Annual Meeting of the German Society for Matrix Biology

Frankfurt, September 08-10, 2022

Invited Speakers

Ishier Raote

PhD from the National Centre for Biological Sciences (NCBS), Bangalore, India with Professor Panicker, studying ligand-dependent trafficking of serotonin receptors.

In 2014, Ishier moved to the Centre for Genomic Regulation (CRG), Barcelona, Spain, to the group of Professor Vivek Malhotra. During his postdoc, Ishier discovered the central role of TANGO1 as a master organiser of early secretory pathway membranes, particularly to export bulky cargoes such as collagens from the endoplasmic reticulum (ER). Historically it was thought that proteins leave this compartment in small vesicles; Ishier proposed and obtained data indicating that larger cargoes including collagen use transient, inter-organelle tunnels to reach the Golgi apparatus

Next year, Ishier will start his own group at the Institut Jacques Monod, Paris, to build on his studies on cells secrete and assemble an extracellular matrix.

Qing-Jun Meng

Professor of Chronobiology, Versus Arthritis Senior Research Fellow, Director of Internationalisation (School of Biological Sciences, University of Manchester). He is the "Chrono-Matrix" Theme Leader of the Wellcome Centre for Cell-Matrix Research, Bursary Chair of the British Society for Matrix Biology, Co-director and Board member of the Wellcome Trust Immuno-Matrix in Complex Diseases (ICD) PhD program.

Qing-Jun started his post-doctoral training on circadian clocks at the University of Manchester in 2003. In 2009, he received a five-year MRC Career Development Fellowship Award to start his independent group. This was followed by a Versus Arthritis Senior Research Fellowship Award in 2015. He was promoted to a Professor of Chronobiology in 2017. His earlier research has contributed to the understanding of the molecular clocks and their pharmacological regulation (Neuron 2008; Curr Bio 2010; PNAS 2010; Nucleic Acids Res 2014). His current interest is the interface between circadian biology, extracellular matrix homeostasis and age-related diseases, including osteoarthritis (Arthritis & Rheum 2013; Osteo & Cartilage 2015/2021; JCI 2016; Theranostics 2022; Science Advances 2022), intervertebral disc degeneration (Annals Rheum Dis 2017/2021; Bone Res 2022), fibrosis and tendinopathies (Genes & Dev 2014; Sci Rep 2014; Nat Cell Bio 2020) and breast cancer (Nat Comms 2017; J Cell Sci 2018; Breast Cancer Res 2018). He has received research funding from the MRC, BBSRC sLoLa, Wellcome Trust, Versus Arthritis, Breast Cancer Now and industries (GSK and Walgreens Boots Alliance). His research profile has received extensive media coverage including BBC Breakfast TV, BBC Radio Stoke, BBC Radio 5, BBC Radio Manchester.

1) Chang J, Garva R, Pickard A, Yeung C-YC, Mallikarjun V, Swift J, Holmes DF, Calverley B, Lu Y, Adamson A, Raymond-Hayling H, Jensen O, Shearer T, Meng*QJ and Kadler*KE (2020). Circadian control of the secretory pathway maintains collagen homeostasis. **Nat Cell Biology**, 22, 74.

2) Yang N, Williams J, Pekovic-Vaughan V, Wang P, OlabiS, McConnell J, Gossan N, Hughes A, Cheung J, Streuli* CH & Meng*QJ (2017). Cellular mechano-environment regulates the mammary circadian clock. **Nat Comms.** 8,14287.

3) Dudek M, Yang N, Ruckshanthi JP, Williams J, Borysiewicz E, Wang P, Adamson A, Li J, Bateman JF, White MR, Boot-Handford RP, Hoyland*JA & Meng*QJ (2017). The intervertebral disc contains intrinsic circadian clocks that are regulated by age and cytokines and linked to degeneration. **Annals Rheum Dis.** 76, 576. Highlighted by Nat Rev Rheum: Catching the rhythm of disc degeneration.

4) Dudek M, Gossan N, Yang N, Im HJ, Ruckshanthi JPD, Yoshitane H, Li X, Jin D, Wang P, Boudiffa M, Bellantuono I, Fukada Y, Boot-Handford* RP and Meng* QJ (2016). Thechondrocyte clock gene Bmal1 controls cartilage homeostasis and integrity. **J Clin Invest.** 126, 365. Cover Story| Highlighted by Nat Rev Rheum: Chondrocyte clock maintains cartilage tissue.

Shireen Lamandé

Shireen co-leads the Musculoskeletal Research group at the Murdoch Children's Research Institute, Melbourne, Australia. Her PhD work defined collagen I mutations underlying osteogenesis imperfecta and since then her research has continued to focus on the molecular genetics of inherited disorders of the musculoskeletal system. Her research team has defined mutations leading to skeletal disorders and muscular dystrophies, and their work is characterized by detailed biochemical studies that explore the effect of the mutations on protein structure and function and the organization of the extracellular matrix of musculoskeletal tissues. In addition to defining genotype-phenotype relationships that pave the way for improved diagnosis and developing rational therapeutic approaches, her work also contributes to understanding the complex multimeric assembly processes and interactions that underlie the formation of architecturally precise and tissue-specific extracellular matrices that provide both a structural scaffolding and regulatory signals for growth and development.

Andrew Pitsillides

My group's research takes place at the structure-function interface, addressing *how biology maintains normal tissues* and w*hy this fails so devastatingly in disease?* It focuses on bone and joint mechanobiology and has embraced embryology - where these relationships emerge - through growth, maturation and ageing, when these tissues frequently fail. My group's research interests have been broad and encompass classic cell/molecular biology of many joint tissues, in *vitro/in vivo/ex vivo* and *in silico* methods applied to both animal and human bone and joint function, embryonic limb development and cell signalling. My research addresses the full pathophysiological range to include osteoarthritis and osteoporosis, commonly addressed from a mechanobiology perspective. Most recently my team have been focused on using in vivo models to study bone mechanoadaptation and post-traumatic osteoarthritis. The group embraces new technologies: to date this includes a multitude of imaging techniques (Synchrotron/RAMAN/MRI, nano- and micro-CT), analysis of gait, measurement of pain, finite element analysis, complex registration techniques including digital image/volume correlation (DIC/DVC) and complex statistical modelling from a comparative biology perspective.

I have given plenary conference presentations at International/National Conferences (>45), Commercial meetings and at International and National Research Institutes, Universities and Medical Schools (>35) on joint formation, bone mechanobiology and the osteochondral interface. My group are currently funded by major grants from MRC, Versus Arthritis, EPSRC and commercial sponsors. I have published ~180 full papers/review articles, have an h-index of 48 (i10-index of 128), ~8000 citations and would estimate that ~50% are collaborative with many ongoing. I have always enjoyed team-working immensely. My natural tendency is to be creative and to strive to solve problems in unconventional ways. This is driven by an inquisitive nature and is coupled to a desire to get to the bottom of a problem and solve it or, more often these days, find someone who can.

Malgorzata Wygrecka

Malgorzata Wygrecka is a W2 professor for diffuse parenchymal lung diseases of the German Center for Lung Research (DZL) at Center for Infection and Genomics of the Lung in Giessen. Her research focuses on the role of hemostatic proteases and their receptors in the pathogenesis of inflammatory and fibroproliferative lung diseases. She has over 100 peer-reviewed publications that are amongst others published in the Nature Communications, Circulation, Blood, Circulation Research, PNAS, Matrix Biology, Kidney International, European Respiratory Journal, and American Journal of Respiratory and Critical Care Medicine. She received many prestigious awards including the research award of the German Society for Pneumology and Respiratory Medicine (DGP), the young investigator award of the German Society for Internal Medicine (DGIM), the Behring-Röntgen Foundation research award, and the young investigator award of the International Society for Serpin Biology. She holds two patents for protease inhibitors in chronic lung diseases. She acknowledges financial support of the German Research Foundation, Else-Kröner-Fresenius Foundation, Von Behring-Röntgen Foundation, LUNGENFIBR02E®e.V. Foundation, Federal Ministry of Education and Research (BMBF), and CSL Behring Innovation. She is a member of the Scientific and Standardization Subcommittee "Factor XI and the Contact System" of the International Society on Thrombosis and Haemostasis and one of the leading scientists of the COSYNE consortium which is dedicated to the validation of the new diagnostic tools for the contact phase system proteins.

Sandra Pohl

Sandra received the PhD in Biochemistry from the University of Hamburg in 2007. She was a project leader at the Children´s Hospital at the University Medical Hamburg-Eppendorf (UKE) before she became the head of the research group Skeletal Pathobiochemistry at the Institute of Osteology and Biomechanics. Since 2021 she holds a Heisenberg Professorship for Subcellular Osteology at the UKE, and also serve as Deputy Head of the international, rare disease Competence Center for Lysosomal Disorders.

She is an experienced cell biologist with strong expertise in functional protein biochemistry. Her research group aims to understand the molecular mechanisms balancing skeletal matrix synthesis and degradation and to elucidate the pathophysiological basis of inherited skeletal disorders in childhood.

Gertraud Orend

Gertraud Orend had studied Genetics, Biochmemistry, Pharmacology and Organic Chemistry in Cologne, Germany. She did her PhD in the laboratory of Walter Doerfler on epigenetic modifications arising from insertional mutagenesis in Adenovirus-induced tumors. Her observations contributed to the understanding of de novo methylation across the genome, a mechanism that plays an important role in cancer. She got interested in the question how cell adhesion regulates cell cycle progression in normal cells and, how this anchorage-dependence is subverted in tumor cells which she studied as postdoc in the laboratory of Erkki Ruoslahti at the Burnham Institute in La Jolla, CA, USA. Inspired by her cell adhesion work she got interested in the signal transduction pathways that are initiated by the interaction of cells with the extracellular matrix molecules fibronectin and tenascin-C, which she studied as research associate in the laboratory of Ruth Chiquet-Ehrismann at the Friedrich Miescher Institute for Biomedical Research, Basel, Switzerland. Then she directed the Tumor Matrix group at the University of Basel, Switzerland in the Department of Biomedicine led by Gerhard Christofori, where her group continued to work on signal transduction pathways that are induced by cell adhesion to tenascin-C. She became lab head of two INSERM teams, U682 and U1109 (MN3T, The Microenvironmental Niche in Tumorigenesis and Targeted Therapy) in Strasbourg. She took tenascin-C research to a new level by developing mouse models with engineered expression of tenascin-C. Her current work is focussed on the understanding of the roles of tenascin-C in the tumor microenvironment promoting angiogenesis and metastasis and, shaping tumor immunity as well as the roles of tenascin-C in chronic inflammation.

Didier Stainier

Born 1963 in Liège, Belgium. Studied Biology at the United World College of the Atlantic (Wales, UK) IB (1981), Université de Liège (Belgium), and Brandeis University (USA) B.A. (1984). Ph.D. in Biochemistry and Molecular Biology at Harvard University (1990). Postdoc at Massachusetts General Hospital (Boston). Assistant Professor at the University of California San Francisco (UCSF) in 1995, Associate Professor in 2000, Full Professor in 2003. Director and Scientific Member at the Max Planck Institute for Heart and Lung Research (since 2012).