ANATOMISCHE GESELLSCHAFT

1886

DAS HISTORISCHE HAUS DER ANATOMIE IN DER KÖLLIKERSTRAßE

... UND ES BLEIBT HISTORISCH IN DER JOSEF-SCHNEIDER-STRAßE

119th Annual Meeting / 35. Arbeitstagung

der Anatomischen Gesellschaft

24. bis 26. September 2025

am Institut für Anatomie und Zellbiologie

der Universität Würzburg

1886

To find your abstract or

an abstract of interest

please use the alphabetical list of

first authors of lectures and posters
starting on next page.

Erstautor/First author	Nr. des Vortrages (V) / Posters (P)
Acker E.	P 9
Amunts K.	P 100
an Mey N.	V 40
Andric L.	P 139
Ankermann P.	P 158
Antipova V.	P 153
Arlt E.	P 75
Auer S.	P 105
Bartelt-Kirbach B.	P 94
Baskar K.	V 8
Bayerbach F.	P 7
Becht M.	P 25
Behrangi N.	V 19
Bellingkrodt A.	P 46
Bernhard R.	P 90
Bester B.	V 49
Beyersdorfer V.	P 28
Blischke L.	P 93
Blumer R.	P 120
Bömmel H.	P 32, P 150
Bosio L.	P 92
Böttcher N.	P 85
Braun L.	P 70
Braunger B.	V 24
Brümmel J.	P 78
Burkert L.	P 124
Busch M.	V 4
Caspary R.	P 81
Chadikovska E.	P 56
Chiot A.	V 41
Christle R.	P 117
Claassen H.	P 48
Colpaert R.	V 1
Cortés Sánchez J.	P 80
Dahlke E.	V 10
Dames C.	P 149
Dharmapuri A.	P 13

Erstautor/First author	Nr. des Vortrages (V) / Posters (P)
Dillinger A.	P 123
Dodel K.	P 102
Dogan L. E.	P 164
Domagala Z.	P 86
Eiberger B.	P 147
Eichler A.	V 23
El Khattabi S.	P 116
Eppler E.	V 29
Erdmann-Wolff I.	P 104
Ermer K.	P 79
Ettner-Sitter A.	P 141
Evers S.	V 5
Fassin C.	P 111
Fleischmann N.	P 11
Freudenmacher L.	P 87
Frintrop L.	P 155
Furch L.	P 21
Galanis C.	V 50
Gebert M.	P 125
Gehrich P.	P 74
Geigenfeind M.	V 6
Gericke M.	V 16
Giesbrecht T.	P 113
Ginoski V.	P 77
Glomb O.	V 34
Göhler J.	P 107
Götz L.	V 47
Grammer M.	P 76
Gruijs da Silva L.	P 131
Guevara C.	P 135
Hainfellner A.	P 88
Hallenberger J.	P 95
Hami J.	P 99
Hannig L.	V 15, P 17
Harrer A.	V 43
Heiden R.	P 145
Heimbucher J.	V 28
Hemkemeyer S.	P 30
Hermann K.	P 151
Hiermaier M.	V 12
Hinganu D.	P 161

Erstautor/First author	**Nr. des Vortrages (V) / Posters (P)**
Hinganu M. V.	P 160
Hoffmann A.	P 110
Hohmann U.	P 114
Horn-Bochtler A.	P 103
Immig K.	P 54
Ivanova N.	P 58
Ji H.	P 121
Joost S.	V 36
Jüngert K.	P 65
Kadr H.	P 24
Kajese E.	V 17
Kampelmann M.	P 44
Karlaftis I.	P 98
Kefalakes E.	V 26
Kenst K.	P 140
Kepser L.	P 118
Klimkina M.	P 8
Klumpp C.	P 29
Koch J.	V 44
Kolothara Unnikrishnan M.	V 33
Kordt L.	P 101
Koretz C.	V 45
Korten N.	P 96
Kruse P.	V 46
Kümmel M.	P 112
Lancelle L.	P 133
Lange T.	V 14
Langer J.	P 136
Lautenschläger L.	P 45, P 57
Lemberger-Viehmann L.	V 3
Lisovaya S.	P 40
Lülsberg F.	P 134
Mahmoud W.	P 59
Märker M.	P 15
Maronde E.	P 6
Martin C.	P 119
Mastaka S.	P 82
Matthias F.	P 20
Matveeva N.	P 83
Maurer S.	P 142
Mei V.	P 143

Erstautor/First author	Nr. des Vortrages (V) / Posters (P)
Meien S.	P 128
Meier T.	P 61
Meinke L.	P 137
Melchers A.	P 12
Merz J.	V 38
Meuser A.	V 20
Mietens A.	P 16
Mirontsev A.	P 50
Montagner A.	P 144
Moysidou Tsiorva P.	V 9
Müller L.	P 73
Naert T.	P 42
Nam H.	P 152
Neckel P.	V 32
Niebauer A.	P 106
Niggetiedt E.	P 122
Offt A.	P 33
Olschewski Ridruejo D.	P 109
Özbaykent B.	P 132
Padilha M.	V 27
Pathak S.	V 11
Paunkoska A.	P 52
Peitz K.	V 48
Peter F.	P 49
Pfeiffer F.	P 127
Pidvalna U.	P 66
Poggi G.	P 154
Pratama A.	P 26, P 27
Pruidze P.	P 67, P 69
Raffauf A.	P 36
Reinert J.	P 157
Röck R.	P 31
Rütten S.	P 1
Sangari M.	P 64
Sanna A.	P 146
Sava A.	P 156
Schameitat M.	P 22
Schaub T.	V 31
Schicht M.	P 2
Schierz A.	P 41
Schlecht A.	V 39

Erstautor/First author	Nr. des Vortrages (V) / Posters (P)
Schmidt J.	P 43
Schmitt T.	P 5
Schneider K.	V 2
Schnell A.	P 108
Schoebel M.	P 130
Schueler J.	P 148
Schulz F.	P 34
Schulze-Tanzil G.	P 18
Schumann S.	P 51
Seyedian Moghaddam A.	P 47
Sommer V.	P 84
Steffke C.	V 25
Steinke H.	P 162
Stelzner S.	V 21
Steudle S.	P 72
Steudte L.	P 4
Streich S.	P 63
Sunjic L.	P 138
Sunohara M.	P 38
Tillmann B.	P 53
Tillmann N.	V 42
Tofan K.	P 97
Tourbier V.	P 71
Trenkel J.	P 68
Trinh S.	P 91
Tulski M.	P 60
Turi Z.	V 35
Veneris S.	V 13
Vidovic N.	P 163
Vlasenko D.	V 18
Vogel L.	P 14
Vogt J.	V 22
Vollmer J.	P 35
Wang Y.	V 30
Weissbach L.	P 89
Wiemann S.	P 39
Wiesehöfer M.	V 37
Winnewisser L.	P 3
Woelfle S.	P 115
Wolters L.	P 126
Wörsdörfer P.	V 7

Erstautor/First author	Nr. des Vortrages (V) / Posters (P)
Wozniak S.	P 62
Wree A.	P 55
Yamamoto T.	P 159
Yurchenko A.	P 129
Zahn I.	P 10
Zhang Z.	P 23
Zhao N.	P 37
Zillmer F.	P 19

Poster 1:

Title:
From Reef to Relief: Coccoliths as Bone Graft Substitutes

Authors:
Anna Sophia Keller (Aachen, Germany)
Johanna Zander (Karlsruhe, Germany)
Stephan Rütten (Aachen, Germany)
Thomas Pufe (Aachen, Germany)
Mersedeh Tohidnezhad (Aachen, Germany)
anna.sophia.keller@rwth-aachen.de

Abstract:
The demand for bone reconstructive surgeries that use grafting techniques to address critical bone defects has increased in recent years. This necessitates advancing current treatment methodologies to overcome limitations of auto-, allografting and synthetic bone grafting materials such as BETA-tricalcium phosphate. The calcite particles, coccoliths, derived from the coccolithophore Emiliania huxleyi, possess a favourable chemical composition and structure, making them a promising candidate to advance the way we treat critical bone defects.

We investigated the cytotoxic effects of coccoliths, in combination with collagenisation, on the osteogenic differentiation of the murine pre-osteoblastic MC3T3-E1 subclone 4 cells. The mineralisation level of a long-term differentiated cell culture, and the VEGF and TNF-ALPHA secretion via ELISA were determined. RUNX2 and Osteocalcin expression were observed via immunofluorescent staining.

Coccoliths exhibited no cytotoxicity on MC3T3-E1 cells, even in high concentrations. A positive trend in the improvement of mineralisation can be observed in cells grown on collagenised coccoliths. No adverse reaction in expression and secretion of VEGF, RUNX2 and Osteocalcin was found. Coccoliths caused no TNF-ALPHA-induced pro-inflammatory response against coccoliths.

Coccoliths have proven themselves to be non-toxic on MC3T3-E1 in the observed period. No pro-inflammatory response was seen, suggesting a high biocompatibility. Coccoliths had no negative influence on the secretion of bone growth-related proteins. The positive trend in mineralisation level, when applied with collagen, shows that coccoliths are a promising candidate as a bone grafting substitute, and further research in the application methodology of coccoliths will demonstrate the potential of marine-derived biomaterials as bone graft substitutes.

Poster 2:

Title:
Differential TGF-β2 expression in the anterior and posterior eye segments in mice with early streptozotocin-induced diabetes

Authors:
Martin Schicht (Erlangen, Germany)
Klaus Scholich (Frankfurt am Main, Germany)
Marco Sisignano (Frankfurt am Main, Germany)
Gerd Geisslinger (Frankfurt am Main, Germany)
Friedrich Paulsen (Erlangen, Germany)
Elke Lütjen-Drecoll (Erlangen, Germany)
martin.schicht@fau.de

Abstract:
The pathogenesis of diabetic retinopathies (DRs), the main cause of blindness in the working population, is still poorly understood. In diabetic renal pathology, the cytokine TGF-β has long been causally implicated. In the eye the results are conflicting. Both decrease and increase in TGF-β are discussed as being causative for the development of DR. In advanced stages of the disease the VEGF is involved in the pathogenesis of DR.

In the present study we investigated expression of TGF-β and VEGF in the anterior and posterior eye segments in diabetic mice with an incipient (10d) and fully developed (20d) diabetes after injection. In previous studies we have shown that TGF-β2 induces increased formation of extracellular material (ECM) in the trabecular meshwork (TM). Therefore, the SC and the TM with their capillaries were examined using qualitative and quantitative ultrastructural methods to clarify whether ECM and vascular changes are present at this early stage of diabetic disease.

The vascular endothelial cells were unchanged. VEGF expression was not changed in the eye segments. We found an increase in expression of TGF-β2 in the anterior eye segment and an increase in ECM in the TM and thickening of the basement membranes of scleral and retinal capillaries. In contrast, in the posterior eye segment there was a decrease in TGF-β2 expression that could explain the conflicting results seen in previous experimental studies.

Further studies are needed to understand why TGF-β2 is expressed differently in the eye segments and which cells are responsible for this.

Poster 3:

Title:
Characterization of the role of plakophilin-1 in the central nervous system of the rat and mouse

Authors:
Leon Winnewisser (Heidelberg, Germany)
leonwnws@web.de

Abstract:
This study involved the electrophysiological characterization of PKP1 knockdown in rat hippocampal neurons and PKP1 knockout in mouse cortical neurons using whole-cell patch-clamp recordings to analyze spontaneous neuronal activity and distinguish between excitatory and inhibitory postsynaptic currents with and without pharmacological agents. Additionally, the subcellular localization of PKP1 was examined by immunofluorescence in primary neuronal cultures and mouse brain sections, including co-localization with known neuronal proteins.

The methods used included whole-cell patch-clamp electrophysiology to analyze spontaneous neuronal activity in primary hippocampal neuron cultures from rats with PKP1 knockdown and in primary cortical neuron cultures from fetal mice with PKP1 knockout. Recordings were performed both with and without pharmacological additives to differentiate between excitatory and inhibitory postsynaptic currents. Additionally, immunofluorescence staining was conducted to examine the subcellular localization of PKP1 in these neuronal cultures as well as in frontal brain sections from mice, including co-localization with established neuronal marker proteins.

The results of this study showed that neither knockdown (KD) of Plakophilin-1 (PKP1) in rat hippocampal neurons nor knockout (KO) in mouse cortical neurons led to significant changes in electrophysiological parameters such as frequency, rise time, decay time, or amplitude of excitatory and inhibitory postsynaptic currents, or action potentials. Immunofluorescence analysis of subcellular localization revealed no clear alterations in PKP1 distribution, likely due to nonspecific antibody binding.

Overall, the findings do not support a direct role of PKP1 in basic neuronal communication in the examined models. Effects in other brain regions or developmental stages cannot be excluded.

Poster 4:

Title:
Role of Complement 1q tumor necrosis factor-related proteins in the Lipid Metabolism of Meibomian Glands

Authors:
Lara Steudte (Erlangen, Germany)
Hagen Nicolaus (Erlangen, Germany)
Antonia Pommer (Erlangen, Germany)
Friedrich Paulsen (Erlangen, Germany)
Fabian Garreis (Erlangen, Germany)
lara.steudte@fau.de

Abstract:
Dry Eye Disease (DED) is a multifactorial ocular surface disorder marked by tear film instability and inflammation. Affecting up to 50% globally, the most common form is evaporative DED, often caused by Meibomian Gland Dysfunction (MGD). MGD disrupts lipid secretion, destabilizing the tear film. PPARγ regulates lipid production in Meibocytes. CTRP proteins may influence lipid metabolism and inflammation via PPARγ. This project will explore their role in Meibogenesis.

Expression of CTRP1, CTRP6, and CTRP8 was examined in immortalized human Meibomian gland epithelial cells (HMGECs). Impact of CTRPs on lipid production and the expression of key lipogenesis markers were studied in HMGECs and organotypic ex vivo slice cultures of murine Meibomian glands. The protein concentration of CTRP1 in the tear fluid of healthy individuals and patients with DED was quantified using an ELISA assay.

RT-PCR analysis detected the gene expression of CTRP1 and CTRP6 in HMGECs. Stimulation with CTRP1 and CTRP6 resulted in increased lipid levels in cultured HMGECs. Similarly, CTRP1 stimulation increased lipid levels in organotypic ex vivo slice cultures of murine Meibomian glands. Additionally, the expression of lipogenesis markers PPARγ, SCD, and FABP4 was modulated by stimulation with analysed CTRPs. ELISA showed significantly lower CTRP1 protein levels in tear fluid of DED patients than healthy individuals.

Our findings reveal the influence of the investigated CTRPs on lipogenesis in the Meibomian gland. This provides a basis for further studies on the physiology of tear film formation and opens new intervention strategies to treat Meibomian gland dysfunction.

Poster 5:

Title:
Evaluation of the impact of vitamin D on keratinocyte adhesion in the context of pemphigus

Authors:
Thomas Schmitt (München, Germany)
Jens Waschke (München, Germany)
thomas.schmitt@web.de

Abstract:
The skin disease pemphigus is induced by autoantibodies targeting desmoglein (Dsg) 1 and 3, adhesive cadherins of desmosomes. The resulting loss of cellular cohesion results in the formation of life-threatening mucosal and epidermal erosion. Current immunosuppressive treatments have limitations, emphasizing the need for alternative approaches. Vitamin D3 (VD3), known to modulate immune and epithelial functions, may offer protective effects. Intriguingly, in the sera of pemphigus patients lower VD3 levels and anti-VD3-IgGs were detected suggesting that VD3 may play a role in the pathogenesis of pemphigus.

Dispase-based dissociation assays, confocal immune-fluorescence microscopy and Western blotting of triton fractionated lysates.

Both physiological (0.12 nmol/l) and high (120 nmol/l) concentrations of 1,25-VD3 and 24,25-VD3 preserved keratinocyte cohesion in the presence of pemphigus autoantibodies, comparable to inhibition of p38MAPK. VD3 prevented desmoglein-1/3 fragmentation and keratin retraction. Interestingly, 1,25-VD3 increased ERK1/2 and p38MAPK phosphorylation. These findings contrast with earlier studies, which showed that active p38MAPK and ERK are involved in loss of cell adhesion in pemphigus adhesion. However, p38MAPK was redistributed from the cytoskeletal to the soluble (cytosolic) fraction, suggesting a possible protective mechanism based on re-localization of the kinase.

VD3 was identified to stabilize keratinocyte adhesion against pathogenic effects of pemphigus autoantibodies and thus to have effects beyond its known immune-modulatory functions. These findings offer new insights into desmosomal regulation and indicate VD3 as a potentially promising adjunctive therapy with likely high patient acceptance rates and fewer side effects.

Poster 6:

Title:
Molecules modulating CRE-dependent transcriptional activity in immortalized neuronal cells from the mouse suprachiasmatic nucleus: PDE inhibitors and autophagy modulators.

Authors:
Erik Maronde (Frankfurt, Deutschland)
Abdelhaq Rami (Frankfurt, Germany)
e.maronde@em.uni-frankfurt.de

Abstract:
We sought to characterize the transcriptional response to different pharmacologically active agents in immortalized neuronal cells derived from the nucleus suprachiasmaticus (SCN) of the mouse hypothalamus.

We used immortalized neuronal cell from the SCN carrying a stably-transfected cyclic-nucleotide-responsive-element (CRE)-luciferase construct (SCNCRE) to characterize the transcriptional response mediated by different agents structurally suggestive of cyclic nucleotide phosphodiesterase (PDE) inhibition and other signaling pathways modulating CRE-binding protein (CREB) activity.

Testing of the non-selective PDE inhibitor isobutyl-methyl-xanthine (IBMX) and selective PDE inhibitors revealed a dominant presence of PDE3, but also weaker activity of inhibitors selective for PDE4 and PDE5 in SCNCRE cells. Other nucleotide-based pharmacologically active agents with an influence on autophagy, especially the widely used autophagy inhibitor 3-methyl-adenine also increased CRE-dependent transcriptional activity in SCNCRE cells dose-dependently.

Experiments provide evidence that, among the various known extracellular signaling pathways components involved, cyclic nucleotide metabolism enzymes, especially phosphodiesterases, or molecule/agents influencing processes like autophagy or inflammation modulate transcriptional CRE-dependent activity in SCNCRE cells. The autophagy inhibitor 3-MA in particular might on top of its well-described inhibition of PI3Kinase class 3, also efficiently inhibit PDE activity. This may significantly contribute to its effect on autophagy in cells and tissues where PDE3 is strongly expressed.

Poster 7:

Poster wurde zurückgezogen

Poster 8:

Title:
Desmosomal hyper-adhesion changes desmosome morphology and desmoglein 3 binding properties within desmosomes

Authors:
Mariia Klimkina (Munich, Germany)
Matthias Hiermaier (Munich, Germany)
Michael Fuchs (Munich, Germany)
Jens Waschke (Munich, Germany)
Franziska Hartig-Vielmuth (Munich, Germany)
m.klimkina@campus.lmu.de

Abstract:
Desmosomes are multiprotein complexes that mediate strong intercellular adhesion in tissues such as the skin, which are continuously exposed to mechanical stress. Desmosomes consist of desmosomal cadherins which are intracellular anchored via plaque proteins, among them desmoplakin (DP), to the keratin intermediate filament cytoskeleton. During differentiation, keratinocytes develop a strong adhesive state in which desmosomal cadherins maintain adhesive strength independently of calcium ion. This condition, referred to as hyper-adhesion, is concisely regulated. Here, we investigate the effects of hyper-adhesion on morphology of desmosomes and desmosomal cadherin binding properties within desmosomes.

Keratinocyte dissociation assay, Immunofluorescence (IF), STED, Western blot, Atomic force microscopy (AFM)

Murine keratinocytes with eYPF-tagged DP develop a Calcium-independent, hyper-adhesive state within 48h of Ca2+-induced differentiation. Acquisition of hyper-adhesion is accompanied with changes in the DP plaque morphology with reduced distance between Dp plaques and increased membrane coverage in live cell STED. In addition, keratin filaments also undergo structural changes during differentiation with a refined and condensed mesh of filaments under hyper-adhesive condition.

Combination of STED imaging and AFM force spectroscopy allow measurement of Dsg3 binding properties within two DP plaques on living murine keratinocytes. Dsg3 adhesion measurements revealed no difference in Dsg3 binding frequency in hyper-adhesive and non-hyper-adhesive state. In contrast, Dsg3 single molecule binding strength was significantly higher under hyper-adhesive conditions and step position reduced within two DP plaques, suggesting that Dsg3 binding properties drastically change during acquisition of hyper-adhesiveness within desmosomes.

Morphology of desmosomes as well as desmosomal cadherin binding properties change during acquisition of hyper-adhesion.

Poster 9:

Title:
Therapeutic Potential of CXCR4 Receptor Targeting in Retinoblastoma

Authors:
Elias Joschua Acker (Essen, Germany)
André Haase (Essen, Germany)
Mirja Harms (Ulm, Germany)
Emily Alefeld (Essen, Germany)
Nicole Dünker (Essen, Germany)
Maike Busch (Essen, Germany)
elias.acker@stud.uni-due.de

Abstract:
Retinoblastoma (RB) is the most common intraocular malignancy in early childhood. While current treatments, including enucleation and eye-preserving chemotherapy, yield high survival rates, challenges such as vitreous seeding, chemoresistance, and secondary malignancies - especially in aggressive subtype 2 tumors -remain. The CXCR4/CXCL12 axis, implicated in tumor progression and metastasis, is dysregulated in various cancers and inflammatory conditions. As the CXCR4 receptor is expressed in RB, it represents a potential therapeutic target. This study evaluates the antitumor efficacy of novel EPI-X4-based CXCR4 antagonists in vitro and in ovo.

We assessed RB cell viability, proliferation, and cell death following treatment with CXCR4 antagonists alone, combined with chemotherapy, or in co-culture with RB stromal cells. In vivo treatment efficacy was examined using the in ovo chorioallantoic membrane (CAM) assay. Western blot analysis was used to detect CXCR4 receptor and CXCL12 ligand expression in RB tumor and stromal cells.

CXCR4 antagonists significantly reduced viability and proliferation while inducing cell death in RB cell lines. In CAM assays, CXCR4 inhibitor treatment significantly reduced tumor size and weight, particularly in chemoresistant RB cells. Stromal cell conditioned medium did not alter the efficacy of CXCR4 inhibition in vitro. Ongoing combination experiments with melphalan or topotecan chemotherapeutics aim at analyzing a potential enhancement of antitumor effects.

CXCR4 represents a promising therapeutic target in RB. Its inhibition reduces tumor viability and growth of chemoresistant RB cells. Further studies are under way to elucidate the impact of tumor microenvironment and downstream signaling, aiming to optimize targeted RB treatment strategies.

Poster 10:

Title:
Influence of environmental toxins like dioxins and polycyclic aromatic hydrocarbons on human meibomian glands

Authors:
Ingrid Zahn (Erlangen, Germany)
Hannah Franke (Erlangen, DE)
Antonio Bergua (Erlangen, Germany)
Friedrich Paulsen (Erlangen, Germany)
ingrid.zahn@fau.de

Abstract:
The meibomian glands producing meibum, which forms the outermost, protective lipid layer of the tear film. Their dysfunction is a key factor in the development of dry eye disease, a condition with few treatment options. Polycyclic aromatic hydrocarbons (PAHs) and dioxins are lipophilic environmental toxins that accumulate in fatty tissues and are therefore likely to accumulate in the lipid-rich meibomian glands. Due to their persistenceand bioaccumulation effects, these compounds may impair meibomian gland function.

We analyzed meibomian glands from six body donors for polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs), dioxin-like PCBs (dl-PCBs), non-dioxin-like PCBs (ndl-PCBs), and PAHs using high-resolution GC-MS/MS. In parallel, the immortalized human meibomian gland epithelial cell line (ihMGECs) was exposed to 0.0001–10 nM 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and 0.1–1000 µM benzo[a]pyrene (B[a]P), and evaluated for viability, cell proliferation, migration, and lipid production.

In human meibomian glands, total toxic equivalents (TEQ) of PCDD/Fs and dl-PCBs were 22.04 ± 6.23 and 24.55 ± 6.10 ng/kg-fat, respectively. The sum of ndl-PCBs was 961 ± 290.01 µg/kg-fat. Stimulation of differentiated ihMGECs did not affect cell-viability but led to a significant, dose-independent reduction in lipid production. In proliferating ihMGECs, both toxins caused a dose-independent decrease in cell proliferation. Additionally, B[a]P, but not TCDD, significantly impaired cell migration.

These findings suggest that lipophilic environmental toxins such as PCBs and dioxins accumulate in human meibomian glands at levels indicating a significant toxic burden. Exposure to TCDD and B[a]P impaired lipid production as well as proliferation and migration of ihMGECs, indicating a potential role in meibomian gland dysfunction.

Poster 11:

Title:
Influence of lacrimal gland acinar cells (lacrimal gland epithelial cell line) on human B cells with regard to plasma cell differentiation.

Authors:
Nils Fleischmann (Erlangen, Germany)
Friedrich Paulsen (Erlangen, Germany)
Wolfgang Schuh (Erlangen, Germany)
Benjamin Frey (Erlangen, Germany)
nils.fleischmann@fau.de

Abstract:
Primary Sjögren's syndrome (pSS) is a chronic autoimmune disease in which exocrine glands such as the lacrimal gland are damaged by lymphocytic infiltration, leading to dry eye disease. There is evidence that lacrimal gland epithelial cells (LGECs) are not only the target of autoimmune processes but may also actively contribute to the pathology. The aim of this study was to investigate the influence of LGECs on B cell activation, differentiation and survival under physiological conditions. For this purpose, a newly developed immortalized human lacrimal gland epithelial cell line (ihGLC) was used.

RT-PCR was performed to test whether ihGLC express relevant genes for the activation and differentiation of B cells. Co-cultures were then performed with human CD19-positive selected B cells and the viability, activation and differentiation of the B cells were determined by flow cytometry.

The cytokines IL-4, IL-21, TGFβ-1 and chemokines such as CCL25 or CXCL12, the B-cell activation factor (BAFF) and A proliferation-inducing ligand (APRIL) could all be detected with RT-PCR in the ihGLC. B cells co-cultured with ihGLC cells showed increased viability compared to the control group, but did not show activation or differentiation into plasma cells.

The results indicate that ihGLCs can create a survival environment for B cells without triggering their activation, at least under non-inflammatory conditions. With the established cell line, it will be possible in the future to specifically simulate B cell activation and differentiation under inflammatory conditions such as pSS and to further investigate the interaction between LGECs and immune cells.

Poster 12:

Title:
The spectrin cytoskeleton contributes to dendritic morphology in a sensory neuron of C. elegans

Authors:
Anna Melchers (Tübingen, Germany)
Oliver Vinzenz Glomb (Tübingen, Germany)
anna.melchers@student.uni-tuebingen.de

Abstract:
Loss of function mutations in ßIII-spectrin are associated with spinocerebellar ataxia type V. Recent work on a ßIII-spectrin mutant rodent model suggests that its pathology is caused by a malformation of the dendritic network in Purkinje cells. In dendrites, ßIII-spectrin forms a highly specialized cytoskeletal network but how this network organizes the formation of highly ordered dendrites remains poorly understood. In this study, we establish a model to study the molecular mechanisms underlying spectrins role in dendrite formation using a highly arborized mechanosensory neuron within a living nematode.

We combined live cell microscopy on endogenously labeled components of the spectrin cytoskeleton with genetic perturbation experiments during the development of the dendritic network.

Spectrin is highly abundant within higher ordered dendrites early during dendritic development. Loss of spectrin or its associated proteins resulted in a specific reduction of higher ordered dendrites whereas primary dendrite formation remained unaffected.

Our results indicate a specific role for the spectrin cytoskeleton in the formation of higher ordered dendrites.

Poster 13:

Title:
Mechanisms Underlying the Axonal Distribution of Ca2+/Calmodulin-dependent protein kinase II/UNC-43 in Caenorhabditis elegans.

Authors:
Aakanksha Dharmapuri (Tübingen, Germany)
Oliver Glomb (Tübingen, Germany)
aakankshad2121@gmail.com

Abstract:
Calcium/Calmodulin-dependent protein kinase II (CaMKII), a soluble cytosolic protein, plays a critical role in the regulation of synaptic plasticity and memory. Disturbance in these pathways forms a basis for many neuropsychiatric disorders. Despite its significance, it remains unclear how CAMKII is delivered to the neurite. We addressed this knowledge gap using Caenorhabditis elegans as a model organism to dive deep into the transport machinery of UNC-43/CAMKII in a bipolar motor neuron.

Using cell specific degradation and endogenous fluorescent labelling approaches, we investigated the role of kinesin motor proteins on CaMKII/UNC-43's distribution and transport dynamics.

We observed that CaMKII/UNC-43 delivery to the axon depends on KIF1A/kinesin-3/unc-104. We are currently performing a small candidate screen to decipher how KIF1A distributes CAMKII/UNC-43.

Our preliminary results suggest the potential involvement of the KIF1A/kinesin-3/unc-104 motor protein in the transport of CaMKII/UNC-43.

