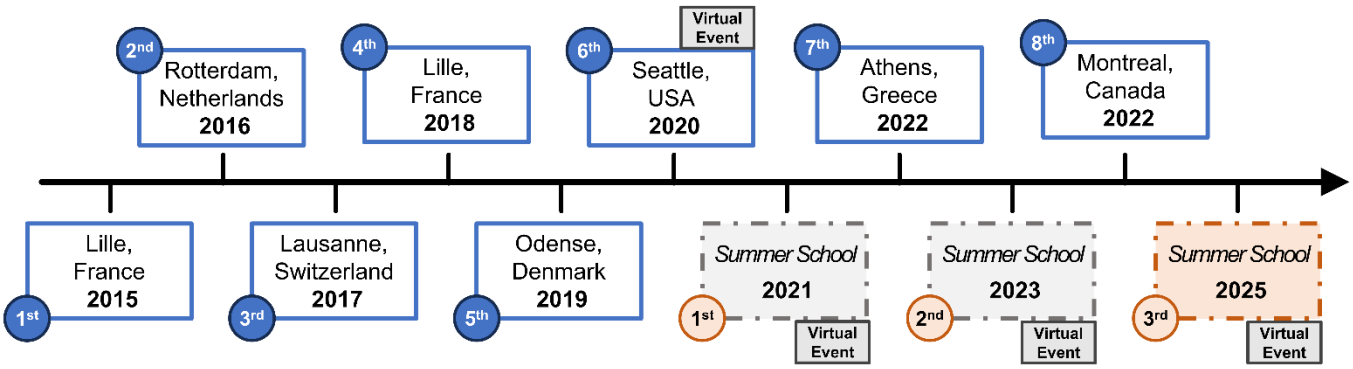
Welcome to the 3rd International Bone Marrow Adiposity Society (BMAS) Summer School 2025!

We are delighted to have you join us for this virtual event, taking place from September 24 to 26, 2025. Since its founding in 2017, BMAS has been dedicated to advancing our understanding of the role of **bone marrow adipocytes in bone health, hematology, cancer, systemic metabolism, and related fields**. BMAS uniquely fosters collaboration between biomedical scientists and clinicians from diverse backgrounds through biannual meetings and focused working groups. Building on the success of the second BMAS Summer School in 2021 and 2023, the **Next Generation BMAS working group** acts to actively engage and support emerging **researchers and clinicians** in the field.

Inspired by the achievements of previous summer schools, we are excited to continue this tradition of **learning, collaboration, and fostering knowledge in the latest technologies applied in BMAT research**. with the 2025 Summer School. We look forward to your participation and hope you find this experience both enriching and rewarding.

BMAS history

THE INTERNATIONAL BONE MARROW ADIPOSITY SOCIETY
AT THE CROSSROADS OF FAT, BLOOD AND BONE



BMAS NEXT GENERATION

Sincerely,

BMAS Summer School 2025 Organizing Committee

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ORGANIZING COMMITTEE

BMAS Summer School 2025 Organizing Committee	
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Tânia Amorim, PhD	*University of Pittsburgh, United States*
Adriana Roque, MD	*Hospital and University Centre of Coimbra, Portugal*
Souad Daamouch, PhD	*University of Southern Denmark, Odense, Denmark*
Tiange Feng, PhD	*MaineHealth Institute for Research, United States*
Young Eun Park, PhD	*University of Oxford, United Kingdom*
Izabela Podgorski, PhD	*Wayne State University, Detroit, United States*
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Young Eun Park, *United Kingdom*


Souad Daamouch, *Denmark*


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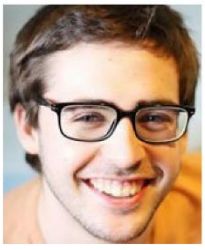
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Izabela Podgorski, *USA*

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Michaela Tencerová	*Czech Academy of Sciences, Prague*
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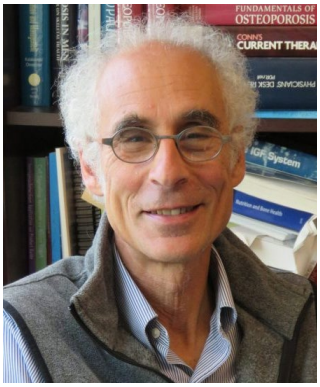
Ling Qin	*University of Pennsylvania, United States*
Kaisa Ivaska	*University of Turku, Finland*
Camille Attané	*Institute of Pharmacology and Structural Biology in Toulouse, France*
Jason Horton	*State University of New York Upstate Medical University, United States*
Abhishek Chandra	*Mayo Clinic, United States*

INVITED SPEAKERS

Bram C J van der Eerden, PhD, BMAS president	*University Medical Center Rotterdam, The Netherlands*
Ralf H. Adams, PhD	*Max Planck Institute for Molecular Biomedicine, University of Münster, Germany*
Clifford J Rosen, MD, PhD	*Maine Medical Center's Research Institute, United States*
César Nombela Arrieta, PhD	*University Hospital and University of Zurich, Zurich, Switzerland*
Ling Qin, MD, PhD	*Perelman School of Medicine, University of Pennsylvania, United States*
Ormond MacDougald, PhD	*University of Michigan, United States*
Ryan C. Chai, PhD	*Garvan Institute of Medical Research, Australia*
Glen Niebur, PhD	*University of Notre Dame, United States*
Claire Edwards, PhD	*University of Oxford, United Kingdom*
Shingo Kajimura, PhD	*Harvard Medical School, United States*
Barna Gal, MD, PhD	*CompagOs AG, Switzerland*
Davide S. K. Komla Ebri, PhD	*UCB Pharma, United Kingdom*
William Cawthorn, PhD	*University of Edinburgh, United Kingdom*
Tim J Schulz, PhD	*German Institute of Human Nutrition Potsdam-Rehbrücke, Germany*
Michaela Reagan, PhD	*MaineHealth Institute for Research, United States*
Dimitrios Karampinos, PhD	*Technical University of Munich, Munich, Germany*
Erica Scheller, PhD	*Washington University School of Medicine, United States*
Ziru Li, PhD	*Maine Medical Center's Research Institute, United States*

Dr. Bram van der Eerden was trained as a cell biologist and obtained his PhD in 2002 at the University of Leiden, the Netherlands. From then onwards he worked at the Erasmus University Medical Center in Rotterdam, the Netherlands, where he is currently group leader of the Calcium and Bone Metabolism research group and coordinating head of the laboratories within the department of Internal Medicine. His research focus areas are bone tissue (re)generation and calcium/phosphate homeostasis. He has been a board member of the Dutch Society for Calcium and Bone Metabolism and within ECTS, he has held positions in scientific program committees, the E-learning and the corporate engagement and sponsoring action groups. Having been the treasurer since its foundation, he is currently the president of the international Bone Marrow Adiposity Society (BMAS) and council member of the IFMRS. He has published peer-reviewed >140 papers in the field.

Dr. Ralf Adams started his independent career at the Cancer Research UK London Research Institute in 2000. Later, he became director at the Max Planck Institute for Molecular Biomedicine and professor at the University of Münster, Germany. The main research interests of Ralf Adams are vascular biology, the growth and organ-specific specialization of blood vessels, and the crosstalk with cells in the surrounding tissue. A key discovery by his team is the identification of specialized vessel subpopulations in bone with critical functional roles in skeletal development, bone homeostasis, age-related bone loss, and osteoporosis. His research uses advanced mouse genetics and confocal/two-photon microscopy together with a range of cell biology approaches. Ralf Adams is an elected member of the European Molecular Biology Organisation. He has received the Otto Hahn Medal of the Max Planck Society, the Werner Risau Memorial Award, the Malpighi Award of the European Society for Microcirculation, and the Feldberg Prize.



Dr. Clifford J Rosen is a basic and translational bone biologist interested skeletal stem cell fate. Dr. Rosen is the Principal Investigator for the Northern New England Clinical and Translational Research Network (U54) that stresses clinical translation of basic investigations. He is the past president of the American Society of Bone and Mineral Research and recently finished a four-year term as council member of the National Advisory Committee on Aging (NIA). He previously served a four-year term on the NIAMS council and as a Board member of the Endocrine Society. He has been an Associate Editor at New England Journal of Medicine for almost 10 years, and previously a Senior Editor at eLife. Dr. Rosen has published more than 600 peer-reviewed publications in Journals such as *Nature*, *Nature Medicine*, *Cell*, *Cell Metabolism*, *PNAS*, *New England Journal*, *Journal of Clinical Investigation* and *Lancet*. He currently serves as chair of the Steering Committee for CALERIE, an R33 from NIA evaluating the impact of calorie restriction in adults.

Prof. César Nombela-Arrieta studied Pharmacy at the Universidad Complutense of Madrid. He undertook a PhD thesis in the National Center for Biotechnology (Spain), and the Theodor Kocher Institute (Bern, Switzerland) under the supervision of Dr. Jens Stein. His work focused on uncovering the intracellular pathways that drive lymphocyte recirculation throughout secondary lymphoid organs. As a postdoctoral fellow in the laboratory of Dr. Leslie Silberstein, at the Division of Transfusion Medicine in Children's Hospital Boston, his interests shifted towards the study of hematopoietic stem and progenitor cell (HSPC) niches and their spatial distribution in hematopoietic tissues.

In 2015 he became Assistant Professor at the Department of Medical Oncology and Hematology of the University of Zurich, where he was promoted to Associate Professor in 2022. His work currently lies at the interface of immunology, experimental hematology and cancer. His group investigates how hematopoiesis is orchestrated through the functional crosstalk of stromal, immune, and progenitor cells, focusing on diverse aspects of these complex cellular communication systems. The aim is to understand how perturbations of this cellular dialogue by inflammatory conditions, infections and ageing result in deregulated hematopoiesis and in the development of hematological disease.

Dr. Nombela-Arrieta has received funding of the European Research Council, the Swiss National Science Foundation and the Swiss Cancer league among other funding institutions. His work has been recognized with the Ellerman Prize of the Swiss Society of Hematology. Dr. Nombela-Arrieta is editor of the journal *Hemasphere*, member of the Fellowships and Grants Committee of the European Hematology Association and the Chair of the PhD program on Cancer Biology at the University of Zurich.

Prof. Ling Qin is a Professor of Orthopaedic Surgery and Co-Director of PCMD Histology Core at the University of Pennsylvania. She is an Associate Editor of Journal of Bone and Mineral Research. She became ASBMR (American Society for Bone and Mineral Research) fellow in 2018 and Fellow of International Orthopaedic Research (FIOR) in 2019. She has served as a standing member of NIH SBDD (Skeletal Biology Development & Disease) study section. To date, she has published more than 100 peer-reviewed research articles, reviewers, and book chapters in journals such as *Cell, Sci Transl Med, J Clin Invest, PNAS, Elife, JBMR, Arthritis Rheumatol, Nature* etc. Her research program is well funded by NIH. The overall goal of her research is to combine studies on fundamental mechanisms of skeletal cell function with translational medicine approaches to treat skeletal diseases. Her group uses a combination of molecular, biochemical, imaging techniques, and animal models to investigate the molecular mechanisms by which hormones and growth factors regulate bone metabolism and skeletal development under normal and pathological conditions. In the past decade, she has made some groundbreaking discoveries that greatly advance our understanding of molecular regulation of bone and cartilage metabolism, stem cell biology, and skeletal development, and provided promising therapeutic tools for skeletal disorders, such as osteoporosis and osteoarthritis.

Prof. Ormond MacDougald is the John A. Faulkner Collegiate Professor of Physiology at the University of Michigan, where his research has long focused on adipose tissue biology, including the unique role of bone marrow adipose tissue. In addition to his scientific contributions, Dr. MacDougald has extensive experience securing research funding from major agencies such as the NIH, and he has mentored numerous trainees and junior faculty on developing successful grant proposals. Today he will share his insights on navigating the grant writing process and strategies for building strong, fundable applications.

Dr. Ryan Chai is a Senior Research Officer at the Garvan Institute of Medical Research in Sydney, Australia. His research focuses on uncovering the genetic and cellular factors that regulate bone homeostasis and contribute to disease. By integrating single-cell technologies with population genetics and in vivo models, he has mapped the cellular and transcriptomic landscape of the bone microenvironment in mice and humans, revealing mechanisms of bone mass regulation and the role of bone as a niche for cancer cells. He has published more than 20 papers in leading journals including *Cell*, *Cancer Cell*, *Nature Genetics*, *Nature Communications*, and *Blood*.

Prof. Glen Niebur is Professor and Chair of the Department of Aerospace and Mechanical Engineering at the University of Notre Dame. He has also served as Director of Graduate Studies for the department of Aerospace and Mechanical Engineering, and Director of the Notre Dame Bioengineering Graduate Program.

Professor Niebur received his bachelor's degree in Mechanical Engineering in 1986 from the University of Minnesota. He has worked as a software developer for Control Data Corporation, and as a biomechanics research engineer at the Mayo Clinic Orthopedic Biomechanics Laboratory from 1986 to 1996. In 2000, he completed the Ph.D. degree at the University of California, Berkeley where his research focused on microstructural and multi-scale modeling of trabecular bone failure properties. In 2009 – 2010, he was a Walton Visiting Scholar in Galway Ireland, where he studied stem cell and bone biology.

Research in the Notre Dame tissue mechanics laboratory is focused on the effects of mechanical loading on trabecular bone and bone marrow mechanobiology, with the aim of understanding how mechanical cues, both normal and abnormal, can be used to drive different types of cells toward behaviors that affect health. Current projects include mechanical cues in breast cancer metastasis to bone, osteocyte gene expression in response to mechanical loads, and mechanobiology of implant osseointegration.

He has over 100 peer reviewed publications and is a Fellow of ASME and of the Orthopaedic Research Society.

Prof. Claire Edwards is a Professor of Bone Oncology at the University of Oxford with a joint appointment between the Nuffield Dept. of Surgical Sciences (NDS) and the Nuffield Dept. of Orthopaedics, Rheumatology and Musculoskeletal Sciences (NDORMS). She is also a Fellow of St. Edmund Hall. Claire is the recipient of multiple awards and fellowships, including recently the Mike Horton Award from the European Calcified Tissue Society. Claire is the past president of the Cancer and Bone Society. Claire runs the Bone Oncology Research Group based at the Botnar Research Centre and became the Director of Graduate Studies for NDS in 2022. Her research is centred upon tumour-bone interactions, with a focus on adiposity and metabolism in prostate cancer bone metastasis and multiple myeloma.

Dr. Shingo Kajimura's laboratory studies the molecular basis of bioenergetics in health and disease, i.e., how we coordinate metabolic demands and control energy homeostasis, and how the processes go wrong in metabolic diseases, cancer, and aging. The overarching goal is to generate a blueprint for rewiring the bioenergetic circuitry by defined factors, thereby restoring metabolic health. A particular focus in Dr. Kajimura's lab is adipose tissues because it dynamically remodels their cellular composition, metabolism, and function in response to external and internal cues, including nutrition, hormonal cues, and temperature. Such metabolic adaptation in adipose tissues, involving differentiation, mitochondrial biogenesis/clearance, lipolysis/lipogenesis, and thermogenesis, plays a central role in the regulation of whole-body energy homeostasis. In turn, a defect in the processes leads to obesity, insulin resistance, dyslipidemia, cardiovascular diseases, and certain types of cancer.

Dr. Barna Gal is a an entrepreneur turned physician and co-founder of Compagos, an ETH Zurich spin-off developing bone organoids and diagnostic solutions for bone disorders, where he led commercial activities. He also led the launch phase of Nimvira, a biotech company developing peptides and small molecules for osteoporosis. He was a product manager at Icotec, a medtech company developing implants for spine tumor patients, where he was responsible for strategic marketing and portfolio management.

Barna has a MAS in biomedical entrepreneurship and translational medicine and is an orthopedic surgeon. At the moment, he is working on his next company, which is about a small molecule therapy to treat progressive forms of multiple sclerosis.

Dr. Davide Komla-Ebri is a Senior Research Scientist at UCB Pharma, Slough, United Kingdom, with over a decade of experience in skeletal biology, molecular genetics, and translational research. Since joining UCB in 2022, Davide has advanced from Research Scientist to Senior Scientist, contributing to innovative projects in joint and bone biology with microCT imaging, mineralisation assays, and transcriptomic analyses.

Before UCB, Davide was a Research Associate at Imperial College London, where he worked on high-impact studies in bone development and disease. His academic journey includes a PhD from Paris University for its research on therapeutic approaches for Achondroplasia at the Imagine Institute of Genetic Diseases in Paris. He also holds degrees in Genetics from Université Paris Diderot and Molecular Cell Biology from Università degli Studi di Milano.

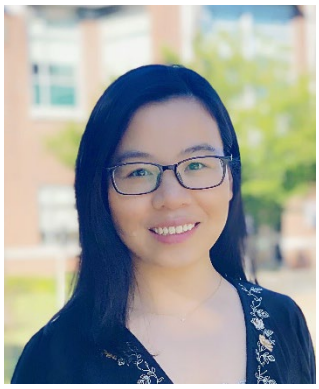
Prof. William Cawthorn is a Reader (Associate Professor) in the Institute for Neuroscience and Cardiovascular Research, University of Edinburgh. His research addresses adipose tissue formation and function, as well as sex differences in health and disease. A key focus is on establishing the fundamental biology and clinical implications of bone marrow adipose tissue. To do so, his studies combine preclinical animal models, human clinical studies and data science approaches using the UK Biobank. Methods include in-depth metabolic phenotyping, immunological analyses, the development of novel biomedical imaging techniques, and artificial intelligence to open new avenues for population-level studies, including molecular epidemiology methods. Together, his research addresses the interplay between metabolism, immunological function and skeletal health, both in the context of fundamental biology and chronic diseases. Dr Cawthorn is also very active in open science, research culture and integrity. He is the University of Edinburgh's Open Science Ambassador for the League of European Research Institutions (LERU); a co-founder of the Edinburgh Open Research Initiative (https://edopenresearch.com); and the University of Edinburgh Institutional co-Lead for the UK Reproducibility Network.

Dr. Tim J. Schulz is head of the Department of Adipocyte Development and Nutrition at the German Institute of Human Nutrition in Potsdam-Rehbrücke, Germany. He received his doctorate in biochemistry and nutritional sciences from the Friedrich-Schiller-University of Jena, Germany and subsequently completed his post-doctoral training on BMPs and brown adipose tissue in the laboratory of Dr. Yu-Hua Tseng at Joslin Diabetes Center and Harvard Medical School, in Boston, USA. His present research interests include the role of aging in regenerative decline and adipocyte formation within the tissues of the musculoskeletal system.

Dr. Michaela Reagan is a Faculty Scientist II at the MaineHealth Institute for Research and an Associate Professor at Tufts University School of Medicine. She has been leading the Reagan Lab since 2015 in interrogating new targets for the blood cancer multiple myeloma and related diseases. She received her B.S. in Engineering from Harvey Mudd College (2006) and Ph.D. from Tufts University in Biomedical Engineering in the field of breast cancer bone metastasis (2011). She did her post-doctoral research at the Dana-Farber Cancer Institute in medical oncology (2011-2015). She is currently a member of the Finance Committee of the American Society of Bone and Mineral Research, and is a past chair of the Women's Committee for this society. She has carved out a unique niche in the myeloma field by studying the interaction of bone marrow adipocytes and tumor cells, which led to her research into Fatty Acid Binding Proteins. She is supported by the NIH and the Leukemia and Lymphoma Society (LLS).

Prof. Dimitrios Karampinos studied engineering at the National Technical University of Athens, Greece. In 2008, he earned his PhD from the University of Illinois at Urbana-Champaign and he was then a Postdoctoral Scholar in the Department of Radiology and Biomedical Imaging at the University of California, San Francisco. In 2012, he joined the Department of Diagnostic and Interventional Radiology at the Technical University of Munich (TUM) as a Junior Group Leader and he obtained his Habilitation in Medical Physics in 2016. He started in 2019 as a Tenure-Track Assistant Professor for Experimental Magnetic Resonance Imaging at the TUM School of Medicine and Health. He was granted tenure at TUM in June 2024. He is a recipient of a European Research Council (ERC) Starting Grant (2016) and an ERC Proof-of-Concept Grant (2020). He joined EPFL as an Associate Professor in March 2025.

Prof. Erica Scheller is an Associate Professor of Medicine, Cell Biology and Physiology, Developmental Biology, and Biomedical Engineering at Washington University in Saint Louis. She is also the current Executive Director of the WashU Center of Regenerative Medicine and Past President of the International Society of Bone Morphometry. The Scheller Laboratory synthesizes concepts from cell biology, physiology, and bioengineering to study the relationships between the nervous system, the adipose tissues, and the skeleton. The Scheller Lab has a longstanding interest in bone marrow adipose tissue, and has contributed to foundational work pertaining to the regulation, function, and origins of this unique depot.



Prof. Ziru Li is a Faculty Scientist at the MaineHealth Institute for Research (MHIR), Assistant Professor at Tufts University School of Medicine, and Graduate Adjunct Faculty at the University of Maine. She earned her Ph.D. in Physiology from Peking University and completed postdoctoral training at the University of Michigan under Drs. Weizhen Zhang and Ormond MacDougald. Dr. Li's research focuses on energy metabolism and its impact on skeletal and bone marrow niche homeostasis, with particular interest in the gut–bone axis, bone marrow adiposity, and bone loss. She has received funding from the NIH and the American Diabetes Association and has authored over 50 peer-reviewed publications. Dr. Li serves on the editorial board of *The Journal of Biological Chemistry* and holds active memberships in ASBMR, the Endocrine Society, and the Bone Marrow Adiposity Society. She is committed to mentoring the next generation of physician-scientists and biomedical researchers.

SCIENTIFIC PROGRAM

Time listed in CET – European time

Wednesday, 24th September 2025 *Meet Bone Marrow Adipocyte*		
14:00 – 14:15	**10 years from the first BMAS meeting**	
	Speaker: **Bram J. van der Eerden**, *University Medical Center Rotterdam, The Netherlands* *Moderator: Drenka Trivanović, Organizing Committee Chair*	
14:15 – 14:55	*Keynote Lecture*	***Specialization of bone marrow cells and microenvironment***
	Speaker: **Ralf H. Adams**, *Max Planck Institute for Molecular Biomedicine, University of Münster, Germany* *Moderators: Kaisa Ivaska & Tânia Amorim*	
15:00 – 15:40	*Invited Lecture*	***Bone marrow adiposity and skeletal health***
	Speaker: **Clifford J. Rosen**, *Maine Medical Center's Research Institute, United States* *Moderators: Kaisa Ivaska & Tânia Amorim*	
15:40 – 15:55	**Poster Pitching I**	
15:55 – 16:35	*Scientific Workshop*	***Cutting-edge techniques for 3D imaging of BM niches***
	Speaker: **César Nombela Arrieta**, *University Hospital and University of Zurich, Zurich, Switzerland* *Moderators: Souad Daamouch & Andre Van Wijnen*	
16:35 – 17:05	*Scientific Workshop*	***Adipose tissue at single-cell resolution: functional genomics and adipocyte signaling***
	Speaker: **Ling Qin**, *Perelman School of Medicine, University of Pennsylvania, United States* *Moderators: Souad Daamouch & Andre Van Wijnen*	
17:05 – 17:45	*Career Development Workshop*	***How to write grants and grant opportunities***
	Speaker: **Ormond MacDougald**, *University of Michigan, United States* *Moderators: Souad Daamouch & Andre Van Wijnen*	
17:45 – 18:30	**Poster Discussion I**	

Time listed in CET – European time

Thursday, 25th September 2025

Bone Marrow Adipocyte: Between Bone and Blood

14:00 – 14:40	*Scientific Workshop*	***Bone at single cell resolution***
	Speaker: **Ryan C. Chai**, *Garvan Institute of Medical Research, Australia*	
	Moderators: Michaela Tencerova & Biagio Palmisano	
14:45 – 15:25	*Keynote Lecture*	***Bone marrow adipocytes and hematopoiesis***
	Speaker: **Ziru Li**, *Maine Medical Center's Research Institute, United States*	
	Moderators: Michaela Tencerova & Biagio Palmisano	
15:30 – 16:10	*Invited Lecture*	***BMAT mechanosensitivity***
	Speaker: **Glen Niebur**, *University of Notre Dame, United States*	
	Moderators: Michaela Tencerova & Biagio Palmisano	
16:10 – 16:25	**Poster Pitching II**	
16:25 – 17:05	*Invited Lecture*	***BMAT and hematologic cancer-induced bone diseases***
	Speaker: **Claire Edwards**, *University of Oxford, United Kingdom*	
	Moderators: Stéphanie Lucas & Rossella Labella	
17:05 – 17:45	*Scientific Workshop*	***Mitochondrial regulation and adipocyte homeostasis***
	Speaker: **Shingo Kajimura**, *Harvard Medical School, United States*	
	Moderators: Stéphanie Lucas & Rossella Labella	
17:45 – 18:25	*Career Development Workshop*	***Industry and academia***
	Speakers: **Barna Gal,** *Switzerland* & **Davide S. K. Komla Ebri**, *United Kingdom*	
	Moderators: Stéphanie Lucas & Rossella Labella	
18:30 – 19:15	**Poster Discussion II**	

Time listed in CET – European time

Friday, 26th September 2025

Bone Marrow Adipocyte: Fat and Beyond

14:00 – 14:40	*Keynote Lecture*	***Endocrine and metabolic diseases related to BMAT***
	Speaker: **William Cawthorn**, *University of Edinburgh, United Kingdom* *Moderators: Adriana Roque & Jeroen Geurts*	
14:45 – 15:25	*Invited Lecture*	***Nutritional interventions in regulation of bone marrow adiposity***
	Speaker: **Tim J. Schulz**, *German Institute of Human Nutrition Potsdam-Rehbrücke, Germany* *Moderators: Adriana Roque & Jeroen Geurts*	
15:30 – 16:10	*Scientific Workshop*	***Ex vivo models of BMAT***
	Speaker: **Michaela Reagan**, *MaineHealth Institute for Research, United States* *Moderators: Adriana Roque & Jeroen Geurts*	
16:10 – 16:25	**Poster Pitching III**	
16:25 – 17:05	*Scientific Workshop*	***Imaging of BMAT: translational and clinical studies***
	Speaker: **Dimitrios Karampinos**, *Technical University of Munich, Munich, Germany* *Moderator: Gustavo Duque & Greet Kerckhofs*	
17:05 – 17:45	*Career Development Workshop*	***Career development and group/lab establishing***
	Speaker: **Erica Scheller**, *Washington University School of Medicine, United States* *Moderator: Gustavo Duque & Greet Kerckhofs*	
17:45 – 18:30	**Poster Discussion III**	
18:30 – 19:15	***Conclusion Remarks and Award Ceremony*** *Izabela Podgorski, Wayne State University, United States (on behalf of the Organizing Committee)*	

POSTER SESSIONS

Time listed in CET – European time

Poster Pitching and Discussion I – Wednesday, 24th September 2025		
	Poster Number	*Presenter*
Pitching 15:40 – 15:55 **Discussion 17:45 – 18:30**	2	**Mareen Strobeck**, *Germany*
	16	**Jelena Jadžić**, Serbia
	17	**Fabien Bonini**, Switzerland
	5	**Veronika Málková**, Czech Republic
	12	**Mai Ceesay**, United States
	21	**Sara Perpinello**, Italy

Poster Pitching and Discussion II – Thursday, 25th September 2025		
	Poster Number	*Presenter*
Pitching 16:10 – 16:25 **Discussion 18:30 – 19:15**	14	**Maria Chiara Galavotti**, Switzerland
	15	**Radoš Stefanović**, Serbia
	10	**Dávid Ernszt**, Hungary
	4	**Maren Döring**, Germany
	6	**Laura Braud**, France
	11	**Steven Van Offel**, Belgium
	14	**Nicko Widjaja**, Finland

Poster Pitching and Discussion III – Wednesday, 26th September 2025		
	Poster Number	*Presenter*
Pitching 16:10 – 16:25 **Discussion 17:45 – 18:30**	1	**Polona Kalc, Germany**
	3	**Abed Alrazak Al Homsi**, France
	7	**Annabel Mueller**, Belgium
	8	**Bexultan Kazybay**, Netherlands
	9	**Chaimae Nouasria**, Hungary
	18	**Gülsena Tonyali**, Turkey
	13	**Zihao Wang**, United States

POSTER DISCUSSIONS

Time listed in CET – European time

Wednesday, 24th September 2025

Poster Discussion I: Zoom Links

	#	Presenter	Zoom Link	ID	Code
17:45 – 18:30	2	**Mareen Strobeck**	https://us05web.zoom.us/j/83388241046?pwd=2nclhGs5YGbWS1evkyVNS1YW5inURo.1	833 8824 1046	3TRbVv
	16	**Jelena Jadžić**	https://us04web.zoom.us/j/8942924882?pwd=WXNCOHJ1bU5xc3hGME4reXIwWGs2UT09&omn=76065257003		
	17	**Fabien Bonini**	https://unil.zoom.us/j/8987612553		
	5	**Veronika Málková**	https://zoom.us/j/96162755564?pwd=3xBxzbbj5WSqHpslA8xa0luvTh2Ai6.1	961 6275 5564	123
	12	**Mai Ceesay**	https://umassmed.zoom.us/j/98454348711?pwd=mNGgxm2I48uI4zTBpxAz5JoFOumdby.1		859297
	21	**Sara Perpinello**	https://unipd.zoom.us/j/88624825399	886 2482 5399	

Thursday, 25th September 2025

Poster Discussion II: Zoom Links

	#	Presenter	Zoom Link	ID	Code
18:30 – 19:15	14	**Maria Chiara Galavotti**	https://us05web.zoom.us/j/87474095187?pwd=bt5jMVoEwAoWCdxiLBYZfxQUe9Wxha.1	874 7409 5187	dq69Ck
	15	**Radoš Stefanović**	https://us04web.zoom.us/j/78201881133?pwd=dNx22Zbrqyf9UzyKbxNFowLoS0iOap.1	782 0188 1133	ZADmV4
	10	**Dávid Ernszt**	https://us02web.zoom.us/j/84821136832?pwd=3Uo0T1PblgYQqPxgajEVxTHyjylXcX.1	848 2113 6832	207702
	4	**Maren Döring**	https://zoom.us/j/94291498845?pwd=sXfSEAxxO3EUK4Tnmaak2Da6dokL7p.1		
	6	**Laura Braud**	https://us04web.zoom.us/j/71984780751?pwd=CgbuU4UZR2D8VJxe7GcVm7CpEr5vd5.1	719 8478 0751	KTLxR2
	11	**Steven Van Offel**	https://zoom.us/j/98640717622?pwd=5MZhnmVeQD5Xbmbx8KeLGQf3x7J3cD.1	986 4071 7622	BMAS2025
	14	**Nicko Widjaja**	https://utu.zoom.us/j/9916373374?omn=62128988049	991 637 3374	

Friday, 26th September 2025

Poster Discussion III: Zoom Links

	#	Presenter	Zoom Link	ID	Code
17:45 – 18:25	1	**Polona Kalc**	https://uni-jena-de.zoom-x.de/j/4330852854?omn=65649400491	433 085 2854	
	3	**Abed alrazak Al Homsi**	Https://zoom.us/j/99354981500?pwd=Lx0RvPAKnkYUMlWaFZh8ELVdaXLZUC.1	993 5498 1500	1
	7	**Annabel Mueller**	https://zoom.us/j/99718704801?pwd=yLIJRIA9LdTgaMtOw08zaXpqg9Kqfq.1	997 1870 4801	BMAS25
	8	**Bexultan Kazybay**	https://us04web.zoom.us/j/75888261180?pwd=ohgiEvGoHXXb7215YmlP4r8PLA4rvQ.1	758 8826 1180	123
	9	**Chaimae Nouasria**	https://us05web.zoom.us/j/83482927757?pwd=33YhUSaul2Y3DLFw6XwUaJv4QfoKCN.1	834 8292 7757	8IxU2x
	18	**Gülsena Tonyali**	https://us04web.zoom.us/j/79825706307?pwd=UA5fbY6h7yJSJ9ka0hlbnrA0b9Pn30.1	798 2570 6307	1Zn397
	13	**Zihao Wang**	https://umn-private.zoom.us/j/99298341736	992 9834 1736	

ABSTRACT BOOK

Bone Marrow Adiposity Society

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P1

Cranial bone marrow adiposity is associated with higher risk of Alzheimer's disease

Polona Kalc[1], Christian Gaser[1,2,3], Robert Dahnke[1,2,3]

[1] Structural Brain Mapping Group, Department of Neurology, Jena University Hospital, Friedrich Schiller University Jena, Germany
[2] Department of Psychiatry and Psychotherapy, Jena University Hospital, Friedrich Schiller University Jena, Germany
[3] German Center for Mental Health (DZPG)

Keywords: calvaria, structural magnetic resonance imaging, bone marrow adiposity

Low bone mineral density (BMD) is associated with a risk of developing Alzheimer's disease (AD) [1,3,5,6]. However, most large neuroimaging databases do not collect bone-related data. We have therefore developed a toolbox Boney [2] to retrospectively approximate BMD and extract other bone- and adiposity-related measures from structural magnetic resonance images (MRI) of the human head. We applied our toolbox to a large sample from the neuroimaging dataset OASIS-3 [4] (n = 1374, MAge = 70.34 ±9.33, 55% women) and employed a Cox proportional hazards model for the risk of developing AD. The results showed that individuals with higher than median cranial bone marrow adiposity had an increased risk of developing AD in comparison to people with lower cranial bone marrow adiposity (HR: 1.48, IC 95% [1.23-1.78], p<.001). This study highlights the importance of investigating the cranial bone marrow adiposity in relation to neurodegenerative diseases.

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P2

Obesity-associated impairment in bone healing is ameliorated by adiponectin signaling in female mice

Mareen Storbeck[1], Tim J. Schulz[3], Katharina Schmidt-Bleek[1,2]

[1] Julius Wolff Institut, BIH at Charité – Universitätsmedizin Berlin, Berlin, Germany
[2] BIH Center for Regenerative Therapies, BIH at Charité – Universitätsmedizin Berlin, Germany
[3] Department of Adipocyte Development and Nutrition, German Institute of Human Nutrition, Potsdam-Rehbrücke, Nuthetal, Germany

Keywords: BMAT, bone healing, obesity, adiponectin

Abstract not available at the author's request.

P3

Bioinformatic tool (TFinder) uncovers key transcriptional regulators of Sirt1 in bone marrow stromal cells extracted from a mouse model of anorexia nervosa

Abed Alrazak Al Homsi[1], Christophe Chauveau[1], Olfa Ghali Mhenni[1]

[1] *MAB Lab EA4490, ULCO, F-62200 Boulogne-sur-mer, France*

Keywords: RNA-Sequencing, Sirt1, Bone Metabolism, Anorexia nervosa, Transcriptional regulation,

In our previous work using the Separation-based Anorexia (SBA) mouse model, we demonstrated that bone loss is associated with an increased adipogenic capacity of bone marrow stromal cells (BMSCs) and a sustained decrease in Sirtuin 1 (Sirt1) mRNA and protein levels. Sirt1, a histone deacetylase with pro-osteoblastic and anti-adipogenic functions, is crucial for maintaining the osteogenic/adipogenic balance. Preliminary analysis of RNA sequencing data of BMSCs from the SBA model identified 10 down-regulated genes that paralleled the decrease in Sirt1. Subsequent bioinformatics analysis using TFinder predicted that several of the proteins coded by these candidate genes including v-rel reticuloendotheliosis viral oncogene homolog B (RELB), retinoic acid receptor alpha (RARA), CCAAT/enhancer-binding protein beta (CEBPb), hypoxia-inducible factor 1-alpha (HIF1A), and estrogen receptor alpha (ESR1) possess potential binding sites within the Sirt1 promoter, suggesting their direct role in regulating Sirt1 transcription.

This study aims to validate these predictions by selectively silencing these transcription factors in the ST2 cell line and assessing their impact on Sirt1 expression. An 80% silencing of RARA led to an approximately 70% decrease in Sirt1 expression, consistent with its known role in modulating osteogenic differentiation. Similarly, a 80% silencing of ESR1 resulted in about a 60% decrease in Sirt1 expression. Given that ESR1 mediates estrogen signalling and estrogen levels are typically downregulated in anorexia nervosa, this finding aligns with the disrupted reproductive functions observed in the disease. We can assume that increase of adipogenesis at the expense of osteogenesis observed in our SBA model could be due to the downregulation of both RARA and ESR1 which significantly suppresses Sirt1 expression and potentially contribute to the altered osteogenic/adipogenic balance.

Furthermore, we are also currently investigating the impact on Sirt1 expression of HIF1A, CEBPB, and RELB silencing.

Future studies will focus on achieving equivalent silencing efficiencies for RARA in ST2 Cells, as current data indicate that while ESR1 silencing reaches 80% in both ST2 cells and BMSCs from the SBA model, RARA silencing is only 60% in BMSCs compared to 80% in ST2 cells. Once comparable levels are established, we will simulate the SBA model conditions by performing double silencing of RARA and ESR1 in ST2 cells and comparing the outcomes with those observed in BMSCs. The outcomes of these experiments are expected to shed further light on the regulatory mechanisms underlying Sirt1 expression and may identify therapeutic targets for restoring bone formation in anorexia nervosa.

P4

Role of HAS2 in bone marrow niche alterations in the development and progression of type 2 diabetes

Maren Döring[1], Katja Heller[1], JW Fischer[2], Ulrich Flögel[3], Maria Grandoch[1,4]

[1] Institute for Translational Pharmacology Düsseldorf, Medical Faculty, University Hospital and Heinrich Heine University Düsseldorf, Germany
[2] Institute for Pharmacology Düsseldorf, Medical Faculty, University Clinics and Heinrich Heine University Düsseldorf, Germany
[3] Experimental Cardiovascular Imaging, Institute for Molecular Cardiology, Medical Faculty, University Clinics and Heinrich Heine University Düsseldorf, Germany
[4] CARID, Cardiovascular Research Institute Düsseldorf, Medical Faculty, University Hospital Düsseldorf and Heinrich Heine University Düsseldorf, Germany

Keywords: Hyaluronan, Diabetes, Lipogenesis, BMAT

Abstract not available at the author's request.

P5

Effect of high-fat diet on fat and bone metabolism in female B6 and A/J mice

Veronika Málková[1,2], Michaela Ferencakova[1], Martina Dzubanova[1,2], Kim Nguyen Dung[1], Tomas Cajka[3], Martin Rossmeisl[4], Jan Kopecky[4], Michaela Tencerova[1]

[1] Laboratory of Molecular Physiology of Bone, Institute of Physiology of the Czech Academy of Sciences, Prague 142 20, Czech Republic;
[2] Faculty of Science, Charles University, Prague, Czech Republic;
[3] Laboratory of Translational Metabolism, Institute of Physiology of the Czech Academy of Sciences, Prague, Czech Republic
[4] Laboratory of Adipose Tissue Biology, Institute of Physiology of the Czech Academy of Sciences, Prague 142 20, Czech Republic

Keywords: bone marrow adipose tissue, high-fat diet, obesity, females, bone metabolism

Obesity is a global health issue associated with increased fracture risk and elevated bone marrow adipose tissue (BMAT). In this context, bone marrow stromal cells (BMSCs) preferentially undergo adipogenic differentiation, influenced by diet and sex hormones. Notably, men and women exhibit distinct BMAT patterns, likely driven by hormonal and physiological differences that may affect bone integrity. However, the molecular basis of these sex-specific effects remains poorly understood. Most animal studies using high-fat diet (HFD)-induced obesity focus on males, limiting insight into female responses. To address this gap, the present study investigates the impact of HFD on BMAT expansion and bone structure in obesity-prone (C57BL/6) and obesity-resistant (A/J) female mice.

Methods: B6 and A/J female mice (n=10–20 per group) were fed either chow (ND) or HFD (60% kcal fat) for 12 weeks and were assessed for metabolic (glucose tolerance test, body composition), bone parameters (μCT), molecular (gene expression of adipogenic and osteogenic genes), and metabolomics.

Results: Female B6 mice on HFD gained more weight, accumulated more fat mass, lost lean mass (+17%; +35%; –20%, all $p<0.0001$), and showed impaired glucose tolerance compared to A/J females. Despite higher bone mineral content (BMC) in HFD-fed B6 mice (+35%, $p<0.0001$), trabecular bone volume fraction (BV/TV) in tibia was reduced in both strains under HFD. Vertebrae (LV5) analysis showed higher BV/TV (ND +20%, $p<0.001$) and trabecular number (Tb.N) (ND 40% $p<0.0001$; HFD +20%, $p<0.01$) in A/J than B6 females. B6 female mice had higher trabecular thickness (Tb.Th) (ND +10% $p<0.01$, HFD +20% $p<0.0001$). HFD induced greater increases in bone marrow (BM) and plasma lipids in B6 mice. IL-1β expression was upregulated in B6 BM compared to A/J females, while Pparγ and Tnfα remained unchanged in both strains.

Conclusion: HFD-induced changes in bone and fat metabolism were more pronounced in B6 than A/J female mice with different effect of HFD-induced bone impairment compared to males, highlighting the need to further explore strain- and sex-dependent mechanisms.

P6

Role of Bone Marrow Adipose Tissue (BMAT) senescence in normal and pathological hematopoiesis during aging and obesity

Laura Braud[1], R Leblanc[1], JH Guervilly[1], V Geli[1], C Fauriat[1], M Aurrand-Lions[1], C Lachaud[1]

[1] Cancer Research Center of Marseille (CRCM), U1068 INSERM, UMR7258 CNRS, UM105 Aix-Marseille University, Institut Paoli-Calmettes, Marseille, France.

Keywords: BMAT, Senescence, Aging, Obesity, – Hematopoietic alterations

Aging and obesity are risk factors for the development of malignant hematological diseases such as acute myeloid leukemia (AML) (Döhner, 2022). Both are associated with increased cellular senescence, in various tissues, triggered by different stimuli that activate p21 and/or p16 pathways, thereby inducing cell cycle arrest (Muñoz-Espín, 2014). While senescence is known as a powerful anti-tumor mechanism, the accumulation of senescent cells in tissues contributes to numerous pathologies and may, under certain circumstances, promote tumor progression.

Within the bone marrow, bone marrow (BM) adipose tissue (BMAT), composed of adipocytes and preadipocytes, accounts for 45–60% of the total BM volume and 10% of total body adipose tissue in adults. As a component of the BM microenvironment, BMAT was shown to finely regulate normal hematopoiesis. However, its regulatory function, whether supportive or inhibitory, remains controversial. Interestingly, BMAT senescence has been recently identified as a key factor in bone fragility during aging (Liu, 2023), but no studies to date have investigated its impact on hematopoiesis.

My research project aims to explore the role of BMAT senescence in hematopoietic alterations and malignant transformation associated with aging and obesity. My objectives are to determine: (i) the molecular mechanisms involved in BMAT senescence linked to aging and obesity, (ii) the impact of senescent BMAT on normal hematopoiesis, and (iii) its role in the development of AML. This project will help to identify signaling pathways that could be targeted to reduce BMAT senescence and prevent associated hematological dysfunctions.

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P7

Bone properties in obesity: the impact of incretin treatment in mice fed a high-fat diet

Annabel Mueller[1], Jente De Gols[1], Bethan Davies[1], Ingrid Stockmans[1], Karen Moermans[1], Geert Carmeliet[1], Steve Stegen[1]

[1] *Clinical and Experimental Endocrinology, Department of Chronic Diseases and Metabolism, KU Leuven, Belgium*

Keywords: Obesity, incretin receptor agonists, bone properties, bone marrow adipocytes

Obesity is a chronic metabolic disease that has reached the status of a global pandemic. Besides reducing the quality of life, obesity is associated with impaired bone quality and accumulation of bone marrow adipocytes (BMAds), resulting in increased fracture risk. Recently, incretin receptor agonists like semaglutide and tirzepatide were proposed as new anti-obesity drugs. These incretin receptor agonists bind to glucose-dependent insulinotropic polypeptide receptor (GIPR) or glucagon-like peptide-1 receptor (GLP-1R), which results in significant weight loss and restoration of metabolic disfunction. Yet, modulation of GLP-1R and GIPR signaling may affect bone properties, as genetic loss of GIPR lowers bone mineral density and increases fracture risk, whereas administration of GIP in healthy subjects is associated with reduced bone resorption. However, whether and how incretin analogs affect bone and bone cell behavior during obesity remains largely unknown.

To address this research question, we used a preclinical rodent obesity model by feeding mice a high-fat diet (HFD) for 8 weeks, which were subsequently treated with semaglutide or tirzepatide for an additional 4 weeks in HFD conditions. Age-matched control diet (CD)-fed mice were used as experimental controls. As expected, incretin receptor agonist treatment restored systemic metabolic dysfunction, but we observed that this intervention did not improve the HFD-dependent reduction in bone mass. Mechanistically, despite a reduction in BMAd number, bone formation parameters were impaired in HFD-fed semaglutide-treated mice, whereas bone resorption markers were elevated and even further increased upon tirzepatide treatment.

Taken together, these data indicate that despite inducing significant weight loss and restoring metabolic disfunction, incretin receptor agonist treatment does not improve HFD-induced bone loss – likely because of disturbed bone remodeling. Whether this persisting bone defect is related to direct effects on the different bone-resident cell types or because of the continued HFD feeding will be further investigated.

P8

B-cell Acute Lymphoblastic Leukemia-Induced Adipogenic Bias of Bone Marrow Mesenchymal Stromal Cells: Evidence of Skewed Fate and Variable Reprogramming

Bexultan Kazybay[1,2], Mauricio N. Ferrao Blanco[1], Olaf Heidenreich[1,3], Josef Vormoor[1,3]

[1] *Princess Máxima Center for Pediatric Oncology, Utrecht, Netherlands*
[2] *Utrecht University, Utrecht, Netherlands*
[3] *University Medical Center Utrecht, Utrecht, Netherlands*

Keywords: Adipogenic Progenitors, Mesenchymal Stem Cells, B-cell Acute Lymphoblastic Leukemia

Pediatric B-cell acute lymphoblastic leukemia (B-ALL) involves the bone marrow stromal niche, yet comprehensive knowledge about B-ALL cells' ability to modulate mesenchymal stromal cell (MSC) differentiation toward specific lineages remains insufficient. To examine B-ALL-induced priming, we derived MSCs from 4 non-leukemic and 12 pediatric B-ALL patient bone marrow samples and assessed their differentiation potential into adipocytes, osteoblasts, and chondrocytes. Flow cytometry was employed for their phenotypic characterization. Pediatric B-ALL patient-derived xenograft (PDX) cells from mouse spleen were used in a co-culture setting with non-leukemic patient-derived MSCs. MSCs isolated from 2 non-leukemic and 3 leukemic patients retained tri-lineage differentiation ability, colony formation capacity at low density (50 cells/cm2), and human MSC surface marker expression (CD73, CD90, CD105). Although osteogenic and chondrogenic differentiation were comparable, leukemic bone marrow-derived MSCs demonstrated greater adipogenic differentiation tendency, evidenced by increased lipid-containing cell numbers (1781.2 versus 278, $p<0.0001$). Further investigation found three groups of MSCs derived from leukemia patients with varying degrees of adipogenesis: 2 leukemic patient-derived MSCs with impaired adipogenic potential, 4 with normal adipogenic potential (similar to non-leukemic patient-derived MSCs), and 6 with hyper-adipogenic potential. These groups presented similar profiles with respect to leukemic patient's gender, age, and molecular subtype. Hyper-adipogenic MSCs exhibited elevated adipogenic progenitor markers: CD106 (previously identified by our group; 34.5% vs 2.9%), CD54 (8.76% vs 0.72%), and CD26 (21.9% vs 4.3%). Additionally, non-leukemic MSCs resulted in 30%, 42%, and 100% adipocyte number increases post-co-culture with B-ALL PDXs from 3 patients ($p<0.05$). They also showed an upregulation of the pro-adipogenic markers comparable to leukemic patient-derived MSCs suggesting B-ALL PDXs may skew MSC differentiation towards adipogenic fate.

Overall, this study reveals that B-ALL cells actively prime stromal MSCs toward an adipogenic progenitor fate creating heterogeneous subgroups which requires further exploration.

P9

Bone marrow adipose tissue influences response to treatment and metabolism of multiple myeloma cells

Chaimae Nouasria[1,2], Dávid Ernszt[1,2], Béla Kajtár[3], Zoltán Kohl[4], Hussain Alizadeh[4], Zoltán Patonai[5], Zoltán Kellermayer[1,2]

[1] *University of Pécs Clinical Center, Department of Immunology and Biotechnology*
[2] *University of Pécs Szentágothai Research Centre, Multiple Myeloma Microenvironment Research Group*
[3] *University of Pécs Clinical Center, Department of Pathology*
[4] *University of Pécs Clinical Center, 1st Department of Internal Medicine, Division of Hematology*
[5] *University of Pécs Clinical Center, Department of Traumatology and Hand Surgery*

Keywords: Multiple myeloma, bone marrow adipose tissue, microenvironment, drug resistance, metabolism

Abstract not available at the author's request.

P10

Increased percentage of multiple myeloma cells in bone marrow adipose tissue

Dávid Ernszt[1,2], Chaimae Nouasria[1,2], Béla Kajtár[3], Zoltán Kohl[4], Hussain Alizadeh[4], Zoltán Patonai[5], Zoltán Kellermayer[1,2]

[1] *University of Pécs Clinical Center, Department of Immunology and Biotechnology*
[2] *University of Pécs Szentágothai Research Centre, Multiple Myeloma Microenvironment Research Group*
[3] *University of Pécs Clinical Center, Department of Pathology*
[4] *University of Pécs Clinical Center, 1st Department of Internal Medicine, Division of Hematology*
[5] *University of Pécs Clinical Center, Department of Traumatology and Hand Surgery*

Keywords: myeloma, immunology, bone marrow, adipose tissue

Abstract not available at the author's request.

P11

Haversian canals in human cortical bone contain adipocytes

Steven Van Offel[1], Bruno Lapauw[1], Nathalie Bravenboer[2], Charlotte Verroken[1]

[1] *Department of Endocrinology, Ghent University Hospital, Ghent, Belgium*
[2] *Department of Laboratory Science, Amsterdam University Medical Centers, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands*

Keywords: Haversian canals, adipocytes, cortical bone, diabetes

Abstract not available at the author's request.

P12

Human Bone Marrow Adipose Tissue Functions as a Pro Inflammatory Niche for Monopoiesis

Mai Ceesay[1,2,5], Amruta Samant[4,5], Haley Cirka[4], David Kostyra[1,2], Zinger Yang Loureiro[3], Silvia Corvera[1,2,5], Tammy T Nguyen[1,4,5]

[1] *Morningside Graduate School of Biomedical Sciences, UMass Chan Medical School, Worcester, MA USA*
[2] *Program in Molecular Medicine, UMass Chan Medical School, Worcester, MA USA*
[3] *3 Broad Institute of MIT and Harvard, Cambridge, MA USA*
[4] *Department of Surgery, Division of Vascular Surgery, UMass Memorial Medical Center, Worcester, MA USA*
[5] *Diabetes Center of Excellence, UMass Chan Medical School, Worcester, MA USA*

Keywords: Bone Marrow Adipose Tissue (BMAT), Monopoiesis, Leptin Signaling, 3D Organoid

Aging is associated with an expansion of bone marrow adipose tissue (BMAT), a shift toward myeloid-biased hematopoiesis, and immune system aging, all of which contributes to a higher risk of age-related complications such as chronic inflammation, cardiovascular diseases, and hematologic cancers. The mechanisms driving the correlation between aging, BMAT expansion, and myeloid skewing of hematopoietic stem/progenitor cells (HSPCs), are still under investigation. To address the ways in which human BMAT influences HSPCs, we developed matched human 3D tissue organoids from adipose-rich diaphyseal marrow (hBMd), trabecular epiphyseal marrow (hBMe), and subcutaneous adipose tissue (hSAT) from patients undergoing lower limb amputations. We characterized niche cellular composition using single-nucleus RNA sequencing, 3D explant culture, and flow cytometry to assess progenitor expansion and lineage-specific differentiation. We found that hBMd was enriched for transcriptional programs associated with myeloid development. Explants from hBMd sustained progenitor expansion in 3D culture and showed increased monocyte output compared to hBMe and hSAT. Furthermore, conditioned media derived from adipocytes significantly enhanced monocyte differentiation of human $CD34^+$ cells in vitro. Our findings reveal that human BMAT provides a myeloid-biased hematopoietic niche and that adipocyte-derived factors support monopoiesis. This study has the potential to provide mechanistic insight into how BMAT expansion may fuel immune aging.

P13

Bone Marrow Stromal OGT Controls Diabetic Osteoporosis

Chenxin Gu[1], **Zihao Wang[1]**, Hai-Bin Ruan[1]

[1] *Department of Integrative Physiology and Biology, University of Minnesota*

Keywords: OGT, Diabetic Osteoporosis, Bone Marrow Stromal

Abstract not available at the author's request.

P14

Burning Marrow Fat: role of the bone marrow adipocyte maturation axis in hematopoietic stem and progenitor cell function

Maria Chiara Galavotti[1,2], Fabien Bonini[1,2], Olaia Naveiras[1,2]

[1] *University of Lausanne (UNIL)*
[2] *Lausanne University Hospital (CHUV)*

Keywords: Bone Marrow Adipocytes, Differentiation, Haematopoiesis

Bone marrow adipocytes (BMAds) are increasingly acknowledged as dynamic regulators of haematopoiesis. Nevertheless, the critical factors driving their positive or negative regulatory effects remain to be clearly defined.

Evidence from previous studies has demonstrated this complexity with findings that appear contradictory depending on context, anatomical site or experimental model. For instance, Naveiras et al. (2009) highlighted how adipocyte-rich marrow from the caudal vertebrae of mice exhibited a reduced frequency of hematopoietic stem cells (HSCs) and short-term progenitors compared to adipocyte-free marrow of thoracic vertebrae in homeostatic conditions, and how preventing adipocyte differentiation in stress haematopoiesis enhanced hematopoietic recovery. In contrast, Zhou et al. (2017) reported that a deletion of SCF or CXCL12 in adipocytes, through Adiponectin-reporter mice, impaired hematopoietic recovery following lethal irradiation and bone marrow transplantation, especially in adipocyte-rich caudal vertebrae.

These contradictory results may stem from an insufficient granularity in the definition and differentiation of BMAds. Addressing this, Ambrosi et al. (2017) proposed a refined BMAd differentiation axis based on immunophenotypic features: a multipotent progenitor presumably differentiates into Adventitial Reticular Cells (ARCs), then transitions into Adipocyte-primed Cxcl12-Abundant Reticular Cells (AdipoCARs), followed by Adipose Progenitor Cells (APCs), then pre-adipocytes, and ultimately mature BMAds. However, the specific role of each stage along the BMAd maturation axis in regulating haematopoiesis remains to be elucidated. To address this, we will utilize the recently developed mouse BMAd-Cre model, which allows to target specifically the BMAds without affecting other adipose tissues (Li et al. 2022). Using this model, we will test the in vivo and in vitro effect of specific BMAd cell lineage deletion to study their effect in the specific hematopoietic progenitor compartments during stress haematopoiesis.

Naveiras et al. (2009) DOI: 10.1038/nature08099

Ambrosi et al. (2017) DOI: 10.1016/j.stem.2017.02.009

Li et al. (2022) DOI: 10.7554/eLife.78496

P15

Comparative Transcriptomics of Human Bone Marrow Adipose Tissue Cells and Mesenchymal Stromal Cells Uncover Distinct Lineage Profiles

Radoš Stefanović[1], Bianca Schlierf[2,3], Konstantin Horas[3,4], Maximilian Rudert[3,4], Marietta Herrmann[2,3], Drenka Trivanović[2,3,5]

[1] *Group for Immunology, Institute for Medical Research, National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia*
[2] *IZKF Group Tissue Regeneration in Musculoskeletal Diseases, University Hospital Würzburg, Würzburg, Germany*
[3] *Bernhard-Heine-Center for Locomotion Research, University of Würzburg, Würzburg, Germany*
[4] *Orthopedic Hospital König-Ludwig-Haus, University of Würzburg, Würzburg, Germany*
[5] *Group for Hematology and Stem Cells, Institute for Medical Research, National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia*

Keywords: Bone Marrow Adipose Tissue, Mesenchymal Progenitor, Adipogenesis, Osteogenesis, Transcriptomics

Bone marrow adipose tissue (BMAT) exhibits unique adipogenesis, energy metabolism, and inflammatory profile. Comparing to bone marrow mesenchymal stromal cells (BM-MSCs), our preliminary findings indicated significantly higher adipogenic potential in BMAT cells in vitro. I Here, we investigated the transcriptional changes in mesenchymal progenitor cells isolated from BMAT, BM-MSCs, and peripheral adipose tissue stromal cells (PAT) to elucidate differences in adipogenic potential and lineage commitment.

Paired bone marrow (acetabulum) and PAT samples were collected from hip osteoarthritis patients undergoing arthroplasty (n=3). BMAT and PAT samples were enzymatically digested using collagenase and cultured under the same conditions as BM-MSCs prior to RNA sequencing analyses. Bulk RNA sequencing was also performed on sub-cultured cells under basal and adipogenic conditions for 7 days.

Comparative analyses revealed substantial differential gene expression: 368 genes were significantly upregulated and 533 downregulated in BM-MSCs vs BMAT cells; 183 upregulated and 194 downregulated in BM-MSCs versus PAT cells. Notably, no significant transcriptional differences were observed between BMAT and PAT cells under both conditions. Gene Ontology analyses (false discovery rate [FDR] < 0.0.5) indicated altered carbohydrate metabolism (42 genes) and developmental patterning (76) in BM-MSCs compared to BMAT under basal conditions, while adipogenic induction led to downregulation of skeletal (37 genes) and cartilage development genes (19) in BM-MSCs and upregulation of extracellular matrix (20 genes) and skeletal development genes (23) in BMAT cells. Osteogenic markers (ALPL, DCN, MSX2) were significantly higher in BMAT cells relative to BM-MSCs, persisting following adipogenic stimulation. Adipogenic gene expression was comparable between BMAT and BM-MSCs, though lipid biosynthesis gene GPAM was elevated in BMAT under both conditions. BMAT cells exhibited reduced expression of inflammatory receptors (TLR4, TLR1) and OPG compared to BM-MSCs, unaffected by adipogenic stimuli.

These findings indicate that BMAT-derived cells possess distinct lineage commitment and transcriptional profiles, underscoring their unique role in BMAT physiology.

P16

Vertebral bone marrow adiposity and osteocyte lacunar network alterations in men with class 1 obesity: postmortem histomorphometric assessment

Jelena Jadžić[1], Aleksa Lekovic[2], Slobodan Nikolic[2], Marija Djurica[1]

[1] *Center of Bone Biology, Faculty of Medicine, University of Belgrade, Dr. Subotica 4/2, Belgrade, Serbia*
[2] *Institute of Forensic Medicine, Faculty of Medicine, University of Belgrade, Deligradska 31a, Belgrade, Serbia*

Keywords: Obesity, Lumbar Vertebrae, Bone Marrow Adiposity, Osteocyte Lacunar Network, Men

Aim: Recent studies suggest that various skeletal sites may be differently affected in individuals with obesity, while the contribution of vertebral bone marrow adiposity and alterations in the osteocyte lacunar network have not been thoroughly investigated in these individuals to date. We aimed to conduct a marrow adiposity and osteocyte lacunar network histomorphometric assessment of the anterior mid-transverse portion of the first lumbar vertebral body collected from adult male cadavers with class 1 obesity (body mass index [BMI]>30kg/m2) and normal-weight counterparts (BMI between 20 kg/m2 and 25 kg/m2).

Methods: We used histomorphometric assessment to analyze bone marrow adiposity and osteocyte lacunar network morphological features in vertebral samples from adult cadaveric men divided into Ob group (n=10, age: 65 ± 12 years) and control group (n=13, age:61 ± 5 years).

Results: Our data revealed a mild alteration in the morphology of the osteocyte lacunar network in the Ob group, characterized by a higher density of empty osteocyte lacunae in the vertebral trabecular compartment ($p < 0.05$). Moreover, increased mean adipocyte diameter and increased adipocyte bone area were observed in men with class 1 obesity compared to control individuals ($p < 0.05$).

Conclusions: Alterations in bone marrow adiposity and osteocyte lacunar network could be involved in the etiopathogenesis of increased vertebral fragility in men with class 1 obesity. Our data support the need for further studies using state-of-the-art methodologies to understand this topic fully.

P17

From red to yellow: engineering ectopic bone marrow niches to decipher adipocyte-hematopoiesis interplay in aplasia

Fabien Bonini[1], Maria Chiara Galavotti[1], Fabrice Renaud[1], Hugo Wood Verzone[1], Olaia Naveiras[1]

[1] *University of Lausanne*

Keywords: Biomaterials, Hematopoiesis, BMAds, Artificial Niches

Over the past decades, biomaterial-based niches have gained significant attention in regenerative medicine. Initially developed for tissue engineering and organ repair, these artificial systems are now increasingly used as platforms to study fundamental biological processes such as cell-cell and cell-microenvironment interactions. Their key advantage lies in their ability to recreate physiologically relevant conditions with reduced complexity and enhanced experimental accessibility. These qualities are particularly valuable when studying complex organs like the bone marrow (BM) that presents unique experimental challenges. For instance, BM exhibits extreme biomechanical heterogeneity, with the soft marrow tissue surrounded by rigid, opaque bone. This structural complexity, coupled with the presence of multiple spatially distinct subniches, makes the in vivo study of specific cellular interactions difficult.

We present a biomaterial-based ectopic bone marrow artificial niche (eBMAN) designed to replicate the red-to-yellow marrow transition observed during aplasia, a hematologic condition in which the bone marrow fails to produce sufficient blood cells as a result of chemotherapy-induced damage. The eBMAN consists of a highly porous biomaterial scaffold resembling trabecular bone structure, seeded with primary human bone marrow stromal cells. These stromal cells are either maintained in an undifferentiated state (red-eBMAN) or differentiated into adipocytes (yellow-eBMAN) prior to minimally invasive ectopic implantation into the subcutaneous space of mice. After three weeks in vivo, the niche develops a dense vascular network composed of host-derived vessels. Subsequently, eBMANs are seeded with human CD34+ hematopoietic stem and progenitor cells (HSPCs) and kept for several week/months, enabling the long-term comparison of stromal- versus adipocyte-derived support for hematopoiesis under physiological and aplasia-like conditions.

This approach provides a physiologically relevant yet experimentally accessible model to study how marrow remodeling along the adipocytic axis affects hematopoeisis. eBMAN addresses a major gap in BM research and may enable innovative therapeutic strategies bone marrow failure.

P18

High-Resolution Lipidomics Reveals Distinct Profiles in Bone Marrow Versus Abdominal Adipose Depots

Gülsena Tonyalı[1,2,3,4], Bihter Muratoğlu[1,2,3,4], Ceren Tuncer[1,2,3,4], Cansu Özdemir[1,2,3,4], Cemil Can Eylem[1,2,3,4], Emirhan Nemutlu[1,2,3,4], Kemal Kosemehmetoglu[1,2,3,4], Fazil Tuncay Aki[1,2,3,4], Duygu Uçkan-Çetinkaya[1,2,3,4]

[1] *Center for Stem Cell Research and Development (PEDI-STEM), Hacettepe University, Ankara, Türkiye*
[2] *Department of Stem Cell Sciences, Hacettepe University Graduate School of Health Sciences, Ankara, Türkiye*
[3] *Division of Hematology-Oncology, Faculty of Medicine, Department of Pediatrics, Hacettepe University, Ankara, Türkiye*
[4] *Research Center for Fanconi Anemia and Other IBMFSs, Hacettepe University, Ankara, Türkiye*

Keywords: Lipidomics, Bone Marrow, Abdominal Depots, Adipose Tissue

Bone marrow adipose tissue (BMAT) is increasingly recognized as a metabolically active depot with distinct physiological functions. While abdominal adipose tissues (including mesenteric, omental, subcutaneous, and perirenal depots) are well studied in metabolic disease, BMAT remains understudied. This study aimed to systematically compare the lipidomic composition of BMAT versus abdominal fat depots to identify depot-specific lipid signatures.

Methods: Adipose tissue samples were collected from five anatomical regions of healthy human bone marrow and renal transplant donors: bone marrow (n=7), mesenteric, omental, subcutaneous, and perirenal (n=10). Untargeted lipidomics was performed using high-resolution LC-MS/MS. Data were analyzed with MetaboAnalyst 6.0 tool.

Results: BMAT exhibited a clearly distinct lipid profile, showing significant separation from all abdominal fat depots. The strongest divergence was observed between BMAT and perirenal adipose tissue. Volcano plot analysis revealed that BMAT is enriched in phospholipids (e.g., PC 34:2, PE 38:4), while abdominal depots are dominated by triglyceride species. These results highlight BMAT's unique lipidomic identity and metabolic role. Still, it should be noted that the processing of BMAT was different from abdominal samples.

Conclusion: BMAT is metabolically distinct from abdominal adipose tissues, suggesting its potential involvement in bone-related pathologies and systemic energy regulation. In our ongoing project, the same tissues are being analyzed at the transcriptomic, proteomic, and targeted metabolomic levels to further uncover depot-specific functions.

P19

The Persistent Preference for Traditional Bonesetting in a large East African Country: Risk Factors and Missed Opportunities

Mengistu Gebreyohanes Mengesha[1], Morgan Askew[2], Abdoulie Njai[2,3], Ephrem Gebrehana[1], Mario Keko[4], Sintayehu Bussa[1], Yisihak Zerihun[1], Matiyas Seid[5], Mahamed Areis[6], Birlew T. Alemayehu[7], Bekalu Wubshet[8], Moa C. Jira[9], Habtamu B. Gula[10], Eyueal Ambaye[11], Claude Martin Jr[12], William J. Harrison[12], Kiran J. Agarwal-Harding[2,4]

[1] *Department of Orthopedic Surgery, Hawassa University Comprehensive Specialized Hospital, Hawassa, Ethiopia*
[2] *Harvard Global Orthopaedics Collaborative, Boston, MA, USA*
[3] *University of Missouri, Department of Orthopaedic Surgery, Columbia, MO, USA*
[4] *Department of Orthopaedic Surgery, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA*
[5] *Department of Orthopedic Surgery, Wachemo University Hospital, Ethiopia*
[6] *Department of Orthopedic Surgery, Jigjiga University Hospital, Ethiopia*
[7] *Department of Orthopedic Surgery, Gondar University Hospital, Ethiopia*
[8] *Department of Orthopedic Surgery, Bahir Dar University Hospital, Ethiopia*
[9] *Department of Orthopedic Surgery, Haramaya University Hospital, Ethiopia*
[10] *Department of Orthopedic Surgery, Assela University Hospital, Ethiopia*
[11] *Department of Orthopedic Surgery, Jimma University Hospital, Ethiopia*
[12] *AO Alliance Foundation, Davos, Switzerland*

Keywords: Traditional Bonesetting, Complications, Musculoskeletal Injury, Preferences, Ethiopia

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P20

The Transcriptomic Analysis of Rat Bone Marrow Adipose Tissue Reveals Enrichments of Pathways Related to The Proliferation of Pre-adipocytes

Nicko Widjaja[1], Niki Jalava[1], Kaisa K. Ivaska[1]

[1] *Institude of Biomedicine, University of Turku, Finland*

Keywords: Bone marrow adipose tissue, Transcriptome, RNAseq, Preadispocyte proliferation, Gene expression

Introduction

Bone marrow adipose tissue (BMAT) is a functional fat depot distinct from extramedullary white (WAT) and brown (BAT) adipose tissues. The effect of metabolic disorders on the expansion of these fat depots has been well documented. However, the differences in the transcriptomic landscape between these adipose tissues under normal physiological conditions are less understood. This study aimed to compare the transcriptome of the adipose tissue triad of BMAT, WAT and BAT in rats.

Methodology

Adipose tissues from 20-wk-old male Sprague-Dawley rats were collected from perigonadal region (WAT), interscapular region (BAT) and bone marrow of femur and tibia (BMAT). Sample quality was validated with histology and mRNA expression of known adipocyte markers by qPCR. Total RNA (N=6/tissue) were extracted and analysed using bulk-RNAsequencing. Transcriptomic analyses were performed to compare gene set enrichment analysis (GSEA) and differentially expressed genes (DEGs) between BMAT and WAT/BAT. Expression of select genes were evaluated by qPCR to verify RNAseq data.

Results

Principal component analysis of the transcriptomes revealed three distinct clusters representing BMAT, WAT and BAT. Expression levels of adipocyte-specific markers, e.g. adipoq, fabp4, cd36 and leptin, were significantly lower in BMAT when compared to WAT/BAT. GSEA revealed enrichment of pathways related to cell cycle and phospholipid processes in BMAT, while pathways related to energy metabolism and adipocyte function were more enriched in BAT and WAT, respectively. Analysis of DEGs revealed higher expression level of transcripts known to be expressed by adipocyte progenitors and related to proliferation of pre-adipocytes in BMAT compared to WAT or BAT.

Conclusion

Our results highlight that the transcriptome of BMAT differs from WAT and BAT, particularly by the lower expression of adipocyte-related genes. Whole-tissue GSEA and DEGs analyses suggest abundant presence of pre-adipocyte population within BMAT. Overall, this may suggest site- and age-dependent adipocyte maturation profile in rats.

P21

Decoding Bone Marrow Adipocytes Contribution to Acute Myeloid Leukemia in a Biomimetic 3D Approach

Sara Perpinello[1], Maddalena Benetton[1], Ambra Da Ros[1], Giorgia Longo[1], Claudia Tregnago[1], Martina Pigazzi[1,2]

[1] *University of Padova, Department of Women's and Children's Health, Padova, Italy*
[2] *Fondazione Istituto Ricerca Pediatrica (IRP), Padova, Italy*

Keywords: Acute Myeloid Leukemia, 3D model

Abstract not available at the author's request.

LIST OF PARTICIPANTS

Surname	Name	Email	Country
A			
Achudhan	David	achudhan.david@mayo.edu	United Kingdom
Al Homsi	Abed alrazak	abedalrazak.alhomsi@univ-littoral.fr	France
B			
Banjanin	Bella	bella.banjanin@bih-charite.de	Germany
Bonini	Fabien	fabien.bonini@unil.ch	Switzerland
Braud	Laura	laura.braud@inserm.fr	France
C			
Ceesay	Mai	mai.ceesay@umassmed.edu	United States
Cheng	Wen-Hui	cheng_w@ukw.de	Germany
D			
Döring	Maren	maren.doering@hhu.de	Germany
E			
Ernszt	Dávid	ernszt.david@pte.hu	Hungary
F			
Falray	Delphine	delphine.farlay@inserm.fr	France
G			
Galavotti	Maria Chiara	maria.galavotti@unil.ch	Switzerland
Gámez Molina	Beatriz	beatriz.gamezmolina@nds.ox.ac.uk	United Kingdom
H			
Hagag	Eman	eman.hagag@ukdd.de	Germany
Hariri	Hadla	hadharir@umich.edu	United States
Hermsmeyer	Isabela	idherms@umich.edu	United States
I			
Ikushima	Yoshiko	yoshiko.ikushima.a5@tohoku.ac.jp	Japan
Iwaniec	Urszula	Urszula.Iwaniec@oregonstate.edu	United States
J			
Jadžić	Jelena	jelena.jadzic@med.bg.ac.rs	Serbia
Jafar	Calar Sedik	calar.jafar@hhu.de	Germany
K			
Kalc	Polona	polona.kalc@med.uni-jena.de	Germany
Kalev-Altman	Rotem	rotemka@uchicago.edu	United States
Kazybay	Bexultan	b.kazybay@prinsesmaximacentrum.nl	Netherlands
Kellermayer	Zoltán	kellermayer.zoltan@pte.hu	Hungary
Kukolj	Tamara	tamara.kukolj@imi.bg.ac.rs	Serbia
L			
Lan	Yanhua	448116276@qq.com	United States
Li	Ziru	liziru019@gmail.com	United States
Lindsay	Jaeshia	jalindsay@augusta.edu	United States

Surname	Name	Email	Country
M			
Málková	Veronika	veronika.malkova@fgu.cas.cz	Czech Republic
Mcgregor	Emma	emma.mcgregor@nds.ox.ac.uk	United Kingdom
Mengesha	Mengistu Gebreyohanes	mengistugy@gmail.com	Ethiopia
Mueller	Annabel	annabel.mueller@kuleuven.be	Belgium
N			
Neto	Nailton	nailton.jose.016@ufrn.edu.br	Brazil
Nouasria	Chaimae	nouasria.chaimae@pte.hu	Hungary
P			
Perez	Oasis	oaperez@students.llu.edu	United States
Pellegrini	Niccolo	n.pellegrini@eramusmc.nl	The Netherlands
Perpinello	Sara	sara.perpinello@phd.unipd.it	Italy
S			
Salbach-Hirsch	Juliane	Juliane.salbach-hirsch@uniklinikum-dresden.de	Germany
Sjostrom	Samuel	s2456042@ed.ac.uk	Scotland
Stefanović	Radoš	rados.stefanovic@imi.bg.ac.rs	Serbia
Storbeck	Mareen	mareen.storbeck@bih-charite.de	Germany
T			
Taibi	Thilelli	thilelli.taibi@cea.fr	France
Tonyali	Gülsena	gulsenatonyali@gmail.com	Turkey
Trikha	Sonia	sonia.trikha@gimm.pt	Portugal
V			
Van Gijseghem	Julie	julievangijseghem@gmail.com	Belgium
Van Offel	Steven	steven.vanoffel@uzgent.be	Belgium
W			
Wang	Zihao	wang9719@umn.edu	United States
Whipp	Danielle	daniwhipp@gmail.com	United Kingdom
Widjaja	Nicko	nicko.v.widjaja@utu.fi	Finland