Poster 14:

Title:
A mutation in the binding pocket of desmogleins impact DSG2-DSG3 adhesive properties and desmosome signaling in keratinocytes

Authors:
Leoni Johanna Vogel (Munich, Deutschland)
Lara Pavelko (Munich, Deutschland)
Volker Spindler (Hamburg, Germany)
Jens Waschke (Munich, Germany)
Matthias Hiermaier (Munich, Germany)
Franziska Vielmuth (Munich, Germany)
leoni.vogel@campus.lmu.de

Abstract:
Pemphigus vulgaris (PV) is an autoimmune blistering disorder characterized by autoantibodies primarily against desmosomal cadherins Desmoglein (DSG) 1 and DSG3, leading to loss of intercellular adhesion in keratinocytes. Interestingly, an upregulation of DSG2 has been observed in pemphigus patients' skin, suggesting a potential compensatory role via heterophilic interactions with DSG3. To investigate the functional relevance of DSG2 and DSG3 crosstalk in cell-cell adhesion in pemphigus, we here compared keratinocytes expressing either wild-type desmogleins or a W2A point mutant which was suggested to abolish DSG binding ability.

Keratinocyte dissociation assay, atomic force microscopy (AFM), Western blot

Membrane recruitment of DSG3 was not changed in DSG3-W2A and DSG2-W2A keratinocytes. In contrast, DSG2 was upregulated in DSG3-W2A but not in DSG2-W2A keratinocytes, showing an impact of W2A-mutation for the crosstalk of DSG2/DSG3 expression. In addition, pamgene analysis revealed altered kinomics in DSG2-W2A keratinocytes. Dissociation assays showed a significantly increased fragmentation in the DSG2-W2A cell line compared to wild-type. AK23 and PV-IgG led to an additional loss of intercellular adhesion in both cell lines. In contrast, acquisition of desmosomal hyper-adhesion was unaltered in DSG2-W2A keratinocytes, indicating that junctional maturation remains unaffected. AFM measurements showed an increased step position of DSG2-mediated interactions in DSG2-W2A cells, indicating that DSG2 binding partners in the DSG2-W2A mutant cells have a weaker anchorage to the cytoskeleton.

The DSG2-W2A mutation compromised intercellular adhesion, altered intracellular signaling and DSG2 binding properties, showing an important role for the DSG2-DSG3 crosstalk in keratinocytes.

Poster 15:

Title:
Role of desmoplakin in mediating heterotypic cellular interactions in melanoma pathogenesis

Authors:
Maike Märker (Hamburg, Germany)
Ramon Castellanos Martinez (Basel, Switzerland)
Jennifer Schröder-Schwarz (Hamburg, Germany)
Volker Spindler (Hamburg, Germany)
Maitreyi Rathod (Hamburg, Germany)
m.rathod@uke.de

Abstract:
Desmosomes are key in regulating tissue integrity, specifically in tissues undergoing pronounced mechanical challenges. Alterations in desmosome proteins lead to multiple diseases. Mutations in desmosome proteins is reported two thirds of skin cutaneous melanoma. However, it is largely unclear, how desmosomes – and in particular the key molecule desmoplakin (DSP) influence melanoma pathogenesis

A point mutant (DSPT576I) in mice and keratinocytes was used. Mouse tissue analysis by histology and Fontana Masson staining to analyze melanin pigmentation in mouse skin was applied. EdU-Click chemistry was used to quantify cellular proliferation. Cytokine array was performed to identify differentially secreted cytokines from keratinocytes.

Homozygous DSP mutants show differentiation defects in the skin with thickening of epidermis. The increased epidermal thickness in mutant mice potentially results from enhanced proliferation in the basal layer of tail skin. In line with these observations, intriguingly, the DSP mutant mice show darker tails, and enhanced melanin pigmentation in the skin similar to a tanning response. This correlated with a significant increase in the number of melanocytes. As desmosomes do not form connections between keratinocytes and melanocytes, we tested paracrine signaling responses. Conditioned media (CM) from DSP mutant keratinocytes increased the proliferation and invasion of melanoma cells. Mechanistically IL-23 and GM-CSF were identified as potential paracrine mediators.

A single point mutation of DSP, leading to loss of function, influences melanocyte homeostasis and may promote melanoma states.

Poster 16:

Title:
Not a simple conduit - Regional differences in cGMP-mediated relaxation along the aorta

Authors:
Andrea Mietens (Giessen, Germany)
Jin Cheng (Giessen, Germany)
Sabine Tasch (Giessen, Germany)
Ralf Middendorff (Giessen, Germany)
andrea.mietens@anatomie.med.uni-giessen.de

Abstract:
Recently acknowledged as an individual organ, the aorta is not merely a conduit vessel, but mediates coupling of the heart to the peripheral circulation. The proximal Windkessel function, usually attributed to elastic fibres within the vessel wall, transforms pulsatile blood flow received from the heart into a more continuous flow. However, aortic function along its length may differ and the contribution of vascular smooth muscle cells (vSMCs) remains underestimated. We investigated relaxing cGMP pathways activated by either atrial natriuretic peptide (ANP) or nitric oxide (NO) in proximal and distal aorta.

To assess potential regional differences of aortic contractile function, ring preparations of proximal and distal aorta from the same Wistar rats were mounted in an organ bath. We compared how pre-incubation with ANP or NO, respectively, affected noradrenaline(NA)-induced vasoconstriction in proximal vs. distal aorta.

Vasoconstriction in response to NA did not differ in proximal vs. distal aorta. Pre-incubation with NO relaxed aortic rings and shifted the dose-response curve for NA to the right with a more pronounced effect in distal aorta. ANP also reduced NA-induced contractility in proximal and distal aorta with a comparable effect size in both locations. ANP was the more effective agent in proximal aorta whereas distally, ANP and NO effects were similar.

Our findings illustrate that cGMP signals along the aorta are not the same, but are differentially regulated in a region-specific manner. Aortic regional dysfunction may impact the adjacent heart and peripheral vasculature thus contributing to cardiovascular disease, but may also enable region-specific therapeutic concepts.

Poster 17:

Title:
The role of CEACAM1 in the healthy eye and in choroidal neovascularization

Authors:
Laura Hannig (Hamburg, Germany)
Nikolai Kleefeldt (Würzburg, Germany)
Lea Cagol (Würzburg, Germany)
Jonathan Müller (Würzburg, Germany)
Mario Vallon (Würzburg, Germany)
Jost Hillenkamp (Würzburg, Germany)
Süleyman Ergün (Würzburg, Germany)
Barbara M. Braunger (Hamburg, Germany)
Anja Schlecht (Hamburg, Germany)
l.hannig@uke.de

Abstract:
Choroidal neovascularization (CNV) is the main feature of age-related macular degeneration (AMD). Despite extensive research the molecular mediators contributing to CNV formation are not fully understood. Here, we investigated if CEACAM-1 (CC1) contributes to CNV formation.

CEACAM-1 expression in the eyes of Cc1-/- and wildtype littermates was characterized using immunohistochemistry and flow cytometry. Retinal and choroidal morphology was analyzed by light microscopy and immunohistochemistry. FITC-dextran perfusion and in vivo imaging was used to investigate the retinal vasculature. Laser-induced CNV were analyzed regarding lesion size and number of accumulating myeloid cells in choroidal flatmounts.

A robust CEACAM-1 expression was determined in endothelial and myeloid cells of retina and choroid in the healthy eye. Deletion of CEACAM1 did not result in morphological changes in retina and choroid. Retinal vascular plexus were evenly formed and distributed and myeloid cells did not differ in cell numbers or morphology. However, following laser-induced CNV-formation, Cc1-/- mice revealed significantly smaller CNV areas concomitant with significantly less accumulating myeloid cells compared to wildtype littermates.

CEACAM-1 is expressed in endothelial and myeloid cells of retina and choroid under physiological conditions. Its deletion does not affect retinal and choroidal morphology. However, during CNV development, deletion of CC1 results in reduced lesion size and accumulation of myeloid cells, suggesting that CC1 might contribute to age-related macular degeneration.

Poster 18:

Title:
The Otsuka Long-Evans Tokushima Fatty rat diabetes model in view of joint cartilage research

Authors:
Gundula Schulze-Tanzil (Nürnberg, Deutschland)
Maria Kokozidou (Nürnberg, Deutschland)
Tobias Braun (Nürnberg, Deutschland)
Patricia Drummond (Nürnberg, Deutschland)
Lukas Motloch (Bad Ischl, Österreich)
Christian Werner (Nürnberg, Deutschland)
Ingo Mrosewski (Berlin, Deutschland)
Vera Paar (Salzburg, Österreich)
gundula.schulze@pmu.ac.at

Abstract:
Type 2 diabetes mellitus (T2DM) is associated with disorders like neuro-, angio-, and nephropathies and steatohepatosis, but it also predisposes for musculoskeletal impairments. These features are also expected in rat models of T2DM. The Otsuka Long-Evans Tokushima Fatty (OLETF) rat is a typical genetic T2DM model reflecting many associated disorders. However, detailed data concerning joint cartilage alterations in the OLETF rat model is lacking. Hence, this study aimed to provide insights into the histopathology of crucial organs (pancreas, kidney, liver and spleen) and knee joint cartilage of non diabetic LE rats (controls) and OLETF rats with T2DM.

A total of 23 male Long-Evans (LE, controls) and 11 male OLETF rats of similar age (both with 36- and 44-week-old subgroups) served as tissue donors. Pancreas, kidney, liver, spleen and knee joint cartilage were macroscopically and histopathologically analysed and suitable scoring systems were established.

No changes in organ sizes were detected, but typical features of endocrinic and exocrinic pancreas degeneration (fibrosis, particularly of the Langerhans islets), nephropathy, steatohepatosis and mild spleen alterations (e.g., disintegration of follicle architecture) could be shown in OLETF rats. In addition, distinct signs of joint cartilage degeneration could be detected in OLETF rats (cysts or clefts). The established scoring systems for organ degeneration were suitable to summarize crucial histopathological features observed in pancreas, kidney, liver and spleen of the rats.

These scores might be useful for standard histopathology in other rat models and the OLETF model could be used to study the interrelation of T2DM and osteoarthritis (OA).

Poster 19:

Title:
Lipocalin-2 Modulates TNFα-Induced Effects in Primary Human Astrocytes: An In Vitro Study

Authors:
Fabio Zillmer (Bonn, Deutschland)
Rittika Chunder (Bonn, Germany)
Franziska Pfeiffer (Bonn, Germany)
Natalie Tillmann (Bonn, Germany)
Tim Clarner (Bonn, Germany)
s4fazill@uni-bonn.de

Abstract:
Multiple sclerosis (MS) is a chronic inflammatory disease of the central nervous system (CNS), characterized by demyelination and blood-brain barrier (BBB) dysfunction. Astrocytes, key regulators of BBB homeostasis, may contribute to either stabilization or breakdown of the BBB during neuroinflammation, depending on their activation state. Lipocalin-2 (LCN2), a small protein secreted by astrocytes, is elevated in the cerebrospinal fluid of MS patients. However, its functional role in the complex cell-cell interactions during neuroinflammation remains unclear. This study investigates the effect of LCN2 under pro-inflammatory conditions using a novel in vitro system of primary adult human astrocytes.

Primary astrocytes were isolated from adult human brain tissue using magnetic-activated cell sorting (MACS) and characterized by GFAP and GLAST expression. Expression of the LCN2 receptor 24p3R was confirmed by immunocytochemistry and western blot. Cells were treated with TNFα (1 ng/ml), LCN2 (100 ng/ml), or both. Morphological changes were assessed by Phalloidin staining and cell circularity analysis. Furthermore, mRNA next-generation sequencing was performed to identify LCN2-modulated genes under inflammatory conditions.

24p3R expression was detected on adult human astrocytes. TNFα induced significant morphological changes, notably reduced cell circularity. Co-treatment with LCN2 partially reversed these effects. Transcriptomic profiling revealed LCN2 as a selective regulator of gene networks involved in astrocytic inflammatory responses. The functional roles of these genes in neuroinflammation remain to be clarified.

Adult human astrocytes respond to inflammatory stimuli with morphological and transcriptional changes. LCN2 modulates these responses, suggesting a regulatory role in astrocyte-driven inflammation relevant to neuroinflammatory diseases such as MS.

Poster 20:

Title:
Ultrastructural characterization of Cardiomyocyte Subtypes in Arrhythmogenic Cardiomyopathy

Authors:
Franziska Matthias (Hannover, Germany)
Susanne Faßbender (Hannover, Germany)
Andrea Kofink-Germershausen (Hannover, Germany)
Julia Schipke (Hannover, Germany)
Ramon Castellanos-Martinez (Basel, Switzerland)
Maximilian Lenz (Hannover, Germany)
Christian Mühlfeld (Hannover, Germany)
Jan Hegermann (Hannover, Germany)
Camilla Schinner (Hamburg, Germany)
matthias.franziska@mh-hannover.de

Abstract:
Arrhythmogenic cardiomyopathy (ACM) is a genetic disease characterized by myocardial fibrosis, inflammation, and arrhythmia leading to sudden cardiac death and heart failure. This study aimed to develop a comprehensive imaging approach to characterize distinct cardiomyocyte subtypes that emerge during disease progression

Hearts from the inducible ACM mouse model iDsg2-W2A were collected at defined disease stages 5 to 130 days post disease induction. We established an FFPE-based immunostaining and semi-automated image analysis workflow using QuPath for whole-section assessment. A cardiomyocyte detection algorithm was established based on membrane staining patterns. As subtype markers, Ki67 and PCNA, were used to detect proliferating cardiomyocytes, while α-smooth muscle actin marked dedifferentiating cells. These subtypes were analyzed regarding their time point of appearance, frequency and spatial distribution within the diseased heart. A correlative transmission electron microscopy protocol was established employing immunostained vibratome sections to assess ultrastructural features of these subpopulations.

We observed a peak of proliferating cardiomyocytes prior to development of cardiac fibrosis 10 days after induction. This response was evident in both ventricles, indicating a transient but spatially widespread activation of cardiomyocyte cycling. Ultrastructural analysis of α-smooth muscle actin-positive cardiomyocytes revealed sarcomeric disarray and pericellular collagen deposition, consistent with early pathological cardiomyocyte remodeling.

We established new tools to analyze cardiomyocyte subtypes on histological and ultrastructural level. Our integrated approach provides insights into pathological remodeling in ACM by characterization of cardiomyocyte subtypes during disease progression. These findings enhance our understanding of ACM pathogenesis and may guide the development of future therapeutic strategies.

Poster 21:

Title:
A pathological in-vitro model of the inflamed lacrimal gland using immortalized human lacrimal gland cells (ihGLC)

Authors:
Lara Furch (Erlangen, Germany)
Friedrich Paulsen (Erlangen, Germany)
Philipp Arnold (Erlangen, Germany)
Sophie Gleixner (Erlangen and Hannover, Germany)
s.gx@gmx.net

Abstract:
Aqueous-deficient dry eye (ADDE) is caused by inflammation and subsequent fibrotic remodeling of the lacrimal gland, resulting in its dysfunction. Moreover, research has demonstrated a correlation between chronic inflammatory bowel disease and dry eye disease. This project provides a pathological model of the lacrimal gland in order to investigate the underlying mechanisms of ADDE, thereby providing insights into the gut-eye or gut-lacrimal gland axis, respectively.

ihGLC were cultured in 2D and 3D and examined after an inflammatory response was triggered. In 2D, the cells were stimulated with lipopolysaccharide (LPS) and outer membrane vesicles (OMVs) from the bacterial strains E. coli BL21 and Clearcoli. The expression of various genes was analyzed using RT-PCR. After 24 hours of stimulation with LPS, the 3D cultured spheroids were stained using HE, Azan, PAS, Alcian blue and immunofluorescence and by transmission electron microscopy (TEM). Total mRNA sequencing of lacrimal gland spheroids stimulated with LPS was also performed.

RNA-Seq analyses show increased expression of genes belonging to the chemokine CXC subfamily. TEM and histological examinations revealed changes in the marginal cells of the spheroids and in the production of secretory vesicles. ihGLC respond to stimulation with LPS or E. coli OMVs with an inflammatory reaction.

This study presents a simple method for creating a pathological 3D in vitro model using the human lacrimal gland cell line ihGLC. Simulating pathological states of the lacrimal gland in spheroids represents a valuable tool for understanding processes in an inflamed human lacrimal gland and facilitating drug testing.

Poster 22:

Title:
Ogremorphin inhibits proliferation and reduces viability in human pancreatic cancer cell models

Authors:
Moritz Schameitat (Halle (Saale), Germany)
William-Alexander Ziem (Halle (Saale), Germany)
Kristin Wächter (Halle (Saale), Germany)
Johannes Kipp (Halle (Saale), Germany)
Heike Kielstein (Halle (Saale), Germany)
Michael Boettcher (Halle (Saale), Germany)
Sonja M. Keßler (Halle (Saale), Germany)
Ivonne Bazwinsky-Wutschke (Halle (Saale), Germany)
moritz.schameitat@uk-halle.de

Abstract:
Ogremorphin (OGM) is a novel synthetic compound with potential therapeutic effects on various tumor entities, due to its specific inhibition of G protein-coupled receptor 68 (GPR68) and activating ferroptosis, a specific form of cell death. GPR68 regulates cellular pH homeostasis, a critical factor enabling tumor cells to shape their microenvironment, thereby contributing to therapy resistance. Given the limited recent research in this area, we aimed to investigate the potential of OGM for treating pancreatic ductal adenocarcinoma (PDAC), a highly aggressive and therapy-resistant cancer type.

We applied OGM to various PDAC cell culture models, including multiple established cell lines (SU.86.86, Mia PaCa-2, PANC-1) and spheroid cultures. To assess cell proliferation and viability, we used live-cell imaging, luminescence viability assays, and resazurin assays. Gene expression and signaling pathway changes were analyzed by microarray transcriptome profiling and qPCR. GSH- metabolism was examined by measuring reactive oxygen species (ROS) levels.

All investigated PDAC cell lines expressed GPR68. OGM treatment significantly reduced cell proliferation and viability in both 2D and 3D cultures, with complete cell death observed at higher concentrations. OGM modulated key genes associated with ferroptosis, and treated cells showed increased ROS accumulation, consistent with the activation of ferroptosis.

In summary, OGM induced significant transcriptomic changes, inhibited proliferation and reduced viability in diverse human PDAC cell models, suggesting activation of ferroptosis. These findings indicate that OGM is a promising candidate for further preclinical evaluation as a potential therapeutic agent for PDAC.

Poster 23:

Title:
Characterization of DMBT1 (Deleted in malignant brain tumors 1 protein) in the ocular surface system and its potential application for artificial tears

Authors:
Zhaoxi Zhang (Erlangen, Germany)
Ingrid Zahn (Erlangen, Germany)
Nils Fleischmann (Erlangen, Germany)
Martin Schicht (Erlangen, Germany)
Philipp Arnold (Erlangen, Germany)
Friedrich Paulsen (Erlangen, Germany)
zhaoxi.zhang@fau.de

Abstract:
Deleted in malignant brain tumor-1 (DMBT1) is dysregulated in dry eye disease, a growing public health problem. On the ocular surface, DMBT1 may interact with tear film proteins to support mucosal defense and contribute to the structural stability of the tear film. However, the distribution patterns and relative concentrations of DMBT1 in ocular surface tissues have not yet been systematically investigated. Therefore, our study focused on confirming the presence of DMBT1 in these ocular components.

To investigate the distribution and relative abundance of DMBT1 in human ocular tissues, RT-PCR, immunofluorescence, immunohistochemistry and ELISA analyses were performed on human tissue samples from the conjunctiva, lacrimal gland, cornea, eyelid and lacrimal drainage system as well as on epithelial cell lines derived from human ocular tissues.

Expression of DMBT1 in human tissues and cell lines was confirmed in the ocular system at both gene and protein levels. The relative abundance of DMBT1 normalized to total protein (pg/mg) is 11.31±2.28 in the conjunctiva, 4.24±0.89 in the lacrimal gland, 9.33±1.20 in the eyelid, and 10.33±7.61 in the lacrimal drainage system. Although DMBT1 could not be detected in meibomian gland tissue, it was measured at a low concentration in the corresponding epithelial cell line under in vitro conditions.

DMBT1 is present in ocular tissues and in tear fluid of healthy individuals. Our results support the functional investigation of the role of DMBT1 in the maintenance of ocular surface integrity and its potential importance for the development and formulation of artificial tears.

Poster 24:

Title:
Atoh8 is a regulator of autophagy

Authors:
Hawi Kadr (Bochum, Deutschland)
Satya Srirama Karthik Divvela (Bochum, Deutschland)
Eric Bekoe Offei (Accra, Ghana)
Maximilian Hausherr (Bochum, Deutschland)
Verian Bader (Bochum, Deutschland)
Xuesong Yang (Guangzhou, China)
Holm Zaehres (Bochum, Deutschland)
Konstanze Winklhofer (Bochum, Deutschland)
Katrin Marcus-Alic (Bochum, Deutschland)
Beate Brand-Saberi (Bochum, Deutschland)
hawi.kadr@rub.de

Abstract:
Atoh8, a basic helix-loop-helix (bHLH) transcription factor, is known to influence development, stem cell regulation, and cancer, but the mechanisms behind these roles remain unresolved. Given its widespread expression in both embryonic and adult tissues, this study aimed to determine whether Atoh8 governs essential cellular processes such as metabolism and autophagy, potentially explaining its broad biological impact.

To assess Atoh8's role in autophagy, we used knockdown, knockout and overexpression approaches in various cell lines and in vivo mouse models. Autophagic activity was evaluated by analysing markers such as LC3B-II, TFEB, and p62 through immunoblotting. Additionally, mass spectrometry was performed on tibialis anterior muscle tissue from wild-type and Atoh8-deficient mice to identify changes in protein expression related to autophagy.

Loss of Atoh8 led to impaired autophagy, resulting in differential LC3B-II and TFEB levels, accompanied by p62 accumulation. Proteomic analysis revealed a marked downregulation of proteins involved in key autophagic steps, including DEPTOR and PRAS40 (initiation), PDCD6IP and CHMP5 (autophagosome maturation), and NPC2, CTSB, and CTSL (autolysosomal degradation). Furthermore, metabolic pathways were significantly altered in the absence of Atoh8.

Our findings establish Atoh8 as a central regulator of autophagy, influencing its initiation, progression, and completion. The disruption of autophagy and metabolism upon Atoh8 loss may help explain its diverse roles in development and disease, highlighting its potential as a target in autophagy-related conditions.

Poster 25:

Title:
Transcriptional effect of TP53 and RB1 knockout in bladder cancer cell lines KU-19-19 and BC-3C

Authors:
Manuel Becht (Essen, Germany)
Marc Wiesehöfer (Essen, Germany)
Gunther Wennemuth (Essen, Germany)
Manuel.Becht@uk-essen.de

Abstract:
More than 90% of bladder tumors are urothelial carcinomas, classified into non-muscle-invasive (NMIBC) and muscle-invasive bladder cancer (MIBC). MIBC exhibits a markedly worse prognosis, with a 5-year survival rate between 50–10%, largely due to early, undetected metastasis (Hurst et al., 2018). Key genetic alterations during progression from NMIBC to MIBC include frequent co-mutation of the tumor suppressor genes TP53 and RB1 (Nassar et al., 2019), which, although central to disease progression, are not considered suitable for direct targeted therapy. Current first-line treatments for MIBC include cisplatin/gemcitabine chemotherapy, immune checkpoint inhibitors, antibody–drug conjugates, and radical cystectomy in non-metastatic cases (Lopez-Beltran et al., 2024).

This study investigates the functional role of TP53 and RB1 in bladder cancer progression.

Using CRISPR/Cas9, single and combined gene deletions were introduced into urothelial carcinoma cell lines.

Transcriptomic profiling via RNA sequencing revealed differential expression in pathways related to epithelial–mesenchymal transition, cell cycle regulation, and epigenetic modifications. Preliminary growth assays demonstrated a shortened doubling time in double knockout cells, indicating impaired proliferation.

Our findings highlight the central role of TP53 and RB1 in maintaining critical cellular functions and suggest their combined loss contributes to invasive phenotypes. Ongoing work focuses on identifying druggable proteins in these models, with further functional characterization via apoptosis, migration, and invasion assays, as well as in vivo studies using the CAM model.

Poster 26:

Title:
Lipid Remodeling and Organelle Alterations in CEACAM1-Deficient Bone Marrow-Derived Macrophages (BMDMs)

Authors:
Anggi Muhtar Pratama (Würzburg, Indonesia)
Heike Bömmel (Würzburg, Germany)
Hevi Wihadmadyatami (Yogyakarta, Indonesia)
Süleyman Ergün (Würzburg, Germany)
Karnati Srikanth (Würzburg, Germany)
anggi-muhtar.pratama@uni-wuerzburg.de

Abstract:
CEACAM1 is a transmembrane glycoprotein abundantly expressed in macrophages. It plays a pivotal role in cellular integrity, immune responses, and cell proliferation. Emerging evidence suggests that CEACAM1 deletion profoundly affects lipid metabolism. This study aims to characterize the lipidomic profile of CEACAM1-deficient BMDMs to gain insights into lipid-mediated mechanisms affected by CEACAM1 loss.

BMDMs were generated from the bone marrow of five CEACAM1 knockout and five wild-type mice by culturing in M-CSF-containing medium for seven days. Lipidomic profiling was performed using an integrated electrospray ionization tandem mass spectrometry (ESI-MS/MS) approach for quantitative analysis of lipid classes and species.

A total of 18 lipid classes comprising 171 individual lipid species were quantified. CEACAM1-deficient BMDMs showed significant reductions in multiple lipid species, including Phosphatidylcholine (PC 32:0), Lysophosphatidylcholine (LPC 16:0, LPC 20:4), Phosphatidylethanolamine (PE 38:4), Lysophosphatidylethanolamine (LPE 20:4), Ether-linked phosphatidylcholine (PCO 36:4, PCO 38:4), plasmalogen-type PE (PEP 38:4), Phosphatidylglycerol (PG 34:1), Sphingomyelin (SM 34:1;O2), Ceramide (Cer 18:1;O2/16:0), Hexosylceramide (HexCer 18:1;O2/16:0), and Diglycerides (DG 38:4). In contrast, levels of Phosphatidylinositol (PI 38:4) and Cholesteryl Esters (CE 16:0, CE 18:1) were significantly elevated. These lipid alterations were accompanied by reduced peroxisomal expression and mitochondrial abnormalities—both organelles central to lipid metabolism. The elevated levels of PI and CE may reflect compensatory mechanisms supporting cellular growth, motility, and signaling in CEACAM1-deficient macrophages.

CEACAM1 deficiency leads to significant dysregulation of lipid metabolism in BMDMs. This lipidomic shift may contribute to altered macrophage function and provides a foundation for further mechanistic studies on CEACAM1’s role in immune cell biology.

Poster 27:

Title:
CEACAM1 Deletion Impairs Peroxisomal Biogenesis and Alters Mitochondrial Morphology in BMDMs

Authors:
Anggi Muhtar Pratama (Würzburg, Indonesia)
Heike Bömmel (Würzburg, Germany)
Hevi Wihadmadyatami (Yogyakarta, Indonesia)
Süleyman Ergün (Würzburg, Germany)
Srikanth Karnati (Würzburg, Germany)
anggi-muhtar.pratama@uni-wuerzburg.de

Abstract:
The transmembrane glycoprotein CEACAM1 is known to modulate immune cell activation and inflammatory responses, particularly in myeloid cells. However, its role in regulating organelle function in macrophages remains largely unexplored. As professional phagocytes, macrophages rely heavily on mitochondria and peroxisomes for lipid metabolism, reactive oxygen species detoxification, and the resolution of inflammation. This study aimed to elucidate the impact of CEACAM1 deletion on peroxisomal and mitochondrial function in BMDMs

BMDMs were isolated from five CEACAM1-knockout mice and five wild-type controls, and differentiated in vitro for seven days using M-CSF. Following differentiation, cells were stimulated with lipopolysaccharide (LPS) for 3 and 6 hours. Peroxisomal gene expression was assessed using quantitative RT-PCR, and protein levels were analyzed by immunofluorescence staining and confocal laser scanning microscopy (CLSM). Ultrastructural changes in organelles were visualized using transmission electron microscopy (TEM)

CEACAM1-deficient BMDMs displayed significantly reduced expression of key peroxisomal biogenesis markers, including ABCD3, Thiolase, and GNPAT, at both the mRNA and protein levels. This downregulation persisted even after LPS stimulation, indicating a sustained impairment in peroxisomal function. TEM analysis revealed alterations in peroxisome number and an increase in mitochondrial size in CC1KO BMDMs compared to wild-type controls. These findings suggest that CEACAM1 deletion leads to organelle dysfunction, potentially affecting the metabolic capacity and inflammatory responsiveness of macrophages.

Deletion of CEACAM1 disrupts peroxisomal biogenesis and alters mitochondrial morphology in BMDMs, pointing to its essential role in maintaining organelle homeostasis. These results provide new insights into how CEACAM1 contributes to the metabolic and immune functions of macrophages.

Poster 28:

Title:
Loss of DSC1 reduces cell cohesion and expression of cell cycle-associated genes in human and murine skin models

Authors:
Vivien Beyersdorfer (Hamburg, Germany)
Marie-Therès Wanuske (Basel, Switzerland)
Chiara Stüdle (Basel, Switzerland)
Ramón Castellano-Martinez (Basel, Switzerland)
Maitreyi Rathod (Hamburg, Germany)
Henriette Franz (Basel, Switzerland)
Volker Spindler (Hamburg, Germany)
v.beyersdorfer@uke.de

Abstract:
Desmosomes mediate strong cell cohesion, but the layer-specific patterning of desmosomal cadherins, desmoglein (DSG) 1-4 and desmocollin (DSC) 1-3, in the epidermis suggest additional functions with regard to signalling and epidermal differentiation, which is only partially understood. The DSC1 isoform is particularly interesting as its expression correlates with the onset of keratinization and terminal differentiation.

The influence of DSC1 on epidermal differentiation was addressed by silencing DSC1 in normal human epidermal keratinocytes (NHEK) followed by immunohistochemistry analysis of 3D reconstructed human epidermis (3D-RHE). RNAseq of 3D-RHEs was performed to identify targets associated with proliferation and differentiation in DSC1 knock-down 3D-RHEs. A conditional DSC1 knockout mouse model was established to study the role of suprabasally expressed DSC1 in regulation of basal cell proliferation by paracrine signalling.

In 3D-RHEs, DSC1 knockdown resulted in thinner epidermis and reduced proliferation as determined by Ki67 stainings. RNAseq revealed alterations in proliferation and cell cycle progression. In gene set enrichment analysis, genes associated with S-phase were significantly downregulated. In suprabasal-specific DSC1 knockout mice, a loss of cell cohesion was evident by blistering in the superficial layers of the epidermis. Downregulation of the S-phase specific marker MCM2 suggests a paracrine regulation of basal keratinocyte proliferation dependent on suprabasal DSC1.

Our data demonstrate that DSC1 is required for the integrity of the epidermis and influences the balance of proliferation and differentiation, potentially by modulating specific phases of the cell cycle.

Poster 29:

Title:
Tracheal epithelial tuft cell populations expand after stimulation or depletion

Authors:
Charlotte Klumpp (Homburg, Germany)
Paul Könnel (Homburg, Germany)
Amanda Wyatt (Homburg, Germany)
Veit Flockerzi (Homburg, Germany)
Ulrich Boehm (Homburg, Germany)
Monika I. Hollenhorst (Homburg, Germany)
Gabriela Krasteva-Christ (Homburg, Germany)
charlotteklumpp@web.de

Abstract:
Tracheal tuft cells are cholinergic chemosensory cells, which regulate innate immune functions after activation of the canonical bitter taste signalling cascade with its member the Trpm5 (transient receptor potential melastatin 5) ion channel. They are considered as a stable population within the epithelial layer with slow turnover. Here, we aimed to characterize the turnover of tracheal epithelial tuft cells and generated two mouse models.

We performed an in vivo stimulation of tuft cells with clozapine-N-oxide (CNO) experiments in a chemogenetic mouse model (Trpm5-DREADD) and a depletion with diphtheria toxin in a mouse model expressing the diphtheria toxin receptor in tuft cells (Trpm5-DTR).

In vivo stimulation of tuft cells with CNO in Trpm5-DREADD mice led to an increase in tuft cell numbers 3 days and 7 days after treatment compared to the CNO application in wild-type mice. After 21 days, no differences in tuft cell numbers were observed between the two experimental groups. Tuft cells were rarely labelled by BrdU. In addition, there were no differences in numbers of other epithelial cells labelled for BrdU at 3, 7 or 21 days. After depletion of tuft cells with diphtheria toxin administered intratracheally, tuft cells were able to regenerate over time. Yet, their population remained reduced compared to untreated wild-type tracheae after 21 days. Interestingly, cells positive for DCAMKL1 emerged in the lungs 21 days after their depletion.

In conclusion, tuft cell numbers transiently increase upon stimulation and likely regenerate through transdifferentiation from other epithelial cell types.

Poster 30:

Title:
A novel desmoglein-2 founder variant leads to Arrhythmogenic Cardiomyopathy with fibrosis in the left ventricular apex leading to thrombus formation and apoplectic events

Authors:
Sandra A. Hemkemeyer (Hamburg, Germany)
Jennifer Schröder-Schwarz (Hamburg, Germany)
Julia Kemmling (Hamburg, Germany)
Katja E. Odening (Bern, Switzerland)
Marina Rieder (Bern, Switzerland)
Camilla Schinner (Bern, Switzerland)
s.hemkemeyer@uke.de

Abstract:
Arrhythmogenic cardiomyopathy (ACM) is a hereditary disease clinically defined by arrhythmias and progressive fibrofatty myocardial replacement with impaired cardiac function and sudden cardiac death. ACM is caused by pathogenic variants in desmosome cell-cell adhesion proteins. A new desmoglein-2 (DSG2) mutation c.523+2T>C was recently identified as a founder variant in the Bernese Oberland, with a series of homozygous carriers presenting with apoplectic events and/or left ventricle-thrombi in addition to ACM. In this study, we evaluate Dsg2 ACM mouse models to gain insight into the cause of the newly identified disease phenotype.

To model the patient variant presumably leading to decay of DSG2, an inducible Dsg2-knock-out mouse model (iDsg2 KO) was evaluated in parallel to different mouse lines carrying the adhesion-deficient Dsg2-W2A knock-in mutation. In vivo echocardiography and ECG was performed followed by serial cardiac histology sections characterized by Sirius Red fibrosis staining. Findings were compared to results from patients homozygous for the DSG2 variant.

iDsg2 KO as well as Dsg2-W2A mutant mice develop biventricular fibrosis, impaired cardiac output and ventricular arrhythmia. Several mice revealed blood clots in the apical region of the left ventricle together with wall motion abnormalities and left ventricular fibrosis in the apex similar to data revealed from patients.

iDsg2 KO mice recapitulate the patient situation with respect to the analyzed cardiac parameters. We suggest left ventricular apical fibrosis and wall motion abnormalities due to DSG2 deficiency to be causative for thrombus formation with subsequent embolism to the brain. Together, this highlights a novel pathological feature of ACM.

Poster 31:

Title:
CELL FATE DETERMINATION OF DIRECTLY REPROGRAMMED MOUSE AND HUMAN KIDNEY CELLS

Authors:
Ruth Röck (Zurich, Switzerland)
ruth.roeck@anatomy.uzh.ch

Abstract:
Despite increasing demand for understanding and treating kidney diseases, in vitro modeling options remain limited. Our work focuses on uncovering the molecular programs underlying kidney development and disease, and we have pioneered the direct reprogramming of renal tubular cells [1]. However, the mechanisms driving this process remain incompletely understood, and further studies are needed to assess assay stability and cellular heterogeneity.

Cell type conversion from mouse fibroblasts to induced renal epithelial cells (iRECs) is achieved using only four transcription factors. iRECs are morphologically and functionally similar to primary kidney cells, yet they can be stably cultured and bioprinted into tubular structures at near-physiological scales [2]. Our current efforts aim to deeply characterize iRECs through bulk and single-cell RNA sequencing.

We found that reprogramming efficiency could be significantly improved by optimizing culturing conditions, timing, and supplementing compounds that enhance cell viability—raising efficiency from 1% to 10%. This advancement allowed for a detailed investigation of iREC transcriptomes, revealing the presence of multiple kidney cell type identities. Importantly, we also succeeded in generating human iRECs, enabling further testing of transcription factors and exploration of their roles in human kidney development.

These findings advance our understanding of direct reprogramming and pave the way toward refining these methods for more reliable and efficient applications in disease modeling and drug testing. By enhancing reprogramming strategies and cellular characterization, we aim to establish a robust platform for studying kidney biology and therapeutic development.

Poster 32:

Title:
Peroxisomal Dynamics and Functional Adaptation During Human Cardiomyocyte Differentiation

Authors:
Heike Bömmel (Würzburg, Germany)
Hanna Eberl (Würzburg, Germany)
Anggi Muhatar Pratama (Würzburg, Germany)
Süleyman Ergün (Würzburg, Germany)
Katrin Streckfuß-Bömeke (Würzburg, Germany)
Srikanth Karnati (Würzburg, Germany)
srikanth.karnati@uni-wuerzburg.de

Abstract:
Peroxisomes are metabolically active and dynamic organelles involved in key cellular processes such as lipid metabolism, reactive oxygen species (ROS) detoxification, and plasmalogen synthesis. Their abundance and functional state are tightly regulated during development and differentiation. In cardiac tissue, peroxisomes contribute to calcium homeostasis and ROS control, thereby influencing myocardial contractility and electrophysiological stability. Peroxisomal dysfunction, as seen in Zellweger spectrum disorders, has been linked to abnormal cardiac development and impaired fatty acid β-oxidation. While mitochondrial remodeling during cardiogenesis is well documented, the role of peroxisomes in this context remains poorly understood.

To address this knowledge gap, we used human induced pluripotent stem cells (iPSCs) to model in vitro cardiac differentiation. Peroxisomal dynamics were assessed across key stages of differentiation using immunofluorescence microscopy for morphology and localization, Western blotting for protein abundance, and quantitative real-time PCR (qRT-PCR) for transcriptional profiling of peroxisomal genes.

Our preliminary data reveal a stage-specific increase in peroxisomal number and expression of peroxisomal enzymes during cardiomyocyte maturation. These changes coincide with metabolic reprogramming and enhanced lipid utilization, indicating an upregulation of peroxisomal β-oxidation during late stages of differentiation. Whether metabolites derived from peroxisomes play a direct role in regulating cardiac lineage commitment remains to be determined.

These findings suggest that peroxisomes contribute significantly to the metabolic and functional maturation of cardiomyocytes. Our study highlights their potential role in cardiac cell fate decisions and opens new avenues for exploring peroxisomal dysfunction in cardiac pathologies.

Poster 33:

Title:
A Reliable AI-Based Method for Reconstructing and Analyzing Three-Dimensional Organotypic Cultures

Authors:
Annike Offt (Hamburg, Germany)
Leon Schwarzenberger (Hamburg, Germany)
Peer Stelldinger (Hamburg, Germany)
Andreas Neueder (Hamburg, Germany)
Iris Haumann (Hamburg, Germany)
Barbara M. Braunger (Hamburg, Germany)
annike.offt@student.uni-luebeck.de

Abstract:
Three-dimensional (3D) organotypic cultures are frequently used to investigate tissue morphogenesis and/or angiogenesis ex vivo. However, analysing these cultures over time in 3D without fixation remains challenging. Therefore, we aimed to establish an automated pipeline for detecting, reconstructing and quantifying angiogenic and neural sprouts from unfixed 3D organotypic cultures using phase-contrast microscopy and machine learning.

Aortic rings, choroid tissue and subventricular zone (SVZ) explants were embedded in matrices for sprouting assays. Z-stacks were acquired via digital inverted phase-contrast microscopy. A machine learning-based segmentation approach was developed to reconstruct and quantify angiogenic and neural sprouting growth, using a custom trained 3D-Unet. The pipeline included image preprocessing, automated stitching, 3D sprout reconstruction and quantitative analysis of sprouting parameters like vessel length, sprouting area and branching points.

Results were evaluated by comparing the automatic segmentation outputs with manually labelled references.

Initial reconstructions demonstrate that vascular and neural sprouting can be reliably visualized and analyzed. In contrast to manual annotations, which often vary between users and can also be inconsistent for the same user across different samples, the automated approach produces stable results that consistently lie close to the average of manual labels. This suggests that the pipeline can reduce user-dependent variability while maintaining biological accuracy.

Our approach offers a biologically relevant, automatic strategy to study sprouting processes in organotypic tissue models without staining or altering the tissue, thus enabling to analyze the same culture over time. The pipeline provides a robust and scalable alternative to manual annotation, by reducing variability and increasing throughput.

Poster 34:

Title:
Impact of LOXL1 and SERPINE1 on Cell Migration and Related Mechanisms in Glioblastoma Cell Lines

Authors:
Falko Schulz (Halle (Saale), Germany)
Tim Hohmann (Halle (Saale), Germany)
Urszula Hohmann (Halle (Saale), Germany)
Faramarz Dehghani (Halle (Saale), Germany)
falko.schulz@student.uni-halle.de

Abstract:
Glioblastoma (GBM) is the most aggressive and common primary tumor of the central nervous system. The current treatment consists of surgical resection followed by adjuvant radio-chemotherapy with temozolomide according to the Stupp protocol.

A hallmark of GBM is its uncontrolled growth, facilitated by collective cell migration. RNA sequencing analyses of cell lines with high (GBM#4, U138) and low (GBM#10, LN229) migratory properties revealed a differential mRNA expression pattern. This study investigates the effects of LOXL1 and SERPINE1 overexpression on their migratory processes.

LOXL1 (lysyl oxidase-like 1) and SERPINE1 (plasminogen activator inhibitor 1) were overexpressed in GBM#10 and LN229 cell lines. Live-cell microscopy was used to directly assess migratory activity, while FACS, immunocytochemistry and atomic force microscopy (AFM) were employed to analyze key parameters such as proliferation, adhesion, and the cytoskeletal structure of the actin cortex.

LOXL1 and SERPINE1 were successfully overexpressed at both RNA and protein levels. In LN229, both proteins affected the average speed and directional persistence of transfected cells. Additionally, they reduced cell-matrix adhesion in LN229, indicating increased cell dynamics. SERPINE1 also increased cellular elasticity and cortical tension in LN229 – markers of a dense cortical actin-myosin network. In GBM#10, LOXL1 led to an increase in the proportion of cells in the G0/G1 phase.

Both proteins have the potential to influence the migratory behavior of GBM cells. Particularly SERPINE1 promotes dynamic changes in cell adhesion and restructuring of the cortical cytoskeleton. These findings suggest that SERPINE1 might play an important role in GBM cell migration.

Poster 35:

Title:
Investigation of Nrf2's effects in tumor-associated macrophages (TAMs) on the tumor immune microenvironment (TIME)

Authors:
Johanna Vollmer (Aachen, Germany)
Julie Dick (Aachen, Germany)
Philipp Krumm (Aachen, Germany)
Nicole Böttcher (Aachen, Germany)
Thomas Pufe (Aachen, Germany)
Athanassios Fragoulis (Aachen, Germany)
jvollmer@ukaachen.de

Abstract:
The transcription factor nuclear factor erythroid 2-related factor 2 (Nrf2) is proposed to promote the polarization of tumor-associated macrophages (TAMs) toward an anti-inflammatory M2-like phenotype. M2-like TAMs express immunomodulatory surface molecules that contribute to tumor immune evasion by suppressing cytotoxic T cell response and promoting the transdifferentiation of CD4+ T cells into immunosuppressive regulatory T cells. This study aims to investigate the role of Nrf2 in shaping the immune-related behavior of TAMs upon the interaction with T cells within the tumor immune microenvironment (TIME) of hepatocellular carcinoma (HCC).

Bone marrow-derived macrophages (BMDMs) with a conditional Nrf2 knockout were co-cultured with primary CD4+ T cells in 2D cultures or in 3D multicellular tumor (MCT)-spheroids, which additionally contained murine N-HCC25 cells to model the TIME. BMDM and T cell distribution in spheroid was assessed by flow cytometry. Gene expression was analyzed by RT-qPCR, focusing on Nrf2 target genes, macrophage markers and macrophage-associated immunoregulatory markers.

We established a 3D culture model suitable for investigating TAM-T cell interactions within TIME. The incorporated amount of BMDMs and T cells was confirmed by flow cytometry. T cell co-cultivation resulted in enhanced expression of macrophage markers in the 2D and 3D cultures, while regulation of immunomodulatory genes were mainly observed in MCT-spheroids.

In conclusion, our findings highlight the suitability of the 3D MCT-spheroid model for studying immune cell interactions within the HCC TIME and suggest that Nrf2 may influence the transcriptional profile of TAMs in response to T cell-associated cues within a tumor-like environment.

Poster 36:

Title:
Effects of Micro- and Nanoplastics on the Ocular Surface

Authors:
Agatha Raffauf (Erlangen, Germany)
Fabian Garreis (Erlangen, Germany)
Friedrich Paulsen (Erlangen, Germany)
Ingrid Zahn (Erlangen, Germany)
Katharina Jüngert (Erlangen, Germany)
agatha.raffauf@fau.de

Abstract:
The etiology of dry eye disease (DED) is complex and not yet fully understood. Consequently, treatment is primarily symptomatic. Contact lens wear is a common risk factor. Despite their mechanical irritation of the ocular surface, plastic particles can be released from contact lenses or reach the ocular surface via blood stream. This study aims to detect interactions of micro- and nanoplastic particles with the ocular surface and explore their role in development of DED.

Immortalized human cell lines from the cornea, conjunctiva, and meibomian gland were stimulated with 0.5, 1.0, and 3.0µm polystyrene micro- and nanobeads (MNBs), a widely used plastic. Particle uptake was analyzed via transmission electron microscopy. Cell viability was assessed using annexin V/PI staining and flow cytometry. Intracellular ATP levels and oxidative stress were measured using bioluminescence and ROS assays. Cell proliferation and migration were evaluated using Hoechst staining and scratch assay.

MNBs were taken up by human epithelial cells from the cornea, conjunctiva, and meibomian gland, most likely through endocytic mechanisms. Their intracellular presence significantly reduced cell viability, without inducing a measurable increase in apoptosis. Furthermore, MNB exposure led to decreased proliferation and migration, accompanied by signs of mechanical and/or oxidative stress.

MNBs were successfully internalized by ocular surface cells and decreased cell viability, cell proliferation and cell migration. These findings suggest that microplastic exposure may contribute to ocular surface stress, and, thus, DED pathogenesis. Further research is needed to investigate the involvement of MNBs in inflammatory processes and their long-term impact on ocular surface health.

Poster 37:

Title:
TRPM5 channel controls exocytosis in tracheal tuft cells

Authors:
Na Zhao1, Amanda Wyatt2, Thomas Gudermann3,5, Vladimir Chubanov3, Ulrich Boehm2,4,6, Gabriela Krasteva-Christ1,4,6

1Institute of Anatomy and Cell Biology, Saarland University, Homburg, Germany
2Experimental Pharmacology, Center for Molecular Signaling (PZMS), Saarland University, Homburg, Germany
3Walther-Straub Institute of Pharmacology and Toxicology, LMU Munich, Munich, Germany
4Center for Gender-Specific Biology and Medicine (CGBM), Saarland University, Homburg, Germany
5Comprehensive Pneumology Center, a member of the German Center for Lung Research (DZL), Munich, Germany
6PharmaScienceHub (PSH), Saarland University, Saarbrücken, Germany
na.zhao@uks.eu

Abstract:
Tracheal tuft cells as specialized epithelial cells play a pivotal role in triggering global epithelial defense programs. Here we investigate whether Trpm5 channel plays a role in Ca2+ dependent exocytosis of tracheal tuft cells and study its physiological role in tuft cell-driven global epithelial responses.

We employed whole-cell patch-clamp recordings combined with UV uncaging, Ca2+ imaging and immunohistochemistry. Primary tuft cells were isolated from reporter mice expressing GFP under the ChAT promoter.

We found that exocytosis in tuft cells is induced in a Ca2-dependent manner and specifically modulated by Trpm5 channel activity. In addition to releasing acetylcholine upon Trpm5 activation, we observed that a subset of tracheal tuft cells also releases ATP, as demonstrated using ATP-sniffer cells. This ATP release was mediated by activation of pannexin-1 channels (Panx1) and depended on activation of Trpm5 channels, as confirmed by the absence of ATP release and Panx1-currents in tuft cells lacking Trpm5 protein. Tracheal tuft cells were found exhibit contacts to sensory nerve fibers, as shown by immunostaining for the pan-neuronal marker PGP9.5. In the coculture experiments with primary vagal sensory neurons and tracheal tuft cells we functionally confirmed that vagal sensory neurons receive signals from tuft cells via activation of Trpm5 channels in tuft cells. This finding was further supported by Ca2+ imaging of nerve fibers and tuft cells in whole tracheal preparations ex vivo.

Taken together, we propose that the Trpm5 channel in tracheal tuft cell plays a crucial role in triggering global signaling networks in the respiratory tract.

Poster 38:

Title:
Potential for odontoblast lineage cells to attract blood vessels into the developing dental papilla

Authors:
Masataka Sunohara (Tokyo, Japan)
Hidetaka Murata (Tochigi, Japan)
Miyoko Yokoyama (Tokyo, Japan)
Kingo Suzuki (Tokyo, Japan)
ma-suno@tky.ndu.ac.jp

Abstract:
During mouse tooth development, vascular endothelial cells invade the dental papilla and form a vascular network through angiogenesis. In adult tissues, megakaryocytes and/or platelets express angiogenic molecules and promote angiogenesis. However, it remains unclear which cells play a role in inducing and guiding vascular invasion into the dental papilla during embryonic tooth development. In this study, we aimed to address this issue by analyzing the spatiotemporal changes in c-Mpl and Ang-1 mRNA expression in mouse fetal molars.

Serial frontal sections were prepared from the heads of mouse fetuses (E12.5, 14.5, 17.5, 18.5), and morphological analysis was performed by H&E staining. mRNA expression analysis of c-Mpl, Ang-1, Tie-2, CD41, and VEGFR2 was also performed by in situ hybridization, and the spatiotemporal distribution profiles were compared.

c-Mpl-expressing cells were scattered throughout multiple regions of the developing tooth germ, including the peridental mesenchyme, dental papilla, enamel organ, and dental lamina. The spatiotemporal distribution pattern of c-Mpl-expressing cells was similar to that of Ang-1-expressing cells, followed by invasion of vascular endothelial cells. During the early bell stage, c-Mpl-expressing cells accumulated at the periphery of the dental papilla along the inner enamel epithelium in the future cusp region, and were morphologically confirmed to be differentiating odontoblasts. These c-Mpl-expressing cells did not express CD41 mRNA.

We propose that c-Mpl-expressing cells spatiotemporally distributed in the dental papilla, at least in part, are odontoblasts and their precursor cells, which may produce angiogenic molecules such as Ang-1 during migration to the odontoblast differentiation site and recruit blood vessels.

Poster 39:

Title:
Spatial and temporal mapping of the postnatal brain metabolome in mice using MALDI-ToF Imaging Mass Spectrometry

Authors:
Susanne Wiemann (Bielefeld, Germany)
Tobias Diekamp (Bielefeld, Germany)
Judith Neumann (Bielefeld, Germany)
Björn Spittau (Bielefeld, Germany)
Susanne.Wiemann@uni-bielefeld.de

Abstract:
The brain metabolome, particularly its lipid composition, plays an important role in maintaining physiological brain functions and is implicated in developmental and neurodegenerative processes. While the adult brain metabolome has been studied, little is known about the spatial and temporal dynamics of lipid metabolites during postnatal brain development.

To investigate these changes, we employed matrix-assisted laser desorption/ionization time-of-flight imaging mass spectrometry (MALDI-ToF-IMS) on fresh frozen brain sections of C57BL/6 wild-type mice at five defined postnatal stages: P0, P5, P10, P15, and P30. We used matrix systems optimized for the detection of small molecules and neurotransmitter-associated lipids to ensure high spatial resolution.

Our analyses enabled the spatially resolved detection of key lipid metabolites, including sphingomyelins, ceramides, and phosphatidylcholines. These metabolites displayed distinct regional and developmental expression patterns reflecting maturation processes in various brain regions. Developmental stage-specific lipid signatures revealed dynamic remodeling of the brain metabolome during postnatal development.

This study shows that MALDI-ToF-IMS is a useful method for mapping metabolic changes in the developing brain. Here, we are able to detect important lipids such as sphingomyelin and phosphatidylcholine, which change during postnatal development. Furthermore, our results demonstrate that imaging mass spectrometry can capture spatial and temporal shifts in brain metabolite composition. Collectively, our results highlight the efficacy of imaging mass spectrometry as a powerful tool for characterizing postnatal brain development at the metabolomic level.

Poster 40:

Title:
Analysis of a human mutation in the transcription factor Bcl11a using a mouse model

Authors:
Sophia Lisovaya (Ulm, Germany)
Anne-Marie Neumann (Ulm, Germany)
Christoph Wiegreffe (Ulm, Germany)
Stefan Britsch (Ulm, Germany)
sophia.lisovaya@uni-ulm.de

Abstract:
Dias-Logan syndrome is a neurodevelopmental disorder caused by mutations in the human BCL11A gene, associated with intellectual disability, speech and language delay, and autism spectrum symptoms. The transcription factor Bcl11a is known to be essential for murine central nervous system development. However, the specific molecular mechanisms linking BCL11A dysfunction to disease symptoms remain largely unclear. Here, we aimed to generate and characterize a mouse model mirroring a previously described human mutation.

We generated the Bcl11aem3Δ529_530fs*25Cw mouse line using i-GONAD, introducing a frameshift mutation in exon 4 that disrupts the zinc finger domains required for DNA binding. We systematically analyzed homozygous mutant mice from this line (Bcl11ahΔ/hΔ) and compared their phenotypes during hippocampal and neocortical development with forebrain-specific Bcl11a knockouts (Bcl11aflox/flox; Emx1IRESCre).

Bcl11ahΔ/hΔ mice die perinatally with no Bcl11a protein detected by Western blot or immunofluorescence. Morphological and transcriptomic analyses revealed both shared and distinct phenotypes when compared with Bcl11aflox/flox; Emx1IRESCre mutants.

Well-established conditional knockout mice rely on, but are also limited by, spatiotemporal expression patterns of driver promoters. Our findings suggest a multifaceted regulation by Bcl11a mediated through transregional communication, early progenitor effects, or its expression in multiple cell types. Hence, the Bcl11aem3Δ529_530fs*25Cw line enables deeper insights into BCL11A-related disease mechanisms and potential therapeutic targets.

Poster 41:

Title:
Quantification of mitochondria and their dispersion in alveolar epithelial type I cells from infant- to adulthood in humans

Authors:
Arne K. Schierz (Hannover, Germany)
Giacomo Rößler (Hannover, Germany)
Jan Philipp Schneider (Hannover, Germany)
Stefan A. Tschanz (Bern, Switzerland)
Christopher Werlein (Hannover, Germany)
Danny Jonigk (Aachen, Germany)
Julia Schipke (Hannover, Germany)
Christian Mühlfeld (Hannover, Germany)
schierz.arne@mh-hannover.de

Abstract:
Alveolar epithelial type I cells (AE1C) form most of the surface area of the outer layer of the air blood barrier. During postnatal human lung development alveolarization and microvascular maturation increase the number of alveoli and lead to a single-layered capillary network with minimal distances between the air and the erythrocytes in the capillaries leading to optimized gas exchange. Images of adult AE1C from serial block face scanning electron microscopy (SBF SEM) showed aggregates of mitochondria at septal connective tissue pillars without underlying capillaries. Therefore, it was hypothesized that AE1C mitochondria are located primarily in cellular compartments above connective tissue and that changes in amount and distribution of mitochondria in AE1C take place during postnatal lung development.

Archived lung material from three infants (26 and 30 days, 6 months) and three adults (20, 39, 40 years) were investigated by light microscopy, transmission electron microscopy and stereology.

The relative amount of mitochondria per AE1C were the same in infants and adults with a mean value of 6.3%. Mitochondrial profiles in AE1C were found with a frequency of 3:1 located on top of capillaries compared to the location at connective tissue pillars in both groups. In relation to the volume fraction of both cellular compartments, mitochondrial profiles in infants were preferentially located at the gas exchange region, while in adults they were preferentially located above tissue pillars.

Postnatal lung development is linked to a change in distribution of mitochondria in AE1C, while the relative fraction stays constant.

Poster 42:

Title:
Precise, predictable genome integrations by deep learning–assisted design of microhomology-based templates.

Authors:
Thomas Naert (Zürich, Schweiz)
Taiyo Yamamoto (Zürich, Schweiz)
Ruth Röck (Zürich, Schweiz)
Soeren Lienkamp (Zürich, Schweiz)
soeren.lienkamp@uzh.ch

Abstract:
Precise CRISPR-based DNA integration and editing remains challenging, largely due to insufficient control of the repair process.

We find that repair at the genome-cargo interface is predictable by deep-learning models and adheres to sequence context specific rules. Based on in silico predictions, we devised a strategy of base-pair tandem repeat repair arms matching microhomologies at double-strand breaks. These repeat homology arms promote frame-retentive cassette integration and reduce deletions at both the target site and within the transgene.

We demonstrate precise integrations at 32 loci in HEK293T cells. Germline-transmissible transgene integration and endogenous protein tagging in Xenopus and adult mouse brains demonstrated precise integration during early embryonic cleavage and in non-dividing, differentiated cells. Optimized repair arms also facilitated small edits for scarless single- or double-nucleotide changes using oligonucleotide templates in vitro and in vivo.

We provide the design-tool Pythia to facilitate precise genomic integration and editing for experimental and therapeutic purposes for a wide range of target cell types and applications.

Poster 43:

Title:
Automated Euler number assessment of the alveolar capillary network based on deep learning segmentation with verification by stereological methods

Authors:
Julia Schmidt (Hannover, Germany)
Jonas Labode (Hannover, Germany)
Christoph Wrede (Hannover, Germany)
Yannick Regin (Leuven, Belgium)
Jaan Toelen (Leuven, Belgium)
Christian Mühlfeld (Hannover, Germany)
labode.jonas@mh-hannover.de

Abstract:
The alveolar capillary network (ACN) plays a major role in various forms of lung disease. One example, that initiated the current study, is bronchopulmonary dysplasia (BPD), where a reduced angiogenesis is postulated to inhibit pulmonary development.

For the assessment of changes in the ACN, an examination of its 3D structure is necessary. This is done by image segmentation, which constitutes a major hurdle, due to the ACN's vast and complex structure, making even the manual segmentation of small regions extremely time consuming. Automatic methods, employing static thresholds, are not sufficient either. Thus, this study set out to train a neural network (NN) for this task, using existing image material. Result verification was performed by comparison to manual segmentations and an unbiased stereological counting method.

Lung samples were acquired from a BPD rabbit model, incorporating prematurity and hyperoxia. Volume images of the ACN were gathered using serial block face scanning electron microscopy. Segmentations for training and testing were created using a watershed filter in conjunction with manual segmentation. The NN used was a U-Net implementation. Verification was conducted using a disector analysis.

Despite limited training images, the NN outperformed the common watershed approach. Nevertheless, the results still required some manual completion.

The overall results of this segmentation workflow could be verified with subsequent calculation of the Euler number from the resulting 3D capillary network and comparison to a stereological connectivity analysis.

This study demonstrates the potential of a NN for ACN segmentation. Nevertheless, model improvements are necessary to achieve full performance.

Poster 44:

Title:
How alimentary obesity affects pulmonary structure and function in hyperoxic acute lung injury

Authors:
Morten Kampelmann (Hannover, Germany)
Christian Mühlfeld (Hannover, Germany)
Julia Schipke (Hannover, Germany)
kampelmann.morten@mh-hannover.de

Abstract:
Oxygen therapy is one of the most common clinical treatments for critically ill patients. However, depending on the fractions, high oxygen doses can be harmful and may induce a hyperoxic lung injury (HALI). An “obesity paradox”, i.e. lower mortality with high body weight, has been described for several pathologies (e.g. COPD). Its existence in critical illness is under discussion. Therefore, this study investigated the extent of hyperoxic lung injury in a controlled animal model of diet-induced obesity.

Male mice were fed for 30 weeks either a control (CD, 11 kcal% fat) or a high fat diet (HFD, 60 kcal% fat). During the last 72h, part of the mice were exposed to hyperoxia (FiO2: 0.9; -Hyper). After assessing lung mechanics and blood gases, lungs were prepared for stereological and molecular analyses.

Hyperoxia induced reduced compliance, hypercapnia and reduced oxygen saturation. Structurally, HALI mice showed edema, fragmentation of alveolar epithelial type 1 (AE1) cells and loss of AE2 cells. These changes were significantly greater in CD-Hyper compared to HFD-Hyper. The HFD-fed hyperoxic mice simultaneously exhibited an increase in AE1 cell volume, while the volume of AE2 cells decreased. The lung proteome varied between the diet groups. While pathways like mTOR signaling and mitotic activity were regulated in CD-Hyper, HFD-Hyper showed alterations in surfactant metabolism and cellular senescence.

HFD-induced obesity significantly alters the susceptibility to HALI, particularly the structural alterations suggest the existence of an obesity paradox in HALI.

Poster 45:

Title:
Contextual fear conditioning leads to hypoinnervation of the left ventricle in female but not male mice

Authors:
Luca M. Lautenschläger (Hannover, Germany)
Lara Mariel Chirich Barreira (Magdeburg, Germany)
Anne Albrecht (Magdeburg, Germany)
Christian Mühlfeld (Hannover, Germany)
lautenschlaeger.luca@mh-hannover.de

Abstract:
About 34% of people will experience an anxiety disorder once in their life, the chances for women being two times higher than for men. As fear formation is well conserved within mammals, rodents are a suitable model. Whilst sex-specific fear conditioning-induced circuits, molecular and cellular pathways are well studied in the brain, the impact of such events on the heart remain elusive. This study investigated morphological changes of the heart and its innervation following fear conditioning in mice, emphasising sex-specific differences.

Contextual fear conditioning was performed in female and male mice, comparing against non-shocked mice. Hearts and stellate ganglia were harvested 24h after fear memory retrieval and analysed by transmission electron microscopy and light microscopy. Stereological assessment of total nerve fibre length within left ventricular myocardial tissue was performed, using an immunostaining against protein gene product 9.5. The ultrastructure of stellate ganglia and hearts was studied.

For statistical analyses, 2W-ANOVA was performed.

At fear memory retrieval, mice that have received a foot-shock, expressed freezing; a defensive behaviour characterized by immobility. Female mice froze longer compared to male mice, whilst no sex difference was present in activity of controls. Total nerve fibre length was significantly reduced between the female fear conditioned group and their control, whilst male mice showed no difference in nerve fibre length, irrespective of treatment.

Sex seems to contribute to subacute effects of fear conditioning on myocardial innervation, indicating differences in integration of autonomous nervous responses leading to different fear reactions and anxiety between male and female mice.

Poster 46:

Title:
Adaptations to extreme habitat of naked mole-rat (Heterocephalus glaber) display unique features in kidney epithelial morphology and gene expression

Authors:
Antonia Bellingkrodt (Berlin, Germany)
Daniel Mendéz Aranda (Berlin, Germany)
Mingzhen Su (Berlin, Germanys)
Susanne Holtze (Berlin, Germany)
Thomas Hildebrandt (Berlin, Germany)
Gary Lewin (Berlin, Germany)
Sebastian Bachmann (Berlin, Germany)
antonia.bellingkrodt@charite.de

Abstract:
The naked mole-rat (Heterocephalus glaber) is a subterranean troglobiont from sub-Saharan Africa. It has extreme longevity, feeds on underground roots and bulbs, does not drink, and may activate fructose-driven glycolysis for ATP generation and metabolic water production. It has a maximum urine concentration capacity of 1500 mOsm/kg H2O. We explored its renal architecture for mechanistic insight and evaluated its transcriptomic profiles compared to laboratory rodents.

Naked mole-rats of both sexes were anesthetized, sacrificed and the kidneys either immersion-fixed or perfusion-fixed via the heart for histology and immunohistochemistry. For transcriptome analysis kidneys were removed and stored frozen until use.

The kidney parenchyma revealed rodent-type zonation of cortex, outer and inner stripe of outer medulla (OSOM, ISOM), and inner zone/papilla (IZ). Zonal proportions, however, differed markedly with IS measuring 51% (rat: 17%) and IZ 26% (rat: 56%) along cortico-papillary axis. Cortical organization of glomerular, tubular, vascular and interstitial components was similar to rodent while in IS, multiple loop bends, formed of descending and ascending thick ascending limb (TAL) portions, were encountered variably at different levels. TAL expressed uromodulin and NKCC2 throughout but showed cell type heterogeneity for K+ channel and claudin expression. Few long-looped AQP1-positive descending thin limbs reached IZ. Transcriptomics indicated energy metabolic pathways significantly distinct from rodent kidney.

Kidney epithelial morphology of the naked mole-rat displays features significantly different from laboratory rodents. In particular, organization of the loop of Henle is diverse. Gene expression profiles confirm adaptations of the nephron with respect to lack of O2, water, and homeothermy.

Poster 47:

Title:
Number, source, and three-dimensional (3D) complexity of the Arteries supplying the Carotid Body

Authors:
Atieh Seyedian Moghaddam (Vienna, Austria)
Stefan Geyer (Vienna, Austria)
Berna DOGAN (Tokat, Turkey)
Paata PRUIDZE (Vienna, Austria)
Jeremias T. WENINGER (Vienna, Austria)
Karoline SCHWENDT (Vienna, Austria)
Wolfgang Weninger (Vienna, Austria)
atieh.seyedianmoghaddam@meduniwien.ac.at

Abstract:
Precise anatomical knowledge of blood supply to the carotid body (CB) is essential for treating CB tumors and for understanding the impact of carotid artery atherosclerosis on breathing and blood pressure. This presentation aims to provide comprehensive and statistically significant information on the number, origin, and morphology of human glomic arteries (GAs).

Carotid bifurcations from 60 body donors (30 females and 30 males) were harvested and subjected to high-resolution episcopic microscopy (HREM). Employing the Amira® software three-dimensional (3D) models of all 120 CBs and their arteries were created and analyzed.

CBs were supplied by up to five GAs: 25% by one, 54% by two, 17% by three, and 4 % by four or five. They originated from the external carotid artery (60.5%), common carotid artery (23%), ascending pharyngeal artery (10.3%), carotid bifurcation (5.4%), and internal carotid artery (0.08%). Their mean length was 2.36 mm, with left-sided and male GAs being significantly longer. Their mean tortuosity value was 1.56 (1.03-3.57), with no significant differences between left and right-sided and female and male specimens.

The results of our systematic study significantly expand existing anatomic knowledge. They suggest rewriting anatomical textbooks, which should state that 2 GAs are the norm, that up to 5 GAs can exist and that the external carotid artery is their main source. Furthermore, atherosclerosis research and preoperative assessment of the GAs for CB tumor embolization will substantially profit from this information.

Poster 48:

Title:
The importance of anatomy for the anthropological analysis of Hallstatt-Celtic skeletal burials: On the question of contemporaneous burial

Authors:
Horst Claassen (D-91054 Erlangen, Germany)
horst.claassen@fau.de

Abstract:
The Hallstatt period covers the epoch of the early Celts (approx. 800 to 500 BC), who for the first time used iron instead of bronze and established a prehistoric industry through salt mining. In the Hallstatt burial mound fields of Dietfurt and Schirndorf burials were repeatedly found that showed two skeletons lying next to each other in almost the same state of preservation.

Individual fragments of the skeletons were reassambled with glue. Ossification of the cranial sutures, density of the cancellous bone in the proximal humerus and femur, condition of the symphysial facies of the symphysis pubica were used for the estimation of age. Features of the skull, the pelvis as well as length measurements of the limb bones and diameter of the caput femoris were consulted for sex diagnosis.

In burial mound 13 at Dietfurt, the skeleton of a 50-year-old man were identified to the left of the skeleton of a 30-year-old woman. In tumulus 2, the skeleton of a 35-year-old man was detected to the left of the skeleton of a 30-year-old man, between whose knees was the skeleton of a foetus / newborn baby. The robust skulls stood out among all the others.

According to the findings, the bodies had been laid down at the same time or at least at imperceptible intervals. Two hypotheses are conceivable: 1. The deceased died almost simultaneously from an infectious disease. 2. One of the two dead had to follow the deceased, which is known as the succession of the dead.

Poster 49:

Title:
Rare vascular variations - The ulnar artery and superficial brachioulnar artery: A case report and review of the literature.

Authors:
Fabian Peter (Mainz, Germany)
Fabian Langenbahn (Mainz, Germany)
Theresa Schöche (Mainz, Germany)
Stefanie Peter (Mainz, Germany)
Stefanie Lagaly- Schmeisser (Mainz, Germany)
Maier Jannik (Mainz, Germany)
Tanja Wolloscheck (Mainz, Germany)
Michael J. Schmeisser (Mainz, Germany)
peter@uni-mainz.de

Abstract:
Cardiovascular disease is among the most common causes of death worldwide.

Interventions, such as percutaneous coronary intervention, used to recanalise coronary stents, require precise anatomical knowledge, particularly regarding vascular variations, to avoid complications.

In our study, we describe a rare anatomical variation of the superficial brachioulnar artery (SBUA).

We present an SBUA originating from the brachial artery in its upper third further running superficially in a straight course to the hand, where it comprises the full function of the ulnar artery.

At the same time, the regular ulnar artery terminates as a hypoplastic vessel which exclusively supplies the local musculature. This combination expands the existing literature and highlights the clinical relevance of vascular variations.

Our case report underscores the importance of precise preoperative imaging to identify vascular anomalies early on and at the same time minimise patient safety-related complications. Additionally, this new variation of the SBUA emphasises the need to integrate anatomical variations more thoroughly into medical education and research. The latter would ensure the systematical capture of their prevalence and implications to improve clinical practice in the long run.

Poster 50:

Title:
Variant anatomy of the fascial canal v.facialis in the parotid-masticatory region.

Authors:
Artem Mirontsev (Moscow, Russia)
Sergey Dydykin (Moscow, Russia)
Ekaterina Blinova (Moscow, Russia)
Yuriy Vasil'ev (Moscow, Russia)
Kirill Zhandarov (Moscow, Russia)
mirontsev_a_v@staff.sechenov.ru

Abstract:
The anatomy of facial fascial coverings has generated increased interest in recent years. The aim of our research is to investigate the structure, position, and variations in the facial vein's facial canal in the area near the anterior border of the masseter muscle.

We used frozen cadaver heads from men and women (n=30), prepared using a silicone compound. The main research methods included standard anatomical dissection. Measurements were made using standard measuring instruments.

The facial vein was present in all 30 cases (100%). Two variants of its position were identified: in the first case, the vein is located in front of the masseter muscle in 63.3% (n=19) of the cases. In all these cases, it was located within a fascial canal formed by the splitting of the superficial and deep layers of the parotidomasseteric fascia. In the second variant, the vein ran more superficially on the surface of the masseter in 36.7% (n=11) of the cases. Moreover, in most of these cases, the vein was also located within the split of the parotideomasseter fascia (23.3%, n=7). In other cases, the vein passed through without a distinct fascial canal (13.3%, n=4).

It can be concluded that there is a large number of observations of the facial vein within its own fascial canal. These data raise the question about the functional significance of this channel in the region, considering the absence of a similar channel in a neighboring facial artery.

Poster 51:

Title:
How to deal with archaeological human remains in anatomical collections? The mummy of Mainz

Authors:
Sven Schumann (Neuruppin, Germany)
Victor Brendel (Mainz, Germany)
Constanze Nieß (Frankfurt am Main, Germany)
Richard Zehner (Frankfurt am Main, Germany)
Michael Schmeisser (Mainz, Germany)
sven.schumann@mhb-fontane.de

Abstract:
A mummy is the body of a dead human or animal whose soft tissues have been preserved either intentionally or accidentally. Mummification was an integral part of the rituals for the dead in different cultures, including ancient Egypt. Mummies have a long history beyond that of archaeological artefacts, being viewed as oddities collected by the curious. Here, we analysed a mummy head of unknown origin, which was found in the collection of the Institute of Anatomy Mainz, using a multidisciplinary approach.

We performed computed tomography, radiocarbon dating, anthropological and molecular sex and age determination, forensic face reconstruction, and provenience research.

The mummy head dates back to 345-55 BC. Morphologically it is that of a young woman. Details in mummification process are highly consistent with an Egyptian origin. It remains unclear how and when the head was included in the anatomical collection of Mainz.

Taken together, the head is most likely that of a woman of the late period of ancient Egypt or the Hellenistic Egypt. Egyptian mummies were often transferred to anatomical institutes in the past. Ethical aspects for further exhibition of Egyptian mummies were discussed with respect to Ancient Egypt religion and culture. Our efforts might serve as a framework how to deal with ancient human remains of unknown origin in anatomical collections.

Poster 52:

Title:
RENAL ARTERY VARIATIONS DETECTED BY CTA

Authors:
Anamarija Paunkoska (Skopje, North Macedonia)
Biljana Zafirova (Skopje, North Macedonia)
Julija Zhivadinovikj (Skopje, North Macedonia)
Niki Matveeva (Skopje, North Macedonia)
Elizabeta Chadikovska (Skopje, North Macedonia)
Ace Dodevski (Skopje, North Macedonia)
Biljana Trpkovska (Skopje, North Macedonia)
Biljana Bojadjieva-Stojanovska (Skopje, North Macedonia)
Jovana Kordoska (Skopje, North Macedonia)
anamarija@medf.ukim.edu.mk

Abstract:
This study aims to determine the prevalence and morphological characteristics of renal artery variations using computed tomography angiography (CTA) of the abdominal aorta.

A cross-sectional study was conducted on 37 CTA images of patients over 18 years of age.

The scans were performed using a multidetector CT scanner (MDCT Siemens, 128-slice), and the images were analyzed with Vitrea® Advanced Visualization Software.

Evaluated parameters included the origin, number and diameter of the renal arteries, presence of accessory arteries and the suprarenal and infrarenal segments of the aorta.

Normal bilateral renal vascularization was observed in 91.9% of cases. Variations were detected in 5.41% (2 cases): one with unilateral and one with bilateral accessory renal arteries. No statistically significant association was found between sex and the presence of variations (p=0.51).

Despite the limited sample size, renal artery variations were identified. Accessory renal arteries are a common anatomical variation with significant implications in clinical and surgical practice. Accurate preoperative identification using CTA is essential for optimal surgical planning, especially in urology and vascular surgery.

Poster 53:

Title:
The rupture of the insertion tendon of the gluteus medius muscle – cause and consequences

Authors:
Bernhard Tillmann (Kiel, Germany)
Wolfram Thomas (Roma, Italy)
bntill@t-online.de

Abstract:
The aim of the study is to determine whether the structure of the insertion tendon of the gluteus medius muscle may be a cause of the tendon rupture observed in osteoarthritis or spontaneous femoral neck fracture

The structure and blood supply of the insertion tendon of the gluteus medius muscle are examined in intact and ruptured tendons using histological and immunohistochemical methods. To demonstrate the tension band effect of the gluteus medius muscle, photoelastic model investigations are conducted.

In the photoelastic model experiments, the order of the isochromatics confirms the tension band effect of the gluteus medius muscle at the femoral neck. In the insertion tendon of the gluteus medius muscle, which consists of two intersecting parts, a zone of fibrocartilage is found proximal to the chondral insertion. The zone of fibrocartilage is avascular in its central area. In contrast, the ruptured fibrocartilage zone in injured tendons is highly vascularized.

The lack or insufficient tension band effect in a tendon rupture can lead to a fatigue fracture. The cause of the formation of fibrocartilage lies in the macroscopic structure of the tendon. Contraction of the gluteus medius muscle causes torsion of the two intersecting tendon segments. This causes compressive stress inside the tendon at the intersection area, is the mechanical stimulus for the formation of cartilage tissue.

The cause of the vascularization of the fibrocartilage zone and the tendon rupture is consistent with previous findings, for example, in the Achilles tendon.

Poster 54:

Title:
Modulating Food Intake by Nasal Application of Peptides Targeting NPY, MC4R and GhrR Receptors

Authors:
Kerstin Immig (Leipzig, Deutschland)
Eva-Maria Jülke (Leipzig, Deutschland)
Benginur Özbay (Leipzig, Deutschland)
Marcin Nowicki (Leipzig, Deutschland)
Annette G. Beck-Sickinger (Leipzig, Deutschland)
Ingo Bechmann (Leipzig, Deutschland)
62edizin.immig@medizin.uni-leipzig.de

Abstract:
This study investigates the modulation of food intake via targeted intranasal administration of peptides that act on key receptors involved in energy homeostasis, including the neuropeptide Y receptors (Y1R and Y2R), the melanocortin 4 receptor (MC4R), and the ghrelin receptor (GhrR). Obesity and cachexia represent two opposing yet equally challenging health conditions, both driven by dysregulation of energy intake. Peptides that selectively interact with these receptors have shown therapeutic potential; however, delivering them effectively to the brain remains a significant hurdle due to the restrictive nature of the blood-brain barrier and poor systemic bioavailability.

In this study, fluorescently labeled peptides were intranasally administered to mice. Following euthanasia under anesthesia, brain tissues were collected and analyzed using fluorescence imaging to assess peptide uptake and distribution in key regions such as the olfactory bulb, hypothalamus, and cortex. Furthermore, the effects of daily peptide administration over a 14-day period were evaluated in terms of food intake and body weight.

The results revealed trends in daily food intake that aligned with the anticipated pharmacological effects: MC4R agonists led to a reduction in food consumption, while GhrR agonists caused an increase compared to the control group.

These findings underscore the potential of intranasal peptide delivery as a non-invasive and effective strategy for influencing appetite regulation, offering promising implications for the treatment of obesity, cachexia, and related metabolic disorders.

Poster 55:

Title:
3D morphology of the fallopian tube – a morphometric post mortem study based on micro-computed tomography and x-ray microscopy – requirements for a microstent-based treatment of proximal fallopian tube occlusions

Authors:
Andreas Wree (Rostock, Germany)
Ariane Dierke (Rostock, Germany)
Marko Schulze (Rostock, Germany)
Swen Grossmann (Rostock, Germany)
Eric Bohne (Rostock, Germany)
Andrea Bock (Rostock, Germany)
Klaus-Peter Schmitz (Rostock, Germany)
Marek Zygmunt (Greifswald, Germany)
Jonas Keiler (Rostock, Germany)
Stefan Siewert (Rostock, Germany)
andreas.wree@med.uni-rostock.de

Abstract:
Approximately 25% of female infertility cases are due to fallopian tube (FT) occlusion, most commonly in the proximal segment. Transcervical catheterization (TCR) often results in re-occlusion. This study introduces a novel microstent-based implant for the proximal FT (uterotubal isthmus, UTI), addressing key biological, biomechanical, and biomedical factors.

Fallopian tubes (n=27) from 15 postmenopausal women were collected via salpingectomy or post-mortem donation. Micro-computed tomography (µCT) and X-ray microscopy (XRM) enabled morphometric analysis of the intramural-isthmic region, including luminal Feret diameter, fitting circle diameter, and mucosal folding. Self-expanding microstents were laser-cut from bioresorbable Poly(L-Lactic) Acid (PLLA). Preclinical ex vivo testing of the microstents and a self-developed micro-applicator was performed using porcine/lapine fallopian tubes.

The mean fallopian tube (FT) length was 75±15 mm, with the uterotubal isthmus (UTI) ranging from 20.0 to 39.0 mm. µCT/XRM analysis showed an outer isthmus diameter of 1.1±0.3 mm and luminal diameters below 1 mm. Microstents (1.2 mm outer diameter, 80 µm wall thickness, 10/20 mm length) were produced. Crimped microstents (0.8 mm diameter) were successfully loaded into a micro-applicator and implanted into porcine/lapine FTs. µCT confirmed precise apposition of the expanded microstent to the tubal lumen.

µCT-based 3D reconstruction and morphometric analysis enhance the understanding of fallopian tube morphology and provide critical insights into the geometric requirements for a microstent-based implant to treat proximal tubal occlusions. However, extensive technological research and development will be required before the fallopian tube microstent can be implemented as a medical device.

Poster 56:

Title:
Dermatoglyphic patterns in population of the R. of North Macedonia

Authors:
Elizabeta Chadikovska (Skopje, North Macedonia)
Ace Dodevski (Skopje, North Macedonia)
Anamarija Paunkoska (Skopje, North Macedonia)
Jovana Kordovska (Skopje, North Macedonia)
Julija Zhivadinovik (Skopje, North Macedonia)
Niki Matveeva (Skopje, North Macedonia)
Biljana Zafirova (Skopje, North Macedonia)
Biljana Bojadzieva Stojanoska (Skopje, North Macedonia)
Biljana Trpkovska (Skopje, North Macedonia)
enikovska@yahoo.com

Abstract:
Dermatoglyphics is the scientific study of fingerprints from the hands and fingers, soles and toes.

Dermatoglyphic study has been conducted in order to determine and analyze individual dermatoglyphic characters in healthy individuals with Macedonian and Albanian nationality.

Some dermatoglyphic parameter on the 210 palm prints were read and classified. The prints were obtained from healthy individuals of Macedonian and Albanian ethnic origin. Palm prints were taken using Cummins and Midlo's ink method .Dermatoglyphics were classified according Henry's system.

The results in our population study showed that UL>Wc>We>DL>A>RL on both hands in all participants, radial loops are present only on the second digit, index and double loops are most present as complex patterns. Formula for present triradii is a>t>b>d>c. The values for Atd angle are equal from 35˚-55˚.Most often found in hypothenar region are distal loops.

In conclusion, a dermatoglyphic patterns on the hands and fingers in population in the R. of North Macedonia have been made. Normal variability of the dermatoglyphic patterns have been presented. The results provide new possibilities for further investigation of dermatoglyphics and their biological and genetic properties. Findings from our populational study can be compared with the results of other nationalities, as well as presenting a control group for comparison with dermatoglyphic patterns in many genetical disorders and different diseases.

Poster 57:

Title:
Remodelling of the alveolar capillary network in unaffected appearing lung regions in human idiopathic pulmonary fibrosis

Authors:
Luca M. Lautenschläger (Hannover, Germany)
Christoph Wrede (Hannover, Germany)
Jonas Labode (Hannover, Germany)
Lars Knudsen (Hannover, Germany)
Julia Schipke (Hannover, Germany)
Christian Mühlfeld (Hannover, Germany)
Lautenschlaeger.Luca@mh-hannover.de

Abstract:
Idiopathic pulmonary fibrosis (IPF) is a lung disease that is characterised by progressive scarring of lung tissue and impairs patient's quality of life greatly.

Ongoing investigations mainly focus on fibroblasts and alveolar epithelial cells, whilst the role of capillary endothelial cells and the morphology of the alveolar capillary network (ACN) remain elusive.

IPF-tissue samples of human upper lobes (n=4) and control samples (tumour resectates, n=2) were prepared for and visualized by serial block-face scanning electron microscopy and transmission electron microscopy (TEM). Capillary lumina within generated image stacks were segmented manually using Microscopic Image Browser. 3D-models were rendered using 3dmod. TEM sections were used for design-based stereology.

Stereological analyses of IPF samples revealed no obvious signs of fibrosis like extracellular matrix deposition or metaplasia of alveolar epithelial type 2 cells – the septal ultrastructure seemed unaffected. However, the ACN morphology was altered already: blunt ending capillaries, bulging ends within the ACN and holes, disrupting the sheet-flow-like structure as well as rarefaction of the ACN as a whole were observed.

Remodelling of the ACN is already present in otherwise healthy appearing upper lobe regions. The mechanisms driving this remodelling remain elusive and thus need further investigation to elucidate, whether the endothelium and the ACN are driving IPF progression or are early victims of fibrotic remodelling.

Poster 58:

Title:
Anatomical and Clinical Criteria for Selecting Safe Zones for Submucosal Infiltration Anesthesia at the maxilla and mandible

Authors:
Nataliya Ivanova (Moscow, “Russia”)
Sergey Dydykin (Moscow, “Russia”)
Yuri Vasilev (Moscow, “Russia”)
Ekaterina Dyachkova (Moscow, “Russia”)
Alexey Kuznetsov (Moscow, “Russia”)
ivanova_n_d_@staff.sechenov.ru

Abstract:
To develop anatomical and clinical criteria for selecting safe zones for infiltration anesthesia at the maxilla and mandible.

1. Experimental Study (N=30, unfixed cadavers):

- Layer-by-layer dissection with vascular contrast enhancement.

- Craniometric analysis (facial/cranial indices, BMI).

2. Clinical Study (N=34 patients):

- Laser fluometry (LAKS-M device) to assess perfusion before and after anesthesia (2% and 4% articaine).

- EPT (Electric Pulp Testing) and VAS (Visual Analog Scale) for evaluating anesthetic efficacy.

1. Zones with minimal vascular density were identified as optimal injection points.

2. A statistically significant correlation was found between anesthetic efficacy, articaine concentration (2% vs. 4%), and injection site (p<0.05, Student’s t-test).

3. An algorithm for jaw bones safe zones selecting based on anatomical landmarks was developed.

1. A personalized approach to anesthesia, considering jaw morphometry, reduces complication risks.

2. The use of 4% articaine prolongs anesthesia duration by 15–20% (p<0.01).

3. Laser fluometry is a promising method for intraoperative perfusion monitoring.

Poster 59:

Title:
The Prevalence and Factors Associated with Biliary Tree Anatomic Variations: A Cross-Sectional Study

Authors:
Wafaa Mahmoud (Irbid, Jordan)
washunnaq@just.edu.jo

Abstract:
Anatomical variations in the intrahepatic (IHD) and cystic ducts (CD) are common, and their identification is essential for preoperative planning. This study assesses the prevalence of biliary ductal variations using magnetic resonance cholangiopancreatography (MRCP) and explores their interrelationship and potential associations with common biliary disorders.

A retrospective analysis of 259 patients who underwent MRCP was conducted, with demographic and clinical data retrieved from the hospital database. IHD and CD variations were classified through image analysis. Associations with biliary stones, cholecystitis, and obstructive jaundice were evaluated using Pearson's χ^2 test and multivariable logistic regression.

IHD variations were observed in 38.6% of cases, most commonly the trifurcation pattern (22%), followed by drainage of the right posterior duct into the common hepatic duct (8.5%) or the left hepatic duct (7.7%). CD variations were observed in 36.7% of cases, with medial anterior (17.8%) or posterior (18.9%) spiral insertions. IHD and CD variations were significantly associated ($p = 0.010$). CD variations were associated with an increased presence of biliary stones ($p = 0.023$) and cholecystitis ($p = 0.048$). In multivariable analysis, IHD variation (aOR = 1.95; 95% CI: 1.13–3.35; $p = 0.016$) and biliary stones (aOR = 1.79; 95% CI: 1.03–3.12; $p = 0.041$) independently predicted variants CD anatomy.

Anatomical variations of the intrahepatic and cystic ducts were common and strongly associated. Recognizing these variants is crucial for preoperative planning and may help anticipate complications, especially due to their link with stone formation and inflammation.

Poster 60:

Title:
The Buccal Fat Pad – An Underappreciated Structure? A Systematic Review of the Anatomy and Clinical Significance of Bichat's Fat Pad

Authors:
Michal Tulski (Wroclaw, Poland)
Alicja Florczak (WROCLAW, POLAND)
Aleksandra Krolikowska (Wrocław, Poland, Poland)
Michal Koterba (Wrocław, Poland, Poland)
Mateusz Mazurek (Wroclaw, Poland)
Pawel Surowiak (WROCŁAW, Poland)
Slawomir Wozniak (Wroclaw, Poland)
Zygmunt Domagala (Wroclaw, Poland)
zygmunt.domagala@umw.edu.pl

Abstract:
This study aimed to conduct a systematic literature review based on the AQUA criteria, with the goal of summarising current anatomical knowledge on the buccal fat pad (BFP) and its clinical relevance, particularly in the context of aesthetic and reconstructive procedures in the facial region.

A structured search was conducted in PubMed, Embase, and Scopus databases (August 2024), yielding 717 records. After applying exclusion criteria and assessing quality using the AQUA tool, 12 original anatomical studies were included for analysis. The review focused on the structure, anatomical variations, vascular supply, and topographical relationships of the BFP, with particular attention to the buccal extension.

The BFP consists of three lobes (anterior, intermediate, posterior) and four extensions (buccal, temporal, pterygoid, and pterygopalatine), each with distinct anatomical positioning and functional importance. It serves as a dynamic component of the midface, supporting muscle movement, shock absorption, and maintaining facial contour. Due to its proximity to facial nerves and the parotid duct, it is both a valuable and delicate structure in surgical practice.

The BFP is a complex, multifunctional anatomical structure. A thorough understanding of its anatomy may reduce surgical complications and improve outcomes in both reconstructive and aesthetic procedures. Further topographic and morphometric research is essential to better understand its variability and to optimise its clinical applications.

Poster 61:

Title:
Bronchopulmonary dysplasia reduces AE2 cells and their lamellar bodies in the rabbit lung

Authors:
Tabea Meier (Hannover, Germany)
Arne K. Schierz (Hannover, Germany)
Yannick Regin (Leuven, Belgium)
André Gie (Cape Town, South Africa)
Jaan Toelen (Leuven, Belgium)
Christian Mühlfeld (Hannover, Germany)
Julia Schipke (Hannover, Germany)
tabea.meier@stud.mh-hannover.de

Abstract:
Bronchopulmonary dysplasia (BPD) is a chronic lung condition correlated to preterm birth, with mechanical ventilation and oxygen therapy further increasing the risk. BPD is marked by simplified alveolar structures and impaired microvascular development. Alveolar epithelial type 2 (AE2) cells play a key role in maintaining lung function due to their ability to produce and store surfactant. Despite their importance, research on their alteration in BPD is limited. Therefore, this study examines how preterm birth and hyperoxia affect AE2 cells.

Control rabbits (CO) were born vaginally at term and kept under normoxia for 4 days. Preterm pups, delivered by caesarean 3 days before term, were assigned to 7 days of either normoxia (NOX) or hyperoxia (HYX; 95% oxygen). Lungs were perfusion-fixed with 1.5% paraformaldehyde/1.5% glutaraldehyde and analysed using stereology.

Preterm birth led to decreased volumes of the lung, parenchyma and AE2 cells, regardless of oxygen levels. Both NOX and HYX groups exhibited reduced lamellar body volumes within AE2 cells compared to CO, along with lower lamellar body volume per septal surface area. The combination of prematurity and hyperoxia caused a significant decline in AE2 cell number compared to CO and NOX.

Preterm birth profoundly impaired the maturation of AE2 cells, leading to reduced cell number, smaller mean volume and lower lamellar body volume, indicating decreased intracellular surfactant reserves. The combination of preterm birth and hyperoxia induced a significantly aggravated decrease in AE2 cell number and pulmonary surfactant reserves, suggesting an additional inhibitory effect of hyperoxia on AE2 cell proliferation and function.

Poster 62:

Title:
Sonographic anatomy of the pubic symphysis during pregnancy

Authors:
Slawomir Wozniak (Wroclaw, Poland)
Aleksandra Piatek (Wroclaw, Poland)
Bozena Kurc-Darak (Wroclaw, Poland)
Zygmunt Domagala (Wroclaw, Poland)
Friedrich Paulsen (Erlangen, Germany)
Jerzy Florjanski (Wroclaw, Poland)
slawomir.wozniak@umw.edu.pl

Abstract:
To better understand pubic changes during pregnancy pubic symphysis parameters were sonographically assed and analysed in pregnant women.

Sonographic measurements were done in 225 pregnant women aged between 23 and 41 years. The parameters included: the middle width of the pubic entry, measured at the midpoint of the entrance, the width of the pubic symphysis, measured at the midpoint of the symphysis, the distance between the right and left pubic tubercles, and the depth of the pubic symphysis, defined as the distance between its proximal and distal borders.

The measurements most strongly correlated with fetal weight included pubic symphysis entry width (6.5 ± 3.4 mm; $p<0.001$), pubic symphysis width (6.4 ± 2.9 mm; $p<0.001$), and depth (14.8 ± 4.8 mm; $p=0.03$). Intertubercular distance showed the strongest correlation with maternal age (15.1 ± 5.4 mm; $p=0.03$). In the singleton pregnancy group, pubic symphysis width was significantly correlated with fetal weight categories: ≤1000g (4.56 ± 1.5 mm; $p=0.02$), 1001-2000g (5.51 ± 2.6 mm; $p=0.02$), and >3000g (7.3 ± 3.9 mm; $p=0.02$). Pubic symphysis entry width also showed significant correlations with fetal weight ranges: 1001-2000g (5.5 ± 3 mm; $p=0.02$), and >3000g (7.4 ± 3.9 mm; $p=0.02$).

Higher fetal weight was associated with increased dimensions of the pubic symphysis entry and disc. Advanced maternal age correlated with greater intertubercular distance. Women with higher body weight and those who experienced substantial weight gain during pregnancy tended to have a wider pubic symphysis entry and disc.

Poster 63:

Title:
Still in the Flow – Long-Term Laparoscopic Insights into Body Donor Fixation

Authors:
Sebastian Streich (Tübingen, Germany)
Ruth Ladurner (Tübingen, Germany)
Lothar Just (Tübingen, Germany)
Bernhard Hirt (Tübingen, Germany)
sebastian.streich@uni-tuebingen.de

Abstract:
In previous work, we have established an easy-to-use protocol for laparoscopic evaluation of anatomical body donors during the fixation process. There, we were able to demonstrate substantial changes of the abdominal organs during the fixation process. Here, we aimed to extend the scope of this laparoscopic assessment over the post-fixation period up to three months post injectionem to document changes of the abdominal structures after injection.

The bodies, donated to our institute, were fixed by an intra-arterial infusion of an INNOBALM® fixation solution (LICIT Solutions, Tübingen, Germany). The tissue changes were monitored with a rigid endoscope via a laparoscopy using photo- and video documentation prior to fixation, during fixation, and after fixation at days 14, 28, 56, and 84.

Our current work provides novel insights into the long-term characteristics of INNOBALM®-fixed cadavers (n ≥ 50), focusing on the intraabdominal structures. Over the course of three months, we were able to demonstrate a reduction of the organ swelling, decreasing intraperitoneal fluid with residual deposits of material on the organ's surfaces, and changes in color ranging from life-like red to darker tones. All of these findings are subject to interindividual differences, indicating potential confounders like vascular status or post-mortal interval.

With our established laparoscopy protocol, we now can demonstrate long-term changes on the abdominal organs in anatomical body donors. These results can form the basis for future advantages in body donor fixation, leading to improved tissue quality and therefore enhanced teaching and training quality in dissection and surgical training courses.

Poster 64:

Title:
Vaso-Ruffini Complexes: Evidence of Stretch-Sensitive Proprioceptors in Human Blood Vessels

Authors:
Mohaddeseh Sangari (Düsseldorf, Germany)
Ali Mobasheri (Oulu, Finland)
Zhixiong Chang (Düsseldorf, Germany)
Timm Filler (Düsseldorf, Germany)
timm.filler@hhu.de

Abstract:
To investigate the existence, distribution, and structural characteristics of Ruffini-type mechanoreceptors in human arteries and veins and to consider their potential role in vascular regulation and pain.

Vessel-containing blocks from 34 adult donors (average age 69.5 years), spanning cranial to limb regions, were processed for histology and immunohistochemistry. Serial sections were stained with hematoxylin-eosin, picrosirius red, and antibodies against tyrosine hydroxylase, S-100, and PGP-9.5. Digitized slides were examined for corpuscles, orientation, size, vessel caliber, and autonomic fibers.

Spindle-shaped Ruffini corpuscles were consistently found in the adventitia of arteries and veins with a lumen diameter of ≥ 300 µm. They oriented transversely to the vessel axis, lacked a complete capsule, and contained intertwined capillaries, Schwann cells, and low-density collagen. Corpuscles were innervated by unmyelinated, tyrosine hydroxylase-positive fibers independent of their intrinsic vasculature, while additional sympathetic varicosities were present in the adjacent adventitia. Individual complexes extended several millimeters in large vessels; none were observed in coronary arteries. The morphology and distribution indicate high sensitivity to tonic stretch and regional blood-volume changes.

We provide the first description of “Vaso-Ruffini complexes” as a widespread afferent interface within human vasculature. Their strategic position and dual association with sympathetic fibers support a role in fine-tuning local perfusion, baroreflex-independent homeostasis, and vascular pain mechanisms. Recognition of this sensory network may inform anatomical risk assessment in filler injections and inspire targeted neuromodulatory or pharmacological interventions for vascular dysregulation and pain.

Poster 65:

Title:
Bilateral Duplex Kidney – Case report from a body donor and implications for clinical practice

Authors:
Katharina Jüngert (Erlangen, Germany)
Fabian Frank (Erlangen, Germany)
Philipp Arnold (Erlangen, Germany)
katharina.juengert@fau.de

Abstract:
During anatomical dissection course, we identified an 80-year-old female body donor with a bilateral duplex kidney and ureter that remained undiagnosed throughout her lifetime, despite a total rectal excision. Studying the anatomical conditions in this case, embedded in the medical history, was of great interest.

The urinary tract was fully dissected considering positional relationships to surrounding structures. The extensive clinical history provided by the attending GP was carefully analyzed. Embryogenesis of duplex kidneys, anatomical variations, and prevalence of symptomatic cases were assessed via literature review.

Both kidneys have an upper and lower renal pelvis. The upper renal pelvis drains into the medial ureter and the lower renal pelvis into the lateral ureter. Both left ureters run parallel through the pelvis, but the right lateral ureter crosses under the medial ureter, creating an additional potential constriction. Both sides open on the dorsal side of the bladder. In general, duplex kidneys occur in 0.8% of humans unilaterally and in 0.15% bilaterally. Ureters can unite or run separately. If separately, one ureter can open ectopically with often an impaired associated kidney.

Duplicated ureter and kidney formation is characterized by a high degree of variability and does not necessarily have to become apparent during life time. Nevertheless, they have the potential to impose problems. Manifestation is mostly during childhood, presenting with fever, pain, and sometimes loss of kidney function. Undiagnosed anomalies may increase the risk of ureteral injury during abdominal and pelvic surgery. Greater awareness can be achieved by demonstrating cases in dissection courses.

Poster 66:

Title:
Julius Planer (1827–1881): Anatomist, Educator, Pioneer – The Lviv–Graz Link in 19th-Century Medical Science

Authors:
Uliana Pidvalna (Lviv, Ukraine)
Lesya Mateshuk-Vatseba (Lviv, Ukraine)
Veronica Antipova (Graz, Austria)
Niels Hammer (Graz, Austria)
upidvalna@gmail.com

Abstract:
We examine the academic and scientific contributions of Professor Julius Planer von Plann (1827–1881), focusing on the periods in Lviv (modern Ukraine) and Graz (modern Austria), and evaluate his legacy in the development of anatomical education and research in Central Europe in the 19th century.

The study followed a historical-biographical approach, analyzing archival materials from Ukraine, Poland, and Austria, as well as published scientific works. Primary sources include Planer's biography, documents from the academic authorities of the Imperial and Royal University of Lviv, including the lecture schedule for the summer semester of 1862/1863, and secondary literature on the development of anatomy in the Habsburg Empire.

Planer, born in Vienna, graduated from the University of Vienna (1845–1849). As an assistant to the renowned Professor Carl von Rokitansky (1804–1878), Planer received the title of professor in 1851. He described pigmented granules in blood, likely related to malaria. From 1855 to 1863, he was head of the Department of Anatomy at the Medical-Surgical Institute in Lviv. As prominent investigation he described the cholesterol crystallization, later recognized as the first description of liquid crystal behavior. He revitalized the collection of physiological and anatomical specimens in Lviv. Moving to the University Graz of in 1863, he became head of Anatomy Department (1863-1881).

Julius Planer significantly shaped anatomical education and scientific research in Lviv and Graz. His interdisciplinary legacy, encompassing macroscopic anatomy and biophysical discoveries, reflects a fundamental period in modern European medicine, with institutional developments and scientific insights that continue to resonate today.

Poster 67:

Title:
Topology of the Lateral Cutaneous Branch of the Iliohypogastric Nerve and Simulation of Ultrasound-Guided Nerve Block in Human Body Donor Cadavers

Authors:
Paata Pruidze (Vienna, Austria)
Tobias Rossmann (Linz, Austria)
Karoline M. Schwendt (Vienna, Austria)
Anton Liederer (Baden, Austria)
Helmut Rauschka (Vienna, Austria)
Wolfgang J. Weninger (Vienna, Austria)
Stefan Meng (Vienna, Austria)
paata.pruidze@meduniwiena.ac.at

Abstract:
Our study aims at providing topologic information on the lateral cutaneous branch of the iliohypogastric nerve (LCBIN) and to evaluate the accuracy of simulated ultrasound guided blockade of this nerve.

Twenty-four randomly selected fresh, non-frozen human body donor cadavers were included. They were positioned first on their right and then on their left sides and a Canon Aplio i800 ultrasound apparatus with high-resolution linear transducers (i18LX5 and i22LH8) was used to bilaterally identify the LCBIN at the level of the iliac crest. Once identified, small ink depots were injected close to the LCBIN. Then the nerves and ink depots were exposed by anatomical dissection. The topology of the LCBINs and the ink depots were documented and the distance between the anterior superior iliac spine (ASIS) and the nerve's crossing of the iliac crest were measured.

In all examined cadavers, ultrasound allowed to identify the left and right sided LCBINs. In 44 out 48 cases, the nerve was stained with ink. The mean distance between LCBIN and ASIS as measured along the iliac crest was 10.4cm (left: 10.3cm; right: 10,5cm). In 50% the nerve was crossing the iliac crest inside an osteofibrous tunnel.

We provide metric information on the position of the LCBIN along the iliac crest and demonstrate that ultrasound guided blockade of the LCBIN is successful in 91.7%. This information is the basis for using cost-effective LCBIN blockade as a routine tool for differential diagnosis of LCBIN neuropathies and greater trochanteric pain syndrome (GTPS).

Poster 68:

Title:
Prospective, randomised study on application intra-arrest ventilation using THIEL-fixed human body donors

Authors:
Justin Trenkel (Bochum, Germany)
Beate Brand-Saberi (Bochum, Germany)
Tamar Gelashvili (Minden, Germany)
Annika Hoyer (Bielefeld, Germany)
Lydia Johnson Kolaparambil Varghese (Minden, Germany)
Vanessa Kühn (Hamburg, Germany)
Jonas Lohmann (Bielefeld, Germany)
Christian Neuhaus (Hamburg, Germany)
Claudia Schneider (Bochum, Germany)
Mahsa Ullrich (Hamburg, Germany)
Jochen Hinkelbein (Minden, Germany)
Gerrit Jansen (Minden, Germany)
justin.trenkel@ruhr-uni-bochum.de

Abstract:
This study investigates the effects of endotracheal intubation (ETI) compared to the IGEL-laryngeal-mask (IGEL) during intra-arrest-ventilation (IRV) with Intermittent-Positive-Pressure-Ventilation (IPPV) using a prospective randomised study in THIEL-fixed human body donors.

The study is approved by the ethics committee of RUHR UNIVERSITY BOCHUM in OSTWESTFALEN-LIPPE (Ref.2024-1183). The body donors were fixed according to THIEL, as the only commonly used fixation method that preserves the flexibility of the tissue. The donors were first bronchoscoped and their lungs recruited. Airway management with IGEL laryngeal masks and endotracheal intubation was then performed in randomised order. Ventilation was performed with IPPV (Vt=7mL/kg ideal body weight, respiratory rate=10/minute, positive-end-expiratory-pressure=3mbar, peak pressure=40mbar) via MEDUMAT STANDARD-2, WEINMANN-EMERGENCY-MEDICAL-TECHNOLOGY. Chest compressions were performed by CORPULS CPR. The primary endpoint was the difference between the ideal and the actually applied Vt (Vtdiff). Secondary endpoints were the expiratory Vt (Vte), the leakage volume (Vleak) and the peak pressure (Ppeak).

A total of eleven body donors were included.

The measured values show that the IGEL laryngeal mask has a significantly higher difference between the applied volume and the measured expiratory tidal volume compared to endotracheal intubation. This results in a significantly higher leakage volume. At the same time, much lower pressures can be measured under the IGEL laryngeal mask.

Emergency medicine studies on Thiel-fixed body donors lead to results that can be used to validate clinical techniques. Intra-arrest ventilation with an IGEL laryngeal mask shows a significantly higher leakage and thus a much lower potential volume exchange than intra-arrest ventilation with endotracheal intubation.

Poster 69:

Title:
The spinal Cord Angiosome of the Descending Thoracic Aorta

Authors:
Paata Pruidze (Vienna, Austria)
Giorgi Didava (Vienna, Austria)
Jeremias T. Weninger (Vienna, Austria)
Stefan H. Geyer (Vienna, Austria)
Christoph Neumayer (Vienna, Austria)
Josif Nanobachvili (Vienna, Austria)
Wolf Eilenberg (Vienna, Austria)
Martin Czerny (Freiburg, Germany)
Wolfgang J. Weninger (Vienna, Austria)
paata.pruidze@meduniwien.ac.at

Abstract:
In order to provide anatomic basic data for save aortic surgery our study aims at providing information on the extension of interspinal angiosomes of the descending thoracic aorta (DTA).

The thoraces of 8 cadavers (post-mortem times <15hours) were opened and the aortae were fully exposed. The branching patterns of the segmental arteries were documented. Then the ascending aorta and the DTA, distal to the left subclavian artery and at the level of T11, were cross clamped and the three separated segments were simultaneously perfused with eosin and methylene blue solutions. Finally, the cadavers were placed in prone position and the spinal canals were opened. The Spinal cord (SC) was extracted and the intraspinal vascular situation and extension of the spinal cord angiosomes were analyzed.

The cranial border of the SC angiosome varied between C7 and T4; the caudal border between T9 and the conus medullaris. In 5 specimens the anterior and posterior borders did not match. Those specimens with stained conus medullares featured voluminous Adamkiewicz arteries, which joined the anterior spinal artery between segments T8 and L2. The spinal artery was always filled with dyed liquid along its entire cranio-caudal extension. Variations in segmental arteries reflected in the staining patterns of the SC and nerve roots.

Our study provides detailed information on the SC angiosome of the DTA. It demonstrates the high potential of collateralization via the anterior spinal artery and highlights they key role of the Adamkiewicz artery in supplying the SC.

Poster 70:

Title:
Variants of the renal arterial supply and their clinical relevance

Authors:
Leopold Braun (Tübingen, Germany)
Ruth Ladurner (Tübingen, Germany)
Sebastian Streich (Tübingen, Germany)
Bernhard Hirt (Tübingen, Germany)
ruth.ladurner@uni-tuebingen.de

Abstract:
The normal anatomy of the renal arterial supply is defined as a single renal artery from the aorta that divides into segmental arteries in the renal hilum. The aim of this study was to determine the frequency of deviating variants of the renal arterial supply in body donors and to evaluate their clinical relevance.

In 22 body donors the frequency of renal artery variants, the occurrence of polar arteries from the main trunk of the renal artery or from the aorta, the gender prevalence, the side comparison of the renal arterial supply and their clinical relevance in comparison with the literature were investigated.

Normal anatomy was present in 12 of 41 kidneys. Deviations were found in 29 of 41 kidneys. 15 normal variants were found in right kidneys, 14 in left kidneys. 15 of 20 right and 14 of 21 left kidneys had an abnormal arterial supply. In total, 17 of the 22 body donors had a normal variant on at least one side: 8 of 11 male, 9 of 11 female donors.

In partial nephrectomy variants play no role, but in kidney transplantation they are associated with a longer “warm ischemia time” and ureter-associated complications. In the treatment of refractory arterial hypertension, renal denervation is more ineffective in the presence of multiple renal arteries.

Knowledge of multiple renal arteries is helpful in renal transplantation, partial nephrectomy and renal denervation for hypertension.

Poster 71:

Title:
Left-right symmetry breaking in the mesentery of the foregut for bursa omentalis formation

Authors:
Vanessa Tourbier (Göttingen, Germany)
Christoph Viebahn (Göttingen, Germany)
Nikoloz Tsikolia (Göttingen, DE)
nikoloz.tsikolia@med.uni-goettingen.de

Abstract:
The asymmetric arrangement of the viscera is commonly explained by the stepwise rotation of the mesenteries, which is due to asymmetry of the mechanical and molecular properties of mesenteric tissue. Although this view is not primarily based on observation, but rather on convincing deductions, and has been criticized repeatedly, most textbooks still adopt it. In view of the repeated criticism of the rotation concept, we decided to study the initial steps of symmetry breaking in human as well as in the chick, which has repeatedly been used as a model for asymmetric gut rotation.

Serial sections were analyzed and used for 3D reconstructions. In addition we studied topographical arrangement in chicken embryo using micro CT and localization of gene expression using mRNA in situ-hybridisation.

Our histological study of the development of the foregut suggests that the initial steps of left-right symmetry breaking involve asymmetric morphogenesis by differential activity of the columnar coelomic epithelium covering the (broad) dorsal foregut mesentery.

Our results reveal, indeed, another case of asymmetric development in the coelomic epithelium of the developing omental bursa and (preceding bilateral) pneumatoenteric recesses, further challenging the prevailing view of asymmetrical properties in the mesenteric tissue leading to gut rotation and, in particular, the development of asymmetries in the foregut.

Poster 72:

Title:
Chemo-Sensitizing effects of Punicalagin and OGT-inhibitors in Osteosarcoma cell lines and patient tissue in a 3D-in-vivo-tumor-model

Authors:
Sabrina Steudle (Regensburg, Germany)
Silke Haerteis (Regensburg, Germany)
Thiha Aung (Deggendorf, Germany)
sabrina.steudle@stud.uni-regensburg.de

Abstract:
Osteosarcomas are the most common primary malignant bone tumors in children and adolescents. Metastasis and the emergence of chemoresistance pose significant therapeutic challenges. This study aims to evaluate the potential chemo-sensitizing effects of Punicalagin and the OGT inhibitors OSMI-1 and OSMI-4 in combination with doxorubicin in parental and doxorubicin-resistant osteosarcoma cell lines and tumors.

Preliminary MTT assays have been performed to evaluate the compounds both individually and in initial combination setups. To test their efficacy, cell-line derived, as well as primary patient-derived tissue tumors will be assessed in the 3R (reduce, replace, refine)-compliant chorion-allantois-membrane (CAM)-based 3D-in-vivo-tumor-model. The cell lines used include MG-63, SaOS-2, and 143B cell lines (parental and resistant. Tumor development and treatment effects will be analysed via LASCA imaging, ultrasound, and histological staining (H&E and Caspase-3).

Initial viability data from MTT assays indicate a promising effect of Punicalagin and OGT inhibitors on both resistant and parental cell lines when used as single substances. As these tests confirm the expected findings, a full analysis of the combination effects and in vivo application is currently in preparation.

Punicalagin and OGT inhibitors are expected to enhance doxorubicin responsiveness. The findings offer promising new combination strategies to overcome chemoresistance in osteosarcoma.

Poster 73:

Title:
Fat & Sugar: Glucose metabolism in murine adipose tissue explants

Authors:
Laura Müller (Leipzig, Germany)
Nora Raulien (Leipzig, Germany)
Martin Gericke (Leipzig, Germany)
nora.raulien@medizin.uni-leipzig.de

Abstract:
In adipose tissue (AT) the abundant fatty acids are used for energy generation in cells. Despite this substrate, macrophages need glucose for survival. We aimed to analyze the role of glucose in organotypic AT cultures, with focus on macrophages

Murine white AT was cultured as explants for 5 to 7 days. After treatment with different glucose concentrations AT was visualized by confocal microscopy, stromal cells were isolated and stained for flow cytometry or cultured with a fluorescent glucose analog (2-NBDG; 30min). Glucose and lactate concentrations were analyzed in the supernatant.

AT explants need a minimum of 5mM glucose for survival, despite an abundance of fatty acids. During 7d of organ culture glucose is taken up by GLUT-1, metabolized and a main portion is released as lactate in the supernatant. Interestingly lactate release is diminished, when cells were treated with insulin, while glucose-uptake increases. Analysis of macrophages, which actively take up glucose on day 5 (NBDG+) shows a higher expression of the marker CD11c and CD301 compared to NBDG- cells. At day 7, the overall uptake of glucose diminishes and the CD11c expression in NBDG+ cells remain high, whereas CD301 is significantly lowered.

Adipose tissue explants highly depend on extracellular glucose, taken up via GLUT-1. The release of large lactate amounts indicates an anaerobic metabolism and supplementation of insulin suspends this lactate release. Furthermore, there are phenotypic differences between NBDG+ and NBDG- macrophages. Therefore, cellular metabolism can influence the inflammatory status of AT and could be used to study AT inflammation.

Poster 74:

Title:
Cystinyl leukotrienes are indispensable to the tuft cell-induced neurogenic inflammation, but promote the bacterial killing capacity of airway macrophages

Authors:
Paula Gehrich (Homburg, Germany)
Noran Abdel Wadood (Homburg, Germany)
Amanda Wyatt (Homburg, Germany)
Markus Bischoff (Homburg, Germany)
Thomas Gudermann (Munich, Germany)
Vladimir Chubanov (Munich, Germany)
Ulrich Boehm (Homburg, Germany)
Mohamed Elhawy (Homburg, Germany)
Gabriela Krasteva-Christ (Homburg, Germany)
mohamed.elhawy@uks.eu

Abstract:
Tuft cells induce neurogenic inflammatory responses in the trachea. While these effects are mediated by the neuropeptide calcitonin gene-related peptide (CGRP), the involvement of additional tuft cell mediators, such as cysteinyl leukotrienes (CysLTs) in these responses remains unclear. Here, we studied the immunological functions of CysLTs generated by activated tuft cells in the context of pneumonia.

Tuft cells were activated by denatonium following blocking of CysLT receptor 2 by HAMI3379 . Evans blue fluorescence in tracheal tissue sections was quantified to estimate plasma extravasation. Neutrophil recruitment to the trachea was analyzed by immunohistochemistry. Blood neutrophil counts were determined by differential cell staining of blood smears. To investigate the antimicrobial effects of CysLTs, we performed bacterial killing assays with tracheal and lung interstitial macrophages isolated by FACS sorting. Before infection with the Pseudomonas aeruginosa strain NH57388A, macrophages were incubated with tracheal supernatants from Trpm5+/+ or Trpm5-/- mice stimulated with denatonium. CysLT synthesis was inhibited by preincubating tracheae with MK-886 before denatonium application.

Treatment of mice with HAMI3379 did not affect the dose-dependent increase in plasma extravasation following tuft cell activation. Similarly, there was no change in neutrophil counts in the tracheae of HAMI3379-treated mice compared to the control (PBS-treated) group. Remarkably, the bacterial killing capacity of tracheal and lung interstitial macrophages increased after incubation with supernatants from stimulated Trpm5+/+ tracheae and was decreased upon the depletion of leukotrienes by MK-886.

Leukotriene signaling impacts tuft cell-mediated immune responses by affecting the antibacterial capacity of macrophages.

Poster 75:

Title:
The killing efficacy of natural killer cells against pancreatic cancer cells is strongly sex-dependent

Authors:
Elise Arlt (Halle, Germany)
Nadine Bley (Halle, Germany)
Theresa Dautz (Halle, Germany)
Sandra Hauser (Dresden, Germany)
Jens Pietzsch (Dresden, Germany)
Thomas Liehr (Jena, Germany)
Heike Kielstein (Halle, Germany)
Ivonne Bazwinsky-Wutschke (Halle, Germany)
elise.arlt@uk-halle.de

Abstract:
Sex differences are known to influence immune responses and the progression of pancreatic cancer. This study investigates the sex-dependent cytotoxicity of natural killer (NK) cells against pancreatic cancer cells to better understand underlying immune mechanisms relevant for targeted therapies.

Primary NK cells were isolated from the spleen of male and female mice. Their cytotoxicity was assessed in vitro using co-cultures with GFP-labeled pancreatic cancer cells. Killing efficacy was quantified using flow cytometry and live-cell imaging. In addition, transcriptomic analyses provided molecular insights into sex-based functional differences. In vivo, both healthy and genetically modified mice of both sexes predisposed to pancreatic cancer were used to examine NK cell function and tumor development under physiological conditions.

NK cells from female mice showed significantly higher cytotoxic activity against pancreatic cancer cells than those from males. However, in vitro as well as in vivo, tumor cells were able to reduce NK cell activity, indicating a mechanism of immune evasion. These results demonstrate a clear sex-specific pattern in the antitumoral immune response, with female NK cells showing greater potential to control tumor growth, but also reveal the capacity of tumors to modulate immune cell function regardless of sex.

This study highlights pronounced sex differences in immune surveillance of pancreatic cancer cells. Considering both hormonal and immunological sex-specific factors is crucial for developing effective and individualized immunotherapeutic strategies for pancreatic cancer. Understanding these mechanisms may improve the design of future treatments and lead to better patient outcomes.

Poster 76:

Titel: Tracheal tuft cells prime acute neutrophilic inflammation to combat bacteria at earlier stages of infection

Authors:
Maria Grammer (Homburg, Germany)
Giuseppina Sole Lanzilli (Homburg, Germany)
Noran Abdel Wadood (Homburg, Germany)
Amanda Wyatt (Homburg, Germany)
Markus Bischoff (Homburg, Germany)
Thomas Gudermann (Munich, Germany)
Vladimir Chubanov (Munich, Germany)
Ulrich Boehm (Homburg, Germany)
Mohamed Elhawy (Homburg, Germany)
Gabriela Krasteva-Christ (Homburg, Germany)
mohamed.elhawy@uks.eu

Abstract:
Tracheal tuft cell activation has been recently reported to modulate immune responses. These reactions are dependent either on acetylcholine released in response to bacterial metabolites in the early phase of bacterial infection or on ATP-signaling in later stages of infection. Here, we describe the role of tuft cells in inducing acute immune responses during Pseudomonas aeruginosa infection.

Bacterial supernatants from the cystic fibrosis P. aeruginosa strain NH57388A were collected after 4 h, representing the early logarithmic growth phase. Supernatants were applied intratracheally in transient receptor potential cation channel subfamily M member 5-deficient (Trpm5-/-) and wild-type mice (Trpm5+/+), following retroorbital injection of Evans Blue (EB) to assess vascular leakage. After 30 min, we collected bronchoalveolar lavage fluid (BALF), blood, and tracheal samples. Neutrophil quantification was performed by immunohistochemistry on tracheal tissue sections. Immune cell populations in BALF and peripheral blood were analyzed by flow cytometry (FACS), respectively.

Application of bacterial supernatants induced an enhanced neutrophil recruitment to the tracheae in Trpm5+/+ mice compared to Trpm5-/- mice. In $Trpm5^{-/-}$ mice, neutrophils accumulated in the bloodstream but did not transmigrate into the site of inflammation, consistent with impaired tissue recruitment. This defect occurred independently of plasma leakage, which remained unaltered. Additionally, BALF analysis revealed increased counts of leukocytes, neutrophils, macrophages, monocytes, and lymphocytes in the bacterial supernatant-treated Trpm5+/+ group when compared to Trpm5-/- mice.

Our findings suggest that TRPM5-dependent signaling in tracheal tuft cells mediates early immune responses to bacterial metabolites and influences immune cell composition in both tracheal and alveolar tissues.

Poster 77:

Title:
Where T cells, B cells, and Antigen-presenting cells (APCs) meet - M cells and their VIP Lounge

Authors:
Vladimir Ginoski (Magdeburg, Germany)
Jose Luis Cortés Sánchez (Magdeburg, Germany)
Stefan Kahlert (Magdeburg, Germany)
Hermann-Josef Rothkötter (Magdeburg, Germany)
vladimir.ginoski@med.ovgu.de

Abstract:
M cells (microfold cells) are specialized epithelial cells within the follicle-associated epithelium (FAE) of Peyer's patches. A hallmark of these cells is the presence of basolateral pockets—invaginations facing the underlying lymphoid tissue. It is hypothesized that these pockets are occupied by B cells, T cells, and professional antigen-presenting cells (APCs), enabling immediate interaction with transcytosed antigens and promoting mucosal immune surveillance.

Jejunal tissue samples were collected from piglets aged 42 and 56 days, cryosectioned at 5 µm thickness, and processed for indirect immunofluorescence. Primary antibodies against CD3 (T cells), CD21 (B cells), CD45 (B cells/leukocytes), and MHC class II (professional APCs) were used. M cells in the FAE were labelled using cytokeratin 18, and nuclei were counterstained with DAPI. Immunostained sections were analyzed by confocal laser scanning microscopy and widefield fluorescence microscopy to evaluate the spatial distribution of immune cell subsets and their localization relative to M cells and basolateral pockets.

$CD3^+$ T cells, $CD45^+$ B cells/leukocytes, and MHC class II^+ APCs were identified within basolateral invaginations of M cells in Peyer's patches, highlighting their integral role in forming a functional immune microenvironment for antigen sampling and presentation. $CD21^+$ B cells were not present in the M cell pockets.

The basolateral pockets of M cells function as immunological synapse hubs within Peyer's patches, where T cells, B cells, and professional APCs converge in strategic proximity to enable rapid antigen sensing, efficient presentation, and finely tuned mucosal immune reactions.

Poster 78:

Title:
Application of a breast cancer spheroid model to evaluate the tumor microenvironment and its impact on immune cells

Authors:
Jonas Brümmel (Halle (Saale), Germany)
Andrea Kindermann (Halle (Saale), Germany)
Heike Kielstein (Halle (Saale), Germany)
jjbruemmel@mail.de

Abstract:
Breast cancer is the most common type of cancer among women in Germany, accounting for 32% of all cases. For variants that do not respond to hormone receptor therapy (triple-negative), the 5-year survival rate drops from over 90% to 80%. Furthermore, tumor cells can inhibit the body's immune response, for example, through T-cells. Three-dimensional spheroids can better simulate the tumor microenvironment closer to in-vivo conditions than conventional monolayer cultures. The objective of this study is to investigate how the secretory profile and gene expression of various breast cancer cell lines affect the function of T-cells.

Two different breast cancer cell lines, MCF-7 (ER positive, PR and HER2 negative) and MDA-MB-231 (ER, PR and HER2 negative), were cultured into spheroids using liquid-overlay technique. Cytokine analysis of the microenvironment and gene analyses were used to reveal differences between 2D and 3D models. Additionally, co-cultures with peripheral T-cells will enable the analysis of cell-cell interactions.

Compared to monolayer, spheroid culture led to significant changes in the expression of genes involved in cholesterol metabolism and immune-relevant genes, including cytokines such as IL-6 and GCSF. MDA-MB-231 cells overall exhibit a more immunosuppressive cytokine profile than MCF-7. The co-cultures for evaluating T-cell activity are work in progress.

3D-models differ in gene and cytokine expression from conventional monolayer cultures and therefore react differently to T-cells in co-culture. Those results can give a better understanding of in-vivo conditions and could lead to the identification of new therapeutic approaches that specifically interrupt immunosuppressive signaling pathways.

Poster 79:

Title:
Mitochondrial Cardiolipin Remodeling Regulates Metabolic Adaptations in Macrophages During Inflammatory Stimulation

Authors:
Katharina Ermer (Würzburg, Germany)
Manuela Erk (Würzburg, Germany)
Werner Schmitz (Würzburg, Germany)
Vasco Sequeira (Würzburg, Germany)
Srikanth Karnati (Würzburg, Germany)
Martin Väth (Würzburg, Germany)
Christoph Maack (Würzburg, Germany)
Jan Dudek (Würzburg, Germany)
Ermer_K@ukw.de

Abstract:
Macrophages orchestrate immune responses following cardiac injury by transitioning between pro- and anti-inflammatory states, each requiring distinct metabolic programs. Mitochondria play a central role in this regulation, with cardiolipin (CL) being critical for mitochondrial function. Barth Syndrome (BTHS), caused by TAFAZZIN mutations, impairs CL remodeling, leading to mitochondrial dysfunction and cardiomyopathy. While CL's role in mitochondrial health is well known, its impact on macrophage polarization is poorly understood.

We isolated bone-marrow derived macrophages (BMDM) from wildtype and Taz-/- mice and treated them with lipopolysaccharide (LPS) and interleukin 4 (IL-4) to either promote a pro- or anti-inflammatory state, respectively. We used nigericin alongside LPS to stimulate the assembly and activation of the NLRP3 inflammasome. Lipid composition was assessed via mass spectrometry. Mitochondrial structure and function, NLRP3 inflammasome activation, and cytokine secretion were evaluated using Seahorse analysis, transmission electron microscopy, RNA-seq, qPCR, Western blot, and ELISA.

Taz-/- BMDMs lacked mature CL and failed to adjust lipid composition upon stimulation. LPS-treated Taz-/- BMDMs showed increased glycolysis and altered cristae structure, suggesting a shift away from oxidative phosphorylation. Despite these metabolic changes and upregulation of mitochondrial genes, cytokine expression and secretion in response to LPS or IL-4 were unaffected.

Although CL remodeling did not alter cytokine secretion, our study reveals its pivotal role in regulating metabolic adaptations in BMDM during inflammatory stimulation. These insights offer foundational knowledge for the development of CL-driven pathway treatments aiming to modulate macrophage function in cardiomyopathies or heart failure diseases.

Poster 80:

Title:
BTNL1 receptor guiding the γδ T-cells in the intestinal porcine gut

Authors:
Jose Luis Cortés Sánchez (Magdeburg, Germany)
Vladimir Ginoski (Magdeburg, Germany)
Stefan Kahlert (Magdeburg, Germany)
Hermann-Josef Rothkötter (Magdeburg, Germany)
jose.cortes@med.ovgu.de

Abstract:
Gamma delta T cells are a special population of T cells that exhibit characteristics of both adaptive and innate immunity. They are highly abundant in the intestinal epithelium and lamina propria of humans. They play an important role in gut immunity, including functions such as fighting infections, tumour surveillance, tissue repair, maintaining homeostasis, and regulating autoimmunity and inflammation. In humans, the location of this T cell subtype is determined by the expression of Butyrophilin-like proteins (BTNLs) and other tissue-specific factors. In the human and mouse gut, BTNL1 expression guides specific γδ T cells, but this mechanism has not yet been studied in piglets.

Tissue samples from the jejunum of piglets aged 42 and 56 days were cryosectioned to a thickness of 5 µm and processed for indirect immunofluorescence. Primary antibodies used were MAC320 for γδ T cells, the BTNL1 receptor and CK18 for epithelial cells. Nuclei were counterstained with DAPI. Confocal laser scanning microscopy was used to analyse the samples.

The BTNL1 receptor was expressed in the gut epithelium and submucosa. γδ T cells were identified in the lamina propria and in regions of high BTNL1 expression in Peyer's patches, indicating their important role in maintaining functional immune microenvironments.

Gut epithelial expression of BTNL1 instructs the development and selection of γδ T cells in the piglet model of immunity, which shares similarities with the human system by directing specific γδ T cells to tissues. The chain specificity (Vγ7+) of γδ T-cells in the gut will be further investigated.

Poster 81:

Title:
Deciphering the HCMV gH/gL/gO–PDGFRα Interaction: A Basis for Antiviral Peptide Development

Authors:
Ruben Caspary (Erlangen, Germany)
Eileen Socher (Erlangen, Germany)
eileen.socher@fau.de

Abstract:
Human cytomegalovirus (HCMV) poses severe risks for unborn children and immunocompromised individuals due to its high prevalence in the human population. A critical step in HCMV infection is the interaction between the viral envelope trimer gH/gL/gO and the human receptor Platelet-Derived Growth Factor Receptor α (PDGFRα). This study aims to identify key molecular interactions at this interface using structural bioinformatics, with the goal of developing targeted antiviral strategies.

We analyzed two cryo-electron microscopy structures of the HCMV trimer (gH/gL/gO) in complex with PDGFRα by computational methods. The focus was on identifying interface residues crucial for viral attachment and entry. These protein–protein interaction hotspots were examined to reveal potential binding regions for antiviral peptides. Comparative analyses of two HCMV strains were performed to determine conserved versus strain-specific interactions. Additionally, we structurally evaluated a PDGFRα-derived peptide previously suggested as a viral entry inhibitor.

The comparative structural analysis revealed both shared and strain-specific interactions between gH/gL/gO and PDGFRα. Differences were observed in key contact residues, interaction energies, and contact surface areas, which may influence binding affinity.

Targeting the gH/gL/gO–PDGFRα interface represents a promising strategy to block HCMV entry and infection. Our findings highlight key interaction sites and provide a structural basis for the rational design of antiviral peptides that disrupt this critical virus–host interaction.

Poster 82:

Mastaka S, Hidetaka M., Miyoko Y., Kingo S. *Tokyo, Japan, Tochigo, Japan*

Potential for odontoblast lineage cells to attract blood vessels into the developingdental papilla

Poster 83:

Title:
Anatomy education and clinical practice: students' views

Authors:
Niki Matveeva (Skopje, North Macedonia)
Julija Zhivadinovikj Bogdanovska (Skopje, North Macedonia)
Biljana Zafirova (Skopje, North Macedonia)
Elizabeta Chadikovska (Skopje, North Macedonia)
Ace Dodevski (Skopje, North Macedonia)
Biljana Trpkovska (Skopje, North Macedonia)
Biljana Bojadzieva Stojanoska (Skopje, North Macedonia)
Anamarija Paunkoska (Skopje, North Macedonia)
niki.matveeva@medf.ukim.edu.mk

Abstract:
Anatomy education and clinical practice: students’ views

This study evaluated students’ perspectives on the relevance of anatomy education to clinical practice throughout their studies at the Faculty of Medicine in Skopje.

The study was designed as a cross-sectional, questionnaire-based investigation. It occurred at the Faculty of Medicine in Skopje at the University of “Ss Cyril and Methodius”, R. North Macedonia. The questionnaire form included demographics, five-point Likert items, and open-ended questions. Questionnaire data were collected from volunteer student participants, currently and not currently involved in anatomy education, consisting of 134 first-year and 87 fourth- and fifth-year students during March and April 2024.

Junior students agreed that their anatomy education helped them achieve some important goals for clinical practice, such as adopting and applying medical terminology and the ability to visualize a three-dimensional human body map. Senior students were less convinced that the structures and concepts acquired during anatomy education were relevant to clinical practice, helped them acquire clinical knowledge and skills effectively, and understand the principles of evidence-based medicine. Both junior students and their senior colleagues disagreed that anatomy education succeeded in developing their self-confidence, teamwork, and communication skills.

Both groups of students had similar preferences and suggestions for redesigning anatomy courses to focus on essential elements that are functionally and clinically relevant. This can be achieved through traditional and modern teaching methods, small group work, and the establishment of learning outcomes within clinically relevant contexts.

Keywords: medical education, anatomy education, clinical practice, questionnaire, survey, medical faculty

Poster 84:

Title:
Analysis of the use of the terms “Fehlbildung”, “Missbildung” and “Anomalie” in the description of disorders/differences of sex development (DSD) in German textbooks for medical students

Authors:
Vanessa Sommer (Hannover, Germany)
Christian Mühlfeld (Hannover, Germany)
Vanessa.Sommer@stud.mh-hannover.de

Abstract:
People with differences of sex development used to be called “Intersex” or “(Pseudo)hermaphrodite”. Previously, a Chicago consensus statement (CCS) proposed “disorders of sex development” (DSD) and a statement of the German Ethics Council suggested “differences of sex development” as preferred terms. The German Medical Association disapproved equalizing DSD with a disease. Common terms to describe differences are malformation (“Fehlbildung/Missbildung”), and anomaly (“Anomalie”) which can be stigmatizing and convey a negative image. The present study investigated the use of terms describing DSD in German textbooks for medical students.

A literature review of fourteen common medical textbooks was conducted to analyze the presence of a consistent definition, the use of the terminology from the CCS and the existence of a definition of DSD. Textbooks were chosen from the fields of anatomy, embryology, pediatrics and gynecology.

No consistent definition of the words “Fehlbildung”, “Missbildung”, and “Anomalie” was found among the textbooks. Some books used the terms in their texts without introducing a definition. The terminology and definition of DSD following the CCS was included in only two or three books since their introduction in 2006, respectively. Therefore, “Fehlbildung”, “Anomalie” or other potentially stigmatizing terms are still used in most German textbooks for medical students.

Despite the availability of consensus papers on the use of terms to describe DSD, potentially stigmatizing terms remain in common use in textbooks for medical students, thus delaying a change of application and prolonging or evoking unintentional discriminatory contact with people with DSD.

Poster 85:

Title:
BioMedStatX – Workflows for reliable BioMedical StatisticX

Authors:
Philipp Krumm (Aachen, Germany)
Nicole Böttcher (Aachen, Germany)
Richard Ottermanns (Aachen, Germany)
Thomas Pufe (Aachen, Germany)
Athanassios Fragoulis (Aachen, Germany)
pkrumm@ukaachen.de

Abstract:
Robust statistical analysis is a fundamental prerequisite for any publication to meaningfully contribute to scientific knowledge. However, investigations show that many researchers misinterpret p-values, apply incorrect measures of variability, and overlook critical assumptions. We introduce BioMedStatX, a user-friendly tool for reliable statistical analysis, designed to guide researchers with or without statistical training through appropriate test selection, execution, and interpretation while minimizing common pitfalls.

Methods: BioMedStatX is a Python-based desktop application featuring a graphical interface. It supports Excel and CSV import, automated assumption checks (Shapiro–Wilk for normality; Levene's test for variance homogeneity), and data transformations (log10; Box–Cox; Arcsine Square-Root). Interactive dialogs assist users in selecting variables, the groups to be tested, and customizing plot aesthetics. The analytical engine performs parametric tests (independent and paired t-tests; one-way, two-way, mixed-model, and Welch's-ANOVA) and nonparametric alternatives (Mann–Whitney U; Wilcoxon signed-rank; Kruskal–Wallis) with integrated post-hoc analyses (Tukey-HSD; Dunn; Dunnett; Dunnett-T3; Holm–Šidák). An optional outlier-detection module flags aberrant values via the Grubbs-Test and modified Z-score method. A decision-tree illustrates the analytical workflow. Comprehensive results and publication-ready plots are exported as an excel file.

Validation on datasets demonstrated accurate replication of standard analysis pipelines and compared to results from professional statistics software like JMP10, the accuracy of the results was the same to the third decimal place. Decision-trees enhanced user understanding of methodological choices made by the user and application.

BioMedStatX offers a user-friendly, reproducible platform for robust statistical analysis, empowering biomedical researchers to correctly apply statistical methods and generate high-quality, publication-ready results.

Poster 86:

Title:
Missing Balance: Gender Representation in Anatomical Atlases Used in Polish Medical Education

Authors:
ZYGMUNT DOMAGAŁA (Wroclaw, Polska)
BARTOSZ ROS (Wroclaw, Polska)
MARCEL ZUBER (Wrocław, Poland, Polska)
Michal Banasiuk (Wrocław, Poland, Polska)
Michal GAS (Wroclaw, Polska)
Oliwia Januszek (Wroclaw, Polska)
Oliwia Swigon (Wrocław, Poland, Polska)
Mateusz Mazurek (Wrocław, Poland, Polska)
Agnieszka Pinkowska (Wrocław, Poland, Polska)
Slawomir Wozniak (Wroclaw, Poland)
Katarzyna Siekaniec (Wroclaw, Poland)
zygmunt.domagala@umw.edu.pl

Abstract:
To assess gender representation in anatomical illustrations from atlases most frequently recommended by Polish medical faculties, based on a comprehensive review of anatomy syllabi. All selected atlases are widely used translations of international works.

We systematically analyzed 5 293 illustrations from three atlases (Prometheus, Netter, Sobotta) identified as most recommended across major Polish medical universities. Each image was classified according to visible gender (male, female, indeterminate), body region, pose, presence of sex-specific anatomy (reproductive organs, breasts), and estimated ethnic origin.

Male figures predominated across all atlases (13.5–25.3 %), while female depictions were significantly fewer (6.8–10.4 %). Most illustrations (64.0–79.4 %) were gender-indeterminate. Sex-specific content was predominantly female and limited to reproductive and breast anatomy. The male-to-female ratio ranged from 2.00 to 2.43. Nearly all figures were ethnically white; none depicted transgender bodies.

Our findings reveal a consistent gender imbalance and limited anatomical diversity in visual content used in Polish medical education. Female anatomy is mostly confined to reproductive contexts, potentially reinforcing stereotypes and narrowing clinical awareness. More inclusive and gender-balanced anatomical resources are needed to support equitable medical training.

Poster 87:

Title:
Advancements in Neuronal Tract Tracing in Postmortem Human Brains

Authors:
Lars Freudenmacher (Düsseldorf, Germany)
Horst-Werner Korf (Düsseldorf, Germany)
Svenja Caspers (Düsseldorf, Germany)
Lars.Freudenmacher@hhu.de

Abstract:
To enhance neuronal tract tracing in postmortem human brains, we established rotating field tracer electrophoresis (RFTE), which significantly accelerates the distribution of polar lipophilic tracers. Passive diffusion typically requires six months for 1 cm tracer diffusion in the human cortex, while RFTE achieves up to 6 cm within one week. When applied to the occipital lobe, RFTE revealed four distinct calcarine pathways, including two previously unconfirmed. The observed laminar organization of striate–extrastriate connections corresponds to known macaque data, offering a direct anatomical perspective on human brain connectivity at single-neuron resolution.

However, RFTE confines tracer transport to the xy-plane, as the electric field is defined by a two-electrode configuration and can only be relatively redirected by mechanically rotating the tissue sample. This limits the ability to guide tracers along oblique or out-of-plane fiber trajectories.

To address this, we developed pulsed field tracer electrophoresis (PFTE), enabling field control in three spatial dimensions. PFTE uses two opposing plates, each with 16 addressable electrodes. Eight electrodes are arranged radially around the tracer application site, eight parallel to the plate edges.

The electrode arrays are controlled via microcontroller and relay board, allowing programmable activation and polarity reversal. This enables generation of electric field vectors within the xy-plane and along the z-axis without moving the tissue. With inner electrodes set as anodes and outer electrodes as cathodes, the electric field drives tracer diffusion outward from the application site. Active cooling maintains temperature during prolonged current application.

Thus, PFTE offers a technical framework for future tract-tracing approaches.

Poster 88:

Title:
A novel interactive virtual reality (VR) application for teaching muscle anatomy to first-year medical students

Authors:
Andreas Hainfellner (Vienna, Austria)
Karoline Schwendt (Vienna, Austria)
Ulrich Renner (Vienna, Austria)
Cyrill Slezak (Orem, UT / Vienna, USA / Austria)
Paul Slezak (Vienna, Austria)
Wolfgang J. Weninger (Vienna, Austria)
andreas.hainfellner@meduniwien.ac.at

Abstract:
We aim at presenting a prototype of an innovative virtual reality (VR) anatomy learning environment, which supports teaching muscle anatomy to first year medical students and students of paramedical subjects.

A customized software tool was developed using the virtual environment of the ESWT learning platform software package (WaveVision). Digital video sequences were integrated, which were produced by using the infrastructure and body donors enrolled to the body donor program of the Division of Anatomy of the Medical University of Vienna. The software operated on 85 headsets.

In the VR environment students watch videos, in which important muscles of the human body are demonstrated on dissected body donor material. In parallel, they identify three-dimensional (3D) muscle models placed in the virtual environment by their morphology and try to virtually orient them along a 3D skeleton model. Muscles, correctly arranged in orientation, origin and insertion virtually slip in position. The application was tested by all 760 first year students in mandatory practical VR courses of the Medical Curriculum of the Medical University of Vienna. Approximately two thirds of the students agree that the tool aids them in preparation for their exams.

We introduce a novel tool, which integrates video demonstration of dissected body donor material and interactive VR learning. Evaluations demonstrate that medical students at the beginning of their anatomy education and prior to dissection classes substantially profit from it.

Poster 89:

Title:
Effect of high-affinity activators on the modified Two-Pore Domain Potassium (K2P) Channel TREK-1

Authors:
Lukas Weissbach (Kiel, Germany)
Peter Lindemann (Berlin, Germany)
Marc Nazaré (Berlin, Germany)
Thomas Baukrowitz (Kiel, Germany)
Marcus Schewe (Kiel, Germany)
lukasweissbach@posteo.de

Abstract:
TWIK-related potassium channel-1 (TREK-1), a member of the two-pore domain potassium (K2P) channel family, generates an outwardly rectifying potassium current that stabilizes the resting membrane potential in excitable cells. Thus, it plays a key role in (patho)physiological processes such as neuronal and cardiac excitability and certain pain conditions highlighting it as pharmacological target. This study aimed to identify high-affinity activators by targeting the ML335 binding site in TREK-1.

ML335 (a non-covalent TREK activator) was modified to carry electrophilic “warheads“, enabling the formation of covalent bonds with nucleophilic cysteine residues. Site-directed mutagenesis was employed to introduce strategically positioned cysteine residues in the ML335 binding site, facing the warheads. 17 ML335 derivatives were tested using electrophysiological assays on wild type (WT) and mutant TREK-1 channels, heterologously expressed in Xenopus laevis oocytes.

Derivative SCM-07-181 (ML335 core structure + acrylamide) showed higher efficacy than ML335 (20 µM: 19- vs. 4-fold on WT; 33- vs. 6-fold on TREK-1 H141C). SCM-07-181 also showed slightly higher affinity for H141C compared to ML335 (EC50: ML335 ~27 µM; SCM-07-181 ~6 µM). However, all derivatives demonstrated washout behaviour, suggesting that covalent binding did not occur. Interestingly, 14 derivatives with warhead substitution at the lower ring of the ML335 core structure failed to activate TREK-1 WT or mutant channels.

Although covalent binding was likely not achieved, SCM-07-181 remains a promising ML335 derivative. Overall, upper ring modification appears most promising for developing high-affinity TREK-1 activators targeting the ML335 binding site.

Poster 90:

Title:
Effects of SiMa neuroblastoma cells on gap junction coupling and neurotransmitter sensitivity in LN405 astrocytoma cells

Authors:
Reuss Bernhard (Göttingen, Federal Republic of Germany)
breuss@gwdg.de

Abstract:
Neuron-glial interactions are important to regulate astroglial functions according to the metabolic demands of neuronal cells.

Here, SiMa human neuroblastoma cells (SiMa cells), and the human astrocytoma cell line LN405 (LN405 cells) were used to investigate whether neuronal cells can modulate astroglial gap junction coupling.

As revealed by immunofluorescent detection of the astroglial gap junction protein Connexin43 (Cx43) in co cultures of SiMa cells and LN405 cells, neuroblastoma cells are indeed able to induce expression of Cx43 in LN405 cells. To clarify whether this was a direct contact effect, or was mediated by soluble factors, LN405 cells were incubated with SiMa cells conditioned medium (SCM). As revealed by immunofluorescent detection, SCM was indeed able to induce a significant increase in size and numbers of Cx43 positive gap junction plaques in LN405 cells. However, in contrast to what one would have expected, scrape loading of LN405 cells with the gap junction permeant dye Lucifer yellow, revealed a significant reduction in dye spreading between LN405 cells incubated with SCM. This was further corroborated by experiments with the fluorescent calcium indicator Fluo3 revealing a significant reduction in glutamate elicited calcium signals in LN405 cells preincubated with SCM as compared to non-treated controls.

In conclusion these experiments demonstrate that diffusible factors released by neuroblastoma cells can modulate astroglial gap junction coupling and neurotransmitter sensitivity, however in a more complex manner than it was initially expected. Future experiments will have to clarify the molecular nature of the factors mediating these effects.

Poster 91:

Title:
Preventive effect of probiotics on periodontal ligament in a rat model of anorexia nervosa

Authors:
Stefanie Trinh (Aachen, Germany)
Larissa Blische (Aachen, Germany)
Clara Voelz (Hannover, Germany)
Marta Rizk (Aachen, Germany)
Fabian Kiessling (Aachen, Germany)
Thomas Pufe (Aachen, Germany)
Michael Wolf (Aachen, Germany)
Rogerio Craveiro (Aachen, Germany)
Jochen Seitz (Essen, Germany)
Cordian Beyer (Aachen, Germany)
ntrinh@ukaachen.de

Abstract:
The rising prevalence of anorexia nervosa (AN) and the limited understanding of its effects on the periodontium often hinder decision making in dentistry and periodontology. As an adjunct to periodontal therapy, probiotics have promising effects on oral health by decreasing pathogens and altering local inflammation. This study investigates changes in morphology and remodelling of alveolar bone and periodontal ligament (PDL) in an AN-like rat model and potential protective effects of probiotics.

Three-week-old female Wistar rats were divided into three groups: a control group with ad libitum food and two groups undergoing an activity-based anorexia (ABA) model. One ABA group received oral multi-strain probiotics during starvation. After five weeks, all rats were sacrificed for ex-vivo micro-CT scans of the maxilla and mandible to assess alveolar bone and PDL morphology, as well as histological evaluations for PDL fibre vitality and structural organization.

Our data show no structural changes in alveolar bone caused by either ABA or probiotic treatment; however, the ABA group exhibited a significant reduction in PDL thickness, which could not be reversed by probiotic treatment. Despite this, histological analysis indicated improved connectivity and density of PDL fibres in the probiotic group compared with the ABA-only group. No differences were found between the mandible and maxilla.

While probiotics did not prevent PDL thinning, they enhanced its composition/vitality compared to the ABA condition alone. Further research is required to elucidate the process of PDL remodelling under starvation and in relation to mechanical stimuli in the periodontium.

Poster 92:

Title:
The influence of the gut microbiome on the hypothalamic appetite regulation in an activity-based anorexia rat model

Authors:
Luisa Bosio (Aachen, Germany)
Stefanie Trinh-Müggenburg (Aachen, Germany)
Jochen Seitz (Essen, Germany)
Cordian Beyer (Aachen, Germany)
Greta Wittemeier (Aachen, Germany)
Larissa Blischke (Aachen, Germany)
luisa.bosio@hotmail.de

Abstract:
Anorexia nervosa (AN) is a severe psychiatric disorder with high mortality rate. Patients suffer from restrictive eating and hyperactivity. Recent research highlights the gut microbiome`s impact on neuropsychiatric diseases. The hypothalamic nucleus arcuatus (ARC) plays a central role in appetite and energy regulation. Thus, we aim to investigate the influence of the gut microbiome on the ARC.

We used the activity-based anorexia (ABA) rodent model, which mimics the core symptoms of AN (weight loss, hyperactivity). ABA combines limited food with permanent running wheel access. Through antibiotic treatment the rats' endogenous gut microbiome was depleted, followed by fecal microbiota transplantations (FMT). The AN group received FMT from patients with AN, while the healthy control (HC) group received FMT from healthy donors. Immunohistochemistry quantified microglia and astrocytes as well as the expression of ARC expressed peptides. Gene expression was assessed by qPCR. Serum leptin levels were measured using ELISA.

ABA induced a downregulation of anorexigenic genes (e.g. POMC), and an upregulation of orexigenic genes (NPY and AgRP). A reduction of GFAP expression was found across all ABA groups, but no significant changes in Iba1 expression. Circulating leptin was significantly decreased in all ABA animals. The gut microbiome only significantly affected the expression of AgRP, which was upregulated in the HC compared to the AN group.

ABA significantly alters hypothalamic appetite regulation toward an orexigenic state. Further ongoing histological analyses and microbiota sequencing data may clarify the gut microbiome's relevance in appetite and energy regulation.

Poster 93:

Title:
Molecular and cellular hypothalamic signatures in an activity-based anorexia rat model

Authors:
Larissa Blischke (Aachen, Deutschland)
Greta Wittemeier (Aachen, Deutschland)
Luisa Bosio (Aachen, Deutschland)
Nele Korten (Aachen, Deutschland)
Clara Voelz (Hannover, Deutschland)
Jochen Seitz (Essen, Deutschland)
Stefanie Trinh (Aachen, Deutschland)
Cordian Beyer (Aachen, Deutschland)
lblischke@ukaachen.de

Abstract:
Anorexia nervosa (AN) is a severe psychiatric disorder with restrictive eating, neural dysfunction and systemic inflammation. The hypothalamic arcuate nucleus (ARC) plays a key role in controlling appetite, metabolic signals, and neuroendocrine functions. This study aimed to characterize gene expression across the hypothalamus and circulating leptin levels under chronic ABA and explore potential modulatory effects of omega-3 fatty acids and probiotics.

The activity-based anorexia (ABA) rodent model combines free access to a running wheel with food restriction. After the rats lost 25% of their initial body weight (acute ABA), the chronic ABA phase was established. Gene expression of ARC-related genes such as NPY, AgRP, POMC, 5-HT2C and glial cell markers (GFAP, Iba1) were studied. Using immunohistochemistry, positive cells of these markers within the hypothalamus were quantified. Serum leptin levels were measured using ELISA.

ABA consistently upregulated the orexigenic markers NPY and AgRP, while expression of the anorexigenic peptide POMC was downregulated. GFAP levels were reduced across all ABA groups, whereas Iba1 expression showed a selective decrease in the probiotic-treated group. In addition, histological alterations at the cellular level within the hypothalamus were observed during ABA. In line with a negative energy balance, leptin concentrations were significantly reduced in all ABA animals, indicating a shift toward enhanced orexigenic signaling.

ABA induces pronounced hypothalamic shifts toward orexigenic signaling, accompanied by alterations in glial marker expression. Nutritional interventions showed only limited gene-specific modulatory effects.

Poster 94:

Title:
Cytoprotection by a HspB5/alphaB-crystallin peptide fragment

Authors:
Britta Bartelt-Kirbach (Ulm, Germany)
Nikola Golenhofen (Ulm, Germany)
britta.bartelt@uni-ulm.de

Abstract:
Development of peptide therapeutics is rapidly increasing with more and more peptide candidates in preclinical and clinical studies. The small heat shock protein HspB5/alphaB-crystallin is a chaperone with known cyto- and also neuroprotective function. To identify a possible neuroprotective HspB5 peptide we evaluated the effect of a known cardioprotective HspB5 peptide comprising amino acids 45-54, termed Cryab1, on cultured rat hippocampal cells.

Primary cultured rat hippocampal cells (neurons and astrocytes, DIV14) were first treated with various concentrations of FITC-labeled Cryab1 peptide solution for 2-24 hours to evaluate cellular uptake. Subsequently, cells were subjected to oxidative stress 2-6 hours after unlabeled Cryab1 application and cell survival was evaluated 48 hours after stress by live-cell staining and assessment of nuclear morphology.

FITC-labeled Cryab1 was taken up by hippocampal cells within 2 hours in a concentration-dependent manner without the need of a transfection reagent or additional cell-penetrating peptide sequences. The addition of unlabeled Cryab1 was found to ameliorate overall and neuronal cell death 48 hours after oxidative stress.

Since endogenous HspB5 upregulation is known to be beneficial in neurodegenerative diseases and our data show a neuroprotective effect of the HspB5 peptide Cryab1 in cultured hippocampal cells this peptide might be an interesting candidate for new therapeutic strategies and should be further investigated in detail.

Poster 95:

Title:
Postnatal Development of the Spine Apparatus in the Murine Hippocampus: A Multimodal Imaging Study

Authors:
Johanna Hallenberger (Düsseldorf, Germany)
Elena Niggetiedt (Düsseldorf, Germany)
Christine Martin (Düsseldorf, Germany)
Max Anstötz (Düsseldorf, Germany)
Johanna.Hallenberger@med.uni-duesseldorf.de

Abstract:
During postnatal brain development, excitatory synapses undergo extensive structural remodelling to support emerging connectivity and synaptic function. Often, dendritic spines on neuronal dendrites serve as primary sites for excitatory input. A key substructure in many spines is the so-called spine apparatus (SA) — a smooth endoplasmic reticulum-derived organelle involved in calcium regulation and synaptic plasticity. While its function is increasingly understood, the developmental emergence and maturation of the SA, particularly in the hippocampus, remain unclear. This study investigates the postnatal development, morphology, and localization of the SA in dentate gyrus granule cells of the murine hippocampus.

A multimodal imaging approach was used to assess dendritic spines and the SA across three developmental stages (juvenile, adolescent, mature). Golgi-Cox staining quantified changes in spine density and morphology. Immunohistochemistry combined with bright-field and confocal microscopy enabled localization of SA markers. In addition, 3D Focused Ion Beam-Scanning Electron Microscopy (FIB-SEM) provided ultrastructural insights into intracellular membrane organization.

Golgi-Cox staining revealed a progressive increase in spine density and morphological complexity, followed by a decline at later stages, suggesting dynamic synaptic maturation and pruning. FIB-SEM analysis indicated corresponding changes in subcellular membrane structures consistent with SA emergence and synapse maturation.

This study integrates complementary imaging techniques to track the developmental trajectory of the spine apparatus. Our findings contribute to a better understanding of synaptic refinement in the hippocampus and may inform research into neurodevelopmental disorders involving synaptic dysfunction.

Poster 96:

Title:
Impact of Fecal Microbiota from Patients with Anorexia Nervosa on Brain Glial Cells in Rats

Authors:
Nele Maria Korten (Aachen, Germany)
Larissa Blischke (Aachen, Germany)
Mai Tran (Aachen, Germany)
Anna Schulze-Eckel (Aachen, Germany)
Anna Carola Thelen (Aachen, Germany)
Cordian Beyer (Aachen, Germany)
Jochen Seitz (Essen, Germany)
Stefanie Trinh (Aachen, Germany)
Clara Voelz (Hannover, Germany)
voelz.clara@mh-hannover.de

Abstract:
Anorexia nervosa (AN) is a complex eating disorder characterized by extreme underweight, fear of weight gain, and body image distortion. Due to the associated malnutrition, the gut-brain axis may play a key role in its pathology. Prior studies have linked the gut microbiome to structural brain changes in AN. This study aims to explore its impact using a humanized rat model by transplanting stool samples from patients with AN.

Female Wistar rats were pretreated with broad-spectrum antibiotics to deplete their native microbiome, followed by four weeks of fecal microbiota transplants from patients with AN or age- matched healthy controls. Microbiome transfer efficiency was assessed via 16S rRNA sequencing. In the hippocampus, glial markers (Gfap, Aif1, Olig2) and genes related to inflammation and plasticity (Bdnf, Mki67, Rbfox3, Dcx, Map2, CD11b, IL6, TNFa) were analyzed.

Antibiotic treatment disrupted microbial diversity, which was partially restored by microbiota transplantation. No changes in eating or activity behavior were observed. While cell counts remained unaffected, hippocampal neuroplasticity was altered: Bdnf expression decreased after antibiotics and increased -along with inflammatory markers- following transplantation with the microbiome samples of the healthy donors.

The treatment protocol itself did not lead to an AN-like phenotype in the rats. However, an impact of the microbiome depletion and transplantation was observed regarding the plasticity in the hippocampus. It would be interesting to characterize the functional impact and to translate alterations to the cognitive impact on patients with AN.

Poster 97:

Title:
Stimulation of Cajal-Retzius Cells Modulates Neuronal Circuitry in the Dentate Gyrus

Authors:
Kelvin Maximilian Tofan (Düsseldorf, DE)
Max Anstötz (Düsseldorf, DE)
kelvin.tofan@hhu.de

Abstract:
Cajal-Retzius (CR) cells are essential for prenatal brain development and persist postnatally only along the hippocampal fissure. However, their functional role in the postnatal hippocampal network remains unclear. Here, we investigate how CR cell activity influences hippocampal circuitry.

To this end, we generated a transgenic mouse line allowing conditional expression of the excitatory DREADD (Designer Receptor Exclusively Activated by Designer Drugs) hM3Dq in CR cells. Activation of hM3Dq by Clozapine-N-Oxide (CNO) enhances CR cell excitability via a G-protein–coupled receptor mechanism. We performed patch-clamp electrophysiology in acute hippocampal slices, assessing action potential frequency and amplitude, as well as excitatory and inhibitory postsynaptic currents (EPSCs and IPSCs) in multiple neuron types before and after CNO application. CR cells were identified by mCitrine fluorescence; GABAergic interneurons and granule cells were confirmed via confocal imaging.

Stimulation of CR cells led to distinct changes in dentate gyrus circuit activity. We observed increased excitatory input to specific GABAergic interneuron subtypes and enhanced disynaptic inhibition of granule cells. Peak amplitude analysis revealed significant modulation of IPSCs in granule cells. Notably, CR cells themselves exhibited increased IPSC frequency upon CNO application, suggesting a positive feedback loop mediated by GABAergic interneurons.

These findings establish CR cells as active modulators within the postnatal hippocampal network. Future studies will examine the long-term effects of CR cell activation on hippocampal function and behavior through DREADD-based approaches.

Poster 98:

Title:
Relationship between Hypothalamic Regulation and Keel Bone Pathology: a Comparative Neuroanatomical Study in Commercial and Wild-Type Hens

Authors:
Ilias-Dimitrios Karlaftis (Düsseldorf, Germany)
Stefanie Petow (Munich, Germany)
Lisa Hildebrand (Celle, Germany)
Saskia Neukirchen (Bonn, Germany)
Mareike Fellmin (Rommerskirchen, Germany)
Julia Mehlhorn (Düsseldorf, Germany)
Ilias-Dimitrios.Karlaftis@hhu.de

Abstract:
Keel bone damage is a prevalent welfare concern in commercial laying hens, linked to high egg production and hormonally driven calcium mobilization from the medullary bone. This process is regulated by diencephalic brain structures, yet the underlying neuronal mechanisms have been poorly understood.

To explore whether selective breeding for high laying performance has altered neuroendocrine systems we conducted an immunohistochemically analysis of Gonadotropin Releasing Hormone (GnRH) positive neurons in the diencephalon of a high performing layer line (Lohmann Selected Leghorn (LSL) and its wild ancestor the Red Junglefowl (RF). We quantified GnRH neuron numbers and fiber densities and performed allometric comparisons of brain structure volumes across seven other chicken breeds.

Our analysis revealed intraregional variation in hypothalamic GnRH expression. In LSL hens, the nucleus of the commissura pallii (nCPa) and eminentia mediana (ME) exhibited elevated GnRH densities, whereas in RF hens, high densities were observed in the nCPa and the nucleus lateralis anterior thalami (LA). The septal region showed no significant variation within LSL hens but exhibited lower GnRH-positive cell counts compared to RF hens. Allometric analysis demonstrated that LSL hens have relatively smaller volumes in several brain regions, including the cerebellum and tegmentum.

These findings suggest that both artificial selection and natural variation influence brain structure and neuroendocrine function in chicken. Our results highlight the potential impact of breeding for productivity on neural architecture, offering important implications for animal welfare and avian neurobiology.

Poster 99:

Title:
Characterization of MrgD Receptor Expression in the Mouse Brain: Insights into its Potential Role in CNS Function

Authors:
Javad Hami (Potsdam, Germany)
Viola von Bohlen Und Halbach (Greifswald, Germany)
Anja Tetzner (Cork, Ireland)
Thomas Walther (Cork, Ireland)
Oliver von Bohlen und Halbach (Greifswald, Germany)
javad.hami@hmu-potsdam.de

Abstract:
Recent findings have revealed a functional renin-angiotensin system (RAS) within the central nervous system (CNS), suggesting its involvement in various brain functions beyond cardiovascular regulation. One novel component of this system is the Mas-related G-protein-coupled receptor D (MrgD), which is activated by Angiotensin-(1–7) and its derivative Ala^{1}-Ang-(1–7). While MrgD has been identified in the brain, its functional role remains largely unclear.

To investigate the expression of MrgD in the brain, we utilized a MrgD IRES-EGFPf transgenic mouse model. Brain sections were immunolabeled using GFP and Cy3 antibodies and analyzed by fluorescence microscopy. Signal intensity was quantified using the corrected total cell fluorescence (CTCF) method, and data were expressed as the mean number of MrgD-positive cells per brain region (±SEM).

Our results demonstrated the presence of MrgD-expressing cells in several forebrain regions, including the cortex, hippocampus, amygdala, hypothalamus, habenular nuclei, striatum, and pallidum. Additionally, MrgD expression was observed in specific midbrain nuclei.

The widespread localization of MrgD in areas associated with emotional regulation, reward processing, and sensory integration points to its potential involvement in modulating pain, synaptic plasticity, memory, and cognition. These findings provide a foundation for future studies on MrgD's role in CNS physiology and its possible relevance in neuropsychiatric or neurodegenerative conditions.

Poster 100:

Title:
Environmental and Behavioral Modulation of Adult Neurogenesis in the Avian Pallium

Authors:
Julia Mehlhorn (Düsseldorf, Germany)
Ilias-Dimitrios Karlaftis (Düsseldorf, Germany)
Serap Kurutas (Düsseldorf, Germany)
Katrin Amunts (Düsseldorf AND Jülich, Germany)
Svenja Caspers (Düsseldorf AND Jülich, Germany)
Christina Herold (Düsseldorf, Germany)
julia.mehlhorn@hhu.de

Abstract:
Adult neurogenesis (AN) has been widely studied, but its functional role still raises a number of questions. In birds, AN is more widespread compared to mammals, enabling comparative studies under different experimental conditions.

To study the influence of different housing and experimental conditions, five groups of homing pigeons (Columba livia f.d.) were initially treated with 5-bromo-deoxyuridine (BrdU) to label newborn cells. Free-Flyers received daily free flight for two hours and were trained in a learning task in a standard operant chamber. Box-Training pigeons were housed permanently in the loft, but trained the same task as Free-Flyers. Non-Flyers were housed permanently in the loft. Flight-Training pigeons were trained with repeated releases from unknown places and the controls received only daily free flight. After that all brains were immunohistochemically processed using specific markers (NeuN, S100b, DCX) to examine newly generated or proliferating cells in the hyper- and mesopallium.

In both areas, adult neuro- and gliogenesis were evident with heterogeneous distributions. The highest counts of newborn and proliferating neurons were detected in Free- and Non-Flyers in the hyperpallium densocellulare and mesopallium. Control pigeons showed high levels of newborn glial cells in the hyperpallium apicale. Box- and flight-trained pigeons showed moderate neurogenic activity.

The results show functionally related and region-specific plasticity. This indicates that individual life history is relevant for AN, while at the same time diverse cognitive tasks as well as housing conditions modulate AN differently.

Poster 101:

Title:
Modeling Reelin functions in human cerebral organoids

Authors:
Linus Kordt (Bochum, Germany)
Marie-Luise Kümmel (Bochum, Germany)
Boris Burr (Bochum, Germany)
Princy Kakani (Bochum, Germany)
Holm Zaehres (Bochum, Germany)
Eckart Förster (Bochum, Germany)
linus.kordt@ruhr-uni-bochum.de

Abstract:
Reelin, a large extracellular glycoprotein encoded by the RELN gene, is essential for cortical development and implicated in various neurodevelopmental and neurodegenerative disorders, including Alzheimer's disease, schizophrenia, autism, and epilepsy. With this study, we aim to model reelin function in early human cerebral development using cerebral organoids derived from wild-type and genetically modified human induced pluripotent stem cells (iPSCs).

Using CRISPR/Cas9, we generated a RELN knockout iPSC line and are attempting to confirm the introduction of the gain-of-function RELN-COLBOS mutation associated with resilience to Alzheimer’s disease in two different iPSC lines. Several guide RNAs targeting six exons of RELN were cloned, and a homology-directed repair template was designed. Post-transfection, puromycin selection enabled isolation of individual clones. Genotyping was performed via PCR and Sanger sequencing. Cerebral organoids were generated as per Lancaster et al. (2013) from wild-type and knockout iPSC lines for immunohistochemical evaluation.

We confirmed a frameshift mutation near Reelin’s central receptor-binding region in a knockout clone. Organoids developed from both wild-type and knockout lines showed Cajal-Retzius cells (Reelin and p73-positive) in the numerous neural rosettes, yet no reelin expression in the knockout, confirming successful gene disruption.

These organoid-based models allow the study of reelin’s role in early human cortical development, its neuroprotective functions, and its role in regeneration. Ongoing and future work includes comparative histological/immunohistochemical analyses of cortical architecture, investigation of reelin-mediated signaling pathways, and transcriptomic and proteomic comparisons. Stress conditions such as elevated temperatures and oxygen/glucose deprivation will be applied to assess reelin’s functions in neuroprotection and regeneration.

Poster 102:

Title:
Mitochondrial dysfunction impacts choroid plexus barrier function

Authors:
Katherine Dodel (Mannheim, Germany)
Lisa Wolters (Mannheim, Germany)
Silke Vorwald (Mannheim, Germany)
E. Ronald Curticean (Heidelberg, Germany)
Irene Wacker (Heidelberg, Germany)
Rasmus Schröder (Heidelberg, Germany)
Victor Costina (Mannheim, Germany)
Christian Schultz (Mannheim, Germany)
Christian Schwerk (Mannheim, Germany)
James Stewart (Newcastle upon Tyne, United Kingdom)
Nina Bonekamp (Mannheim, Germany)
nina.bonekamp@medma.uni-heidelberg.de

Abstract:
Pathogenic variants in mitochondrial genes cause a wide spectrum of mitochondrial diseases such as Leigh Syndrome or MELAS. Using a mouse model of mitochondrial tRNA disease (m.5019A>G), we observed a severe mitochondrial dysfunction in the choroid plexus (ChP), localized in the ventricles of the brain. The major function of the ChP is the energy-consuming production and secretion of the cerebrospinal fluid (CSF). Furthermore, the choroid plexus epithelial cells (CPECs) form the blood-cerebrospinal fluid barrier (BCSFB) that is crucial for maintaining the brain microenvironment, serving as an entry point between the periphery and the central nervous system. Of note, morphological alterations of CPECs were observed in Leigh syndrome and MELAS patients in the late 1980s, but potential consequences for disease etiology remain elusive. Here, we examined the link between mitochondrial dysfunction and ChP homeostasis.

We use m.5019A>G mice and an in vitro model of the human BCSFB as model systems. We combine confocal and electron microscopy and proteomics measurements with measurements of transepithelial transport.

Electron microscopy reveals morphological alterations of mitochondrial cristae and cellular contacts. Upon induction of mitochondrial dysfunction in vitro by application of an inhibitor of mitochondrial transcription (IMT), we observe disrupted cell-cell contacts as evidenced by confocal microscopy and proteomics. This results in increased transepithelial transport through the CPEC barrier.

Our data indicates that mitochondrial dysfunction compromises ChP barrier function, leading to increased leakiness through the BCSFB. We hypothesize that this may lead to higher susceptibility for neuroinflammation and may exacerbate neurological symptoms in primary mitochondrial disease.

Poster 103:

Title:
Inhibitory control of the interstitial nucleus of Cajal in the mesencephalon in primates: role in the generation of vertical eye movements.

Authors:
Anja Horn-Bochtler (Munich, Germany)
Ümit Mayadali (Munich, Germany)
Pia Anthuber (Munich, Germany)
Sunil Yeruva (Munich, Germany)
Aasef Shaikh (Cleveland, USA)
Anja.Bochtler@med.uni-muenchen.de

Abstract:
Saccadic eye movements are generated by the interaction of inhibitory omnipause neurons (OPN) and excitatory and inhibitory premotor burst neurons. Burst neurons for horizontal and vertical gaze lie anatomically separate in the brainstem. The well-studied premotor circuit for horizontal saccades includes the reciprocal connection of inhibitory burst neurons of both sides, whose malfunction may cause horizontal eye oscillations. A similar circuit was proposed for vertical saccadic inhibitory burst neurons located in the mesencephalic interstitial nucleus of Cajal (INC).

To test this assumption in primates we investigated the inhibitory transmitter inputs to premotor burst neurons in INC and correlated the findings with tract-tracing studies in rhesus monkeys.

Premotor burst neurons in the monkey INC are ensheathed by perineuronal nets, which helped to identify the homologous neurons in humans. Similarly large populations of premotor GABAergic – presumed IBNs - and non-GABAergic neurons – presumed EBNS – exist, which are contacted by numerous GABAergic terminals. Only a subset of excitatory premotor neurons are contacted by glycinergic terminals most likely arising from OPNs. GABAergic and glycinergic inputs to INC in human resembled the findings in monkey.

The presence of premotor GABAergic neurons associated with GABAergic terminals and strong projection to contralateral INC suggest a similar reciprocal projection between IBNs of both sides for vertical saccades as seen for horizontal saccades. Unlike for horizontal saccades, no evidence was found for an OPN input to vertical saccadic IBNs in INC, which rather targeted a small population of excitatory neurons, whose function has to be clarified.

Poster 104:

Title:
IL-11 might regulate vGAT and vGlut-1 protein levels in hippocampal neurons

Authors:
Ines Erdmann-Wolff (Magdeburg, Germany)
Nele Hahn (Magdeburg, Germany)
Juliane Lokau (Hannover, Germany)
Christoph Garbers (Hannover, Germany)
Anne Albrecht (Madgeburg, Germany)
ines.erdmann-wolff@med.ovgu.de

Abstract:
Interleukin (IL-)11 is a proinflammatory cytokine of the IL-6 family that is involved in autoimmune disease, fibrosis and cancer. A recent study demonstrates a potential role of IL-11 within the brain, with elevated IL-11- or IL-11Raplha2-receptor levels in the hypothalamus being associated with depressive behavior in mice. Here, we investigated the impact of IL-11 on neuronal plasticity in organotypic slice cultures of mouse hippocampus (OHSCs).

OHSCs were stimulated with recombinant IL-11 peptide. Proteins relevant for synaptic plasticity were investigated using immunofluorescence staining, western blot and qPCR analysis.

IL-11 stimulation activated the JAK/STAT pathway in OHSCs. A screen for regulatory expression of synaptic proteins per western blot identified a downregulation of vGAT (vesicular GABA transporter) after 24h of IL-11 stimulation. Immune fluorescent stainings revealed a layer specific regulation of vGAT in OHSCs, with an upregulation in the pyramidal layer of the CA3 region. In addition, the vesicular glutamate transporter 1 vGlut-1 was downregulated in this layer. mRNA levels were not altered for vGAT or vGlut-1 at 24h of IL-11 treatment.

Our findings suggest that IL-11 regulates plasticity-relevant targets, including vGAT and vGlut-1. The opposing regulation of these transporters in the CA3 pyramidal layer may shift the excitation/inhibition balance, which warrants further investigation. Unraveling the neuronal and glial impact of proinflammatory IL-11 may support the development of new therapeutic strategies for neuropsychiatric disorders associated with (micro-)inflammation such as neurodegeneration during aging and/or depression.

Poster 105:

Title:
Quantitative Description of Perineuronal Nets in Focal Cortical Dysplasia Type IIb

Authors:
Sophia Auer (Erlangen, Germany)
Lucas Hoffmann (Erlangen, Germany)
Ingmar Blümcke (Erlangen, Germany)
Martin Schicht (Erlangen, Germany)
Friedrich Paulsen (Erlangen, Germany)
sophia.auer@fau.de

Abstract:
Focal cortical dysplasia type IIb (FCDIIb) is a frequent cause of pharmacoresistant epilepsy in children and is histopathologically characterized by cortical dyslamination, dysmorphic neurons, and balloon cells. Although somatic mutations in the mTOR signaling pathway are implicated in a subset of FCDIIb cases, the contribution of extracellular matrix (ECM) alterations to its pathophysiology remains poorly understood. Perineuronal nets (PNNs) are specialized ECM structures that primarily enwrap parvalbumin-expressing (PV+), GABAergic inhibitory interneurons and are essential for maintaining synaptic stability and regulating cortical excitability. While PNN alterations have been reported in various forms of epilepsy, their presence and distribution in FCDIIb has not been systematically investigated.

Human brain tissue samples from FCDIIb patients and non-epileptic controls were examined using immunohistochemistry and immunofluorescence with specific markers for PNNs, GABAergic interneurons, PV+ interneurons, and balloon cells. PNN density and cellular association were quantified in lesional, perilesional, and subcortical white matter regions.

We observed a region-specific increase in PNN density within FCDIIb lesions compared to perilesional cortex and control tissues. Furthermore, an age-dependent increase in PNN density was detected across both cohorts, with overall higher levels in FCDIIb tissue.

These findings demonstrate abnormal PNN accumulation in FCDIIb lesions, pointing to ECM remodeling as potential contributor to cortical hyperexcitability seen in epilepsy. These results underscore the importance of the ECM in epilepsy and suggest PNNs as modulators of epileptogenicity in FCDIIb.

Poster 106:

Title:
Secreted Protein Acidic and Rich in Cysteine (SPARC) Localisation and Expression in Glaucomatous Retina

Authors:
Anna Niebauer (Regensburg, Germany)
Rudolf Fuchshofer (Regensburg, Germany)
anna.niebauer@vkl.uni-regensburg.de

Abstract:
Primary open-angle glaucoma is a chronic progressive neuropathy. The primary risk factor is intraocular pressure (IOP) that results in loss of optic nerve axons. SPARC is a matricellular protein regulating the extracellular matrix. SPARC overexpressions have been associated with elevated IOPs, while its knockout has been linked to reduced IOPs, suggesting a potential involvement in the development of glaucomatous changes. SPARC is expressed in the retina, so we aimed to localize it and analyze how elevated IOP affects SPARC in a mouse model of glaucoma on retinal ganglion cells (RGCs).

Retinae from 12-week-old βB1-CTGF1-CD1 mice and wildtype (WT) littermates were analyzed. Immunohistochemical staining on sagittal sections and retinal flatmounts was used to localize SPARC. Double-immunostaining with SPARC and an RGC marker allowed for specific investigation of SPARC expression in RGCs. SPARC expression levels were assessed via rt-qPCR and Western Blot. Bioinformatic analysis of single-cell RNA sequencing (scRNA-seq) data was used to validate expression patterns.

SPARC was localized in the RGC layer, as well as in inner and outer nuclear layers, confirming previous scRNA-seq data showing a consistent expression pattern in the retina. Fewer SPARC-positive RGCs were observed in glaucomatous retinae, despite increased SPARC expression within individual RGCs. Total retinal SPARC expression was significantly reduced, as confirmed by rt-qPCR and Western Blot.

Our data demonstrate that SPARC expression is significantly altered in the glaucomatous retina compared to WT retina. These findings imply the potential involvement of SPARC in the survival of RGCs and the pathogenesis of glaucoma.

Poster 107:

Title:
Role of EGFL7 in glioma-neuron interactions

Authors:
Julia Göhler (Dresden, Germany)
Philipp Abe (Dresden, Germany)
Kathrin Barth (Dresden, Germany)
Mirko HH Schmidt (Dresden, Germany)
julia.goehler2@tu-dresden.de

Abstract:
Glioblastoma are fatal brain tumors whose progression is highly driven by their microenvironment and neuronal activity. Especially synaptic interactions between neurons and cancer cells gained more importance in the recent years. This tumor microenvironment is significantly influenced by EGFL7 (Epidermal growth factor-like domain 7), that plays a versatile role in the regulation of multiple mechanisms. It is a angiogenic protein secreted by endothelial cells of blood vessels and present in the extracellular matrix. Additionally it is expressed in neurons of the grey matter. EGFL7 is not only a actor in angiogenesis but also showed its role in neurogenesis. Therefore the influence of EGFL7 on the connection between neurons and tumor cells, that are higly influenced by the tumor microenvironment, as well as on the synaptic plasticity will be investigated.

In vitro and in vivo models have been instrumentalized to analyze the neuron-to-tumor synapses of different gliomablastoma models, their dependence on EGFL7 and how it might influence synaptic plasticity.

Analysis from gliobalstoma tissue showed a reduction of glutamatergic synaptic markers in EGFL7 overexpressing tumors. The amount of neuron-to-tumor synapses was discoverd to additionally reduce in the presence of EGFL7 in vitro, indicating a potential antagonistic effect of EGFL7 on those synapses.

Understanding the regulation of neuron-glioma synaptic interactions through EGFL7 offers novel insights into the physiology of glioblastomas. Targeting EGFL7 or its downstream pathways may disrupt the supportive tumor microenvironment and improve outcomes for patients and therefore can open new treatment possiblities and understanding of those severe forms of cancer.

Poster 108:

Title:
Nociception in the Dish: Investigating Neuropathic Pain Mechanisms Using a Human Neuro-Mesodermal Assembloid Model

Authors:
Ann-Sophie Schnell (Würzburg, Germany)
Nurcan Üçeyler (Würzburg, Germany)
Philipp Wörsdörfer (Würzburg, Germany)
Anna Rockel (Würzburg, Germany)
schnell_a1@ukw.de

Abstract:
We present the development of a fully human in vitro assembloid model to study the pathophysiology of neuropathic pain, using Fabry disease — a rare X-linked lysosomal storage disorder — as a representative example. This model aims to replicate critical aspects of the human somatosensory system to better understand disease-related dysfunction and pain mechanisms.

Neural and mesodermal organoids were generated from human control iPSC lines using an adapted protocol based on Wörsdörfer et al. (2020), and next fused into neuro-mesodermal assembloids. These assembloids were structurally analyzed via fluorescence microscopy, focusing on peripheral nervous system (PNS) and central nervous system (CNS) progenitors, nerve fiber formation, and associated vascular components. The same protocol is currently being applied to assembloids generated from Fabry patient-derived iPSCs and their isogenic control with a corrected GLA mutation.

In control iPSC-derived assembloids, peripheral nerve fibers were observed forming between neural and mesodermal compartments. Schwann cell-like cells partially ensheathed these fibers. After 30 days of culture, ganglion-like structures resembling spinal ganglia emerged, indicating progressive neural maturation. Ongoing comparisons between Fabry and isogenic lines reveal phenotypic differences in nerve development and organization.

This neuro-mesodermal assembloid model successfully replicates key features of the human somatosensory system and serves as a promising tool for studying neuropathic pain. By enabling functional analysis under physiological conditions, it provides valuable insights into disorders like Fabry disease and holds potential for broader application in pain research and therapeutic development.

Poster 109:

Title:
Glioblastoma cells rarely invade the Hippocampus: Are specific extracellular matrix compounds the key?

Authors:
David Olschewski Ridruejo (Bochum, Germany)
Marie-Luise Kümmel (Bochum, Germany)
Maurice Meseke (Bochum, Germany)
Eckart Förster (Bochum, Germany)
David.OlschewskiRidruejo@ruhr-uni-bochum.de

Abstract:
Glioblastoma multiforme (GBM) is a highly malignant brain tumor characterized by a poor prognosis and low survival rates despite therapeutic advances. A major challenge in GBM treatment is the widespread infiltration throughout the brain parenchyma. Interestingly, GBM migratory routes tend to follow distinct anatomical structures and respect some anatomical borders. While migrating GBM cells frequently accumulate in regions like the caudate putamen, thalamus or pons, the hippocampus appears to resist to tumor invasion. This suggests that the hippocampal tissue contains molecular signals that hinder the infiltration by GBM migratory cells. Our study investigates how degradation of hyaluronan (HA), a large ECM carbohydrate which is produced and secreted by GBM, but also represents a major component of the hippocampal extracellular matrix (ECM), influences hippocampal infiltration by GBM.

By using different in vitro assays including MTT- and scratch-assays, BrdU-staining and scrape loading, we analyzed how degradation of GBM-derived HA impacts the metabolic activity, cell proliferation, migration and cell–cell communication of the rat glioblastoma C6 cell line.

Our preliminary data indicate that hyaluronidase treatment significantly reduces the metabolic activity of GBM without affecting its proliferative activity. Furthermore, we used a fluorescent ECM-dye to monitor ECM remodeling. In ongoing experiments, we investigate how degradation of GBM-derived HA and/or tissue-derived HA affects glioma migration by using a co-culture model of transplanted C6 glioma cells on organotypic rat hippocampal slice cultures.

The aim of our project is to identify ECM components that limit GBM infiltration in the hippocampus."

Poster 110:

Title:
Regional differences in hyaluronan localization in the murine outflow pathway during postnatal development

Authors:
Anja K. Hoffmann (Regensburg, Germany)
Ester Reina-Torres (London, United Kingdom)
Darryl R. Overby (London, United Kingdom)
Ernst R. Tamm (Regensburg, Germany)
Andrea E. Dillinger (Hamburg, Germany)
anja.hoffmann@vkl.uni-regensburg.de

Abstract:
The outflow resistance required to maintain intraocular pressure arises predominantly at the interface between the trabecular meshwork and the Schlemm's canal (SC) endothelium (inner-wall-region). The extracellular matrix of this region is thought to contribute to outflow resistance. This study investigates the spatial distribution and temporal appearance of hyaluronan in the murine outflow tract during postnatal development.

C57BL/6J wildtype mice were analyzed at postnatal days (P)1, 5, 7, 10, 15, 21, and 8 weeks. Meridional cryosections were labeled with fluorescent hyaluronan-binding-protein (BHABP) and SC was visualized using cluster-of-differentiation (CD)31 immunostaining. Corneoscleral wholemounts were co-labeled for BHABP and CD31 to assess the three-dimensional hyaluronan distribution. Wholemounts were prepared from eyes perfused (10–15mmHg) with FluoSphere™ fluorescent-tracer-microbeads to label segmental aqueous humour outflow pathways followed by BHABP labelling.

Hyaluronan first appeared in the trabecular meshwork at P15, coinciding with morphological maturation of the outflow system. CD31 co-labelling indicated a predominant localization of hyaluronan in the inner-wall-region. Wholemounts showed regional differences in hyaluronan staining intensity. Microbead perfusion identified high- and low-tracer-labelled-regions across the outflow circumference, indicative of regions of high vs low outflow filtration and segmental outflow. Some regions along the inner wall appeared to exhibit an inverse relationship between microbead accumulation and hyaluronan signal (i.e., high tracer in regions low in hyaluronan).

Hyaluronan emerges in the trabecular outflow pathway upon functional maturation. Its preferential localization in the inner-wall-region and regionally variable distribution suggest a role in modulating aqueous humor outflow resistance. Ongoing analysis is examining the spatial relationship between hyaluronan signal and microbead accumulation.

Poster 111:

Title:
Heterogeneous distribution of extracellular matrix components and lymphocytes in juvenile and adult human leptomeninges

Authors:
Corinna Fassin (Rostock, Germany)
Elise Vankriekelsvenne (Rostock, Germany)
Andreas Büttner (Rostock, Germany)
Markus Kipp (Rostock, Germany)
Sarah Joost (Rostock, Germany)
corinna.fassin@uni-rostock.de

Abstract:
The leptomeninges constitute a heterogeneous structure that plays a critical role in immunosurveillance and homeostasis of the central nervous system (CNS). Lymphocytes are key mediators of leptomeningeal immune responses, and their migration patterns can be influenced by the composition of the extracellular matrix (ECM). Meningeal disorders vary by age and region, suggesting that differences in leptomeningeal structure and immune cell distribution may be clinically relevant. We hypothesize that the human leptomeninges exhibit immune and structural heterogeneity, reflected by region- and age-specific differences in lymphocyte distribution and ECM composition.

Human leptomeninges from defined CNS regions were obtained from juvenile and adult post-mortem tissue. Lymphocyte distribution was quantified using immunohistochemical staining with antibodies against CD3, CD8, and CD20. Elastic, reticular, and collagen type I fibers were visualized through histological and immunohistochemical techniques. Quantitative analysis of lymphocyte distribution and qualitative assessment of ECM composition were performed to examine region- and age-related variation.

Lymphocyte densities varied between CNS regions and age groups, with juvenile samples exhibiting overall lower densities than adult ones. In both age groups, cerebellar and spinal regions displayed higher densities than cortical regions. While regional differences in ECM composition were less pronounced, age-related changes were evident across all components.

Our findings demonstrate both regional and age-related heterogeneity in lymphocyte distribution and ECM composition within the human leptomeninges, providing a morphological basis for understanding age- and region-specific leptomeningeal conditions.

Poster 112:

Title:
The role of Dab1 in reelin-induced modulation of the cholinergic system

Authors:
Marie-Luise Kümmel (Bochum, Germany)
Boris Burr (Bochum, Germany)
Maximilian Hausherr (Bochum, Germany)
Katrin Marcus-Alic (Bochum, Germany)
Eckart Förster (Bochum, Germany)
marie-luise.kuemmel@ruhr-uni-bochum.de

Abstract:
The extracellular matrix protein reelin plays a crucial role in neuronal migration and brain lamination during embryonic development. In the mature brain, reelin is mainly expressed by inhibitory interneurons and affects central neurophysiological processes, such as learning and memory, by modulating both excitatory glutamatergic and inhibitory GABAergic neuronal signaling.

By using calcium imaging of primary neurons, different neuronal cell lines and rodent brain slices, we found that reelin modulates acetylcholine-induced calcium signals.

Notably, reelin affected cholinergic signaling in both Dab1-deficient human neuroblastoma SHSY-5Y cells and Dab1-expressing rodent primary neurons. Since Dab1 is a key component of the canonical Reelin signaling pathway and, for example, essential for mediating Reelin's effects on glutamatergic signaling, it remains to be determined how Dab1-independent Reelin signaling is transmitted to modulate acetylcholine-induced calcium signals. To further investigate the role if Dab1 in this context, we are establishing a protocol to transfect Dab1-deficient SH-SY5Y cells with a Dab1-RFP-expression vector, allowing us to directly compare Dab1-dependent and Dab1-independent Reelin effects on cholinergic signaling in this cell line.

Furthermore, we want to take advantage of this experimental approach to investigate whether Reelin-induced changes in transcription factor expression, epigenetic protein modifications and proteome composition depend on Dab1-expression.

Poster 113:

Title:
Heterogeneous Patterns of Meningeal Immune Cell Accumulation in Models of Infectious and Autoimmune Neuroinflammation

Authors:
Tobias Giesbrecht (Rostock, Germany)
Elise Vankriekelsvenne (Rostock, Germany)
Johann Rößler (Rostock, Germany)
Jonas Keiler (Rostock, Germany)
Lars-Ove Brandenburg (Rostock, Germany)
Sarah Joost (Rostock, Germany)
tobias.giesbrecht@uni-rostock.de

Abstract:
The meninges are critical transit points for immune cells entering the central nervous system. Although clinical evidence points to regionally distinct infiltration patterns, the meninges are often regarded as an uniform structure. We hypothesize that the meninges are functionally heterogeneous, featuring specific "hotspots" for immune cell accumulation under neuroinflammatory conditions.

Contrast-enhanced micro-computed tomography (µCT) was used to generate 3D models of large, penetrating blood vessels and to map their distribution across different meningeal regions. Subsequently, meningeal immune cell distribution was investigated in murine models of autoimmune (Cuprizone-EAE) and infectious (bacterial meningitis) neuroinflammation. Immunohistochemical staining was used to analyze the distribution of key immune cell populations, including neutrophils (anti-Ly-6G) and T-cells (anti-CD3), relative to the vascular tree.

The analyses revealed distinct regional differences in leptomeningeal vascular architecture: the basal meninges featured larger and less penetrating arteries compared to the cortical meninges. The meningitis model was characterized by invasion of granulocytes that predominantly accumulated in the cortical meninges. In contrast, meningeal infiltration of T cells in the autoimmune EAE model was concentrated within the fissures of the brain.

These findings suggest that the accumulation of immune cells in the meninges during neuroinflammation follows highly organized, regional patterns. This highlights the functionally heterogeneous nature of the meninges and points to their active role in shaping disease-specific immune responses.

Poster 114:

Title:
Optimizing Metabolic Measurement of 3D Tumor Spheroids

Authors:
Urszula Hohmann (Halle, Germany)
Samiya Al-Robyai (Halle, Germany)
Andreas Simm (Halle, Germany)
Faramarz Dehghani (Halle, Germany)
Tim Hohmann (Halle, Germany)
urszula.hohmann@medizin.uni-halle.de

Abstract:
Tumor are characterized by a multitude of genetic and epigenetic alterations, including a deregulation of the metabolism, driving migration and infiltration. To mimic the energetic landscape of in vivo tumors, 3D models surpass traditional 2D cultures, by introducing regions of different nutrient and oxygen supply. Yet, the analysis of 3D metabolism, including the mitochondrial answer and extracellular fluxes is more challenging. The extracellular flux analyzer is a powerful tool for investigating cellular metabolism, offering valuable insights that can drive advancements in biomedical research, but protocols for analysis of 3D cultures are sparse.

Here, we present a protocol for optimized extracellular flux analysis, starting from the choice of the 3D culture model, dependencies on 3D culture size, testing multiple normalization approaches for two different glioblastoma and two primary cell lines.

It was demonstrated that our approach was feasible for different glioblastoma cell lines, showing cell type dependent responses to metabolic challenges and spheroid size. In addition, the analysis of data normalization approaches demonstrated that normalization approaches using essentially 2D characteristics of spheroids are insufficient to account for different spheroid sizes and cell lines. The data showed that using bio-printed spheroids using magnetic beads , combined with normalization to the initially seeded cell number, provided the most reliable normalization results.

Thus, we provided an approach that enables a straightforward and reproducible generation of 3D cell cultures and offers strategies to optimize metabolic measurements within these cultures.

Poster 115:

Title:
From macroscopy to microscopy: post mortem human tissue as valuable source to study brain architecture

Authors:
Sarah Woelfle (Ulm, Germany)
Michael Schoen (Ulm, Germany)
Tobias M. Boeckers (Ulm, Germany)
sarah.woelfle@uni-ulm.de

Abstract:
Besides different anatomical conditions, diverging cellular features were uncovered between human and rodent brains. However, the knowledge on synaptic subtypes and proteins is still largely based on rodent tissue. This is probably due to restricted access to human tissue and/or difficulties in immunostaining of long-term PFA-fixed tissue. We aimed to optimize the utility of human brain tissue from body donors for immunofluorescence microscopy. Finally, the autism-related protein ProSAP1/SHANK2 and presynaptic VGLUT1 were studied in human control brains.

Post mortem human brain tissue from permanent body donors of the gross anatomy course (they had given their consent to scientific use during their lifetime, a positive ethics committee vote has been obtained) was used to study synaptic proteins via immunofluorescence staining. Representative antibodies were tested in combination with the tissue clearing technique CLARITY.

VGLUT1 was identified as marker to delineate all layers in the human hippocampus. Second, CLARITY ameliorated immunofluorescence staining quality for some antibodies. An antibody toolbox covering synaptic and cellular proteins was established for perfusion- and immersion-fixed tissue. Finally, differences in SHANK2 protein expression were identified, i.e. in the human vs. mouse brainstem.

Brain tissue from permanent body donors is a valuable source for a better understanding of synaptic subtypes in the human brain. Growing evidence for diverging features between rodents and humans underlines the need for studies in human-derived tissue. In conclusion, the tissue source studied here can help to extend the knowledge on synaptic subtypes and finally unravel their involvement and decline during the progression of neurodegenerative diseases.

Poster 116:

Title:
Aging and memory - a matter of grey and white matter architecture?

Authors:
Safae El Khattabi (Düsseldorf, Deutschland)
Julian Quabs (Düsseldorf, Deutschland)
Nora Bittner (Düsseldorf, Deutschland)
Svenja Caspers (Düsseldorf, Deutschland)
safae.elkhattabi2@gmail.com

Abstract:
Age-related episodic memory decline is a major challenge in aging societies. While grey matter atrophy and white matter integrity loss have been linked to memory decline in small to medium-sized samples, their independent and combined contributions remain to be validated in large, population-based cohorts.

Using multimodal data from 900 participants of the 1000BRAINS cohort (mean age: 59) white matter integrity in fornix, uncinate fasciculus and hippocampal cingulum was assessed via atlas-based segmentation and quantified with NODDI parameters. Voxel- (CAT12) and surface-based (FreeSurfer) analyses identified grey matter regions associated with episodic memory, serving as seeds for exploratory tractography with bootstrapping-thresholded tracts. Multivariate regression analyses including structural measures, age, and sex modelled age-related structural memory decline (using p<0.05).

Higher neurite density in the fornix and uncinate fasciculus, and higher orientation dispersion (ODI) in the uncinate and hippocampal cingulum were associated with better memory; higher ODI in the fornix linked to reduced memory. Better performance was also associated with higher grey matter volume in cerebellum, prefrontal cortex, and medial temporal lobe. Tractography showed stronger cerebellar and frontal connectivity to frontal regions supported better memory, whereas stronger connections to the temporal lobe were linked to poorer performance. In the multivariate model, structural measures explained little variance, with age and sex significantly accounting for most.

Neither grey nor white matter strongly influenced episodic memory decline in our large cohort model. This suggests additional parameters may be needed and memory differences in aging are likely multifactorial, with structural factors contributing only limited to the overall effect.

Poster 117:

Title:
Tumor-nerve-interactions in pancreatic ductal adenocarcinoma

Authors:
Rebekka Christle (Heidelberg, Germany)
Thomas Kuner (Heidelberg, Germany)
Rohini Kuner (Heidelberg, Germany)
Nadja Bühler (Heidelberg, Germany)
Johannes Knabbe (Heidelberg, Germany)
Simone Hoppe (Heidelberg, Germany)
Yvette Dörflinger (Heidelberg, Germany)
rebekka.christle@uni-heidelberg.de

Abstract:
This project investigates the interaction between peripheral nerves and tumor cells in ductal pancreatic adenocarcinoma (PDAC) and how this contributes to pain and tumor progression. The central hypothesis is that sensory nerve fibers promote tumor growth and migration by directly interacting with cancer cells within the tumor and via perineural invasion, with synapse-like contacts driving tumor progression and pain.

This project applies a multifaceted approach to investigate tumor–nerve interactions and pain mechanisms in pancreatic cancer. Central to the study are mouse models in which sensory neurons (nociceptors) are selectively ablated. Pain behavior is assessed through standardized behavioral tests, while chemogenetic methods are used to modulate neuronal activity. Structural insights are gained through light, fluorescence, and electron microscopy, enabling detailed analysis of tissue architecture and synaptic organization.

Nerve ablation in genetically modified mice was used to study how neuronal loss affects tumor growth, behavior, weight, and pain; histological analyses confirmed successful ablation but revealed no major structural tumor differences except for a necrotic core. MicroCT imaging enabled precise tumor volume analysis. Initial findings suggest that nociceptive neurons do not significantly affect tumor progression. However, systemic ablation of nociceptors caused severe complications and premature death, halting further studies with this model.

Our findings highlight the complexity of tumor-nerve interactions in PDAC and suggest that systemic ablation of nociceptors cannot be used for this purpose. Future studies will focus on organ-specific ablation of nociceptors, targeting pancreatic nerves directly to gain deeper insights into their role in tumor progression and pain perception.

Poster 118:

Title:
Synaptic Lipid Modulation of Cortical Excitability: A Key Player in Motor Control and Brain Disorders

Authors:
Lara-Jane Kepser (Cologne, Germany)
Jan Götz (Cologne, Germany)
Johannes Vogt (Cologne, Germany)
lara-jane.kepser@uk-koeln.de

Abstract:
Bioactive synaptic lipids, specifically lysophosphatidic acid (LPA), play a vital role in modulating synaptic homeostasis and the excitation/inhibition (E/I) balance in cortical networks. Disruption of the plasticity-related gene 1 (PRG-1) protein, a key regulator of synaptic LPA, leads to enhanced glutamatergic release probabilities and increased neuronal excitability, ultimately shifting the cortical E/I balance. These shifts are associated with behavioral phenotypes such as hyperlocomotion and impaired sensorimotor gating, commonly observed in psychiatric disorders involving the dopaminergic system. Given PRG-1's expression in diverse motor control centers we aim to analyze how E/I balance alterations impact motor control at distinct anatomical levels.

To understand the consequences of synaptic lipid-induced E/I shifts, we investigated a range of mouse models, including systemic Prg-1 knockout mice, as well as mice with targeted deletions in specific cell types crucial for motor control within various brain regions. The neuronal populations investigated included striatal D1-expressing neurons, D2-expressing neurons and Purkinje neurons. Our comprehensive research involved electrophysiological, morphological, and behavioral evaluations.

Across the various mouse models investigated, we observed notable behavioral alterations. These included changes in motor coordination and function, indicating an impact on motor control at different levels. Furthermore, our findings showed modified exploratory behavior and altered anxiety levels when compared to controls.

Our research highlights the critical role of synaptic lipid-mediated E/I balance in regulating motor control and associated behaviors. The observed alterations in motor function, exploration, and anxiety across various models underscore the putative impact of synaptic lipid signaling on different motor aspects of brain disorders.

Poster 119:

Title:
Developmental Refinement of Synaptic Elements in the Murine Dentate Gyrus of Hippocampal Formation

Authors:
Christine Martin (Düsseldorf, Germany)
Elena Niggetiedt (Düsseldorf, Germany)
Max Anstötz (Düsseldorf, Germany)
christine.martin@uni-duesseldorf.de

Abstract:
Synaptic circuits play a fundamental role in processing, integration, and transmission of information within the nervous system, forming the basis for neural computation and behaviour. To explore how the synaptic architecture of the dentate gyrus in the hippocampal formation develops postnatally, we examined key structural features at distinct developmental stages in mice. Focussing on presynaptic boutons, the goal is to understand morphological changes and elucidate how functional synaptic networks mature during postnatal development.

We employed focused ion beam scanning electron microscopy (FIB-SEM) to reconstruct axons and dendrites forming excitatory synapses in three dimensions with nanometer resolution. Analyses included measurements of synaptic vesicle number, vesicle pool densities, vesicles within the readily releasable pool, dendritic spine morphology, and postsynaptic density area. Parameters were assessed for correlative patterns, allowing identification of systematic associations among synaptic features.

Quantitative analyses indicate that synaptic architecture varies across developmental stages, with specific structural features emerging over time. During postnatal development, presynaptic axon boutons undergo fundamental morphometric changes, indicating a higher degree of functional performance. These synaptic modifications encompass increased vesicle availability, elevated axonal mitochondrial presence, and the formation of additional release sites, accompanied by structural changes in dendritic spines.

Together, our findings highlight the dynamic and stage-specific reorganization of synaptic architecture during development. The observed structural variability and interdependence among key parameters suggest a maturation sequence in which an initial phase of synaptic refinement is followed by a subsequent phase of pruning. Both phases appear tightly controlled and contribute to the emergence of functionally optimized network architectures

Poster 120:

Title:
Characterization and number of nerve fibers in the human hypoglossal nerve

Authors:
Roland Blumer (Vienna, Austria)
Giorgi Didava (Vienna, Austria)
Arzu Petersen (Vienna, Austria)
Genova Carrero-Rojas (Vienna, Austria)
Paata Pruidze (Vienna, Austria)
Angel Pastor (Seville, Spain)
Wolfgang Weninger (Vienna, Austria)
roland.blumer@meduniwien.ac.at

Abstract:
The hypoglossal nerve (CN XII) plays a critical role in motor control of the tongue. The CN XII is considered a pure motor nerve, although animal studies and one human study indicate that it has a mixed fiber composition in the periphery. In this study, we analyzed the fiber composition of the human CN XII along its course.

Samples of the human CN XII were collected from locations that varied with respect to the ansa cervicalis, which temporarily connects to the CN XII. The following five sampling sites were selected: at the CN XII exit point (position 1), proximal to the ansa (position 2), at the level of the ansa (position 3), immediately distal to the ansa (position 4), and at the entry site to the tongue (position 5). Samples were subjected to immunofluorescence staining and semiautomated quantification.

At position 1, the mean total number of axons was 6,446.3 ± 1,494.7. All axons were myelinated and cholinergic. At position 2, the hypoglossal nerve exhibited a mixed fiber composition. Besides cholinergic fibers, a low number of myelinated non-cholinergic (90.7 ± 45.6) and sympathetic fibers (393 ± 157.6.) were present. The mixed fiber composition of the CN XII persisted in positions 3 through 5, and importantly, the number of cholinergic (7.456,25 ± 883), non-cholinergic (1,453 ± 518.4), and sympathetic fibers (2.312,5 ± 599) significantly increased.

The present findings suggest that the ansa cervicalis makes a substantial contribution to the fiber composition of the distal hypoglossal nerve.

Poster 121:

Title:
Activity-dependent postsynaptic PRG-1 function links lipid metabolism to neuronal homeostasis and proper cortical information processing

Authors:
Haichao Ji (Cologne, Germany)
Heiko Endle (Cologne, Germany)
Guilherme Horta (Mainz, Germany)
Konstantin Radyuschkin (Mainz, Germany)
Thomas Deller (Frankfurt am Main, Germany)
Robert Nitsch (Münster, Germany)
Johannes Vogt (Cologne, Germany)
heiko.endle@uk-koeln.de

Abstract:
Neuronal circuit and network homeostasis are prerequisites for proper brain function. The postsynaptically located transmembrane protein plasticity-related gene 1 (PRG-1), plays a crucial role in the modulation of glutamate release via removal of lysophophatidic acid (LPA) from the synaptic cleft. This implies that regulation of PRG-1's LPA-uptake function plays a critical role in cortical information processing, yet intracellular regulation of PRG-1's LPA-uptake remains to be fully elucidated.

We used primary neurons to identify an activity dependently cleaved region of the PRG-1 c-terminal domain. We then used pharmacological tools to identify the protease and ion channels responsible for PRG-1's cleavage and assessed effects on PRG-1 function using labelled LPA. Finally, we generated a mouse line missing the protease binding site and used behavioral and electrophysiological readouts to assess the consequences of PRG-1 deregulation.

Here, we show that following sustained neuronal activity, the C-terminal intracellular domain of PRG-1 undergoes a two-step molecular modification. First, postsynaptic Ca2+-increase induces CaM binding to PRG-1 inhibiting its LPA-uptake function. Upon further stimulation, NMDA receptor signaling activates calpain, which cleaves the C-terminal PRG-1 domain resulting in an increased LPA-uptake of the remaining transmembrane domain. A non-cleavable PRG-1 variant (PRG-1Δ515-523) shows impaired neuronal homeostasic regulation, which affects cortical information processing as shown by impairments in some forms of LTP as well as worse learning performance in complex learning tasks of animals carrying the mutation.

We describe a novel mechanism hinging on PRG-1 modifications regulating synaptic hemostasis, which is important for proper cognitive function.

Poster 122:

Title:
Ultrastructural and Functional Analysis of Perforant Path-Granule Cell Synapses in the Murine Hippocampus across Postnatal Development

Authors:
Elena Niggetiedt (Düsseldorf, Germany)
Joel Nelson (Düsseldorf, Germany)
Christine Martin (Düsseldorf, Germany)
Christine R. Rose (Düsseldorf, Germany)
Max Anstötz (Düsseldorf, Germany)
elena.niggetiedt@hhu.de

Abstract:
As part of the hippocampal formation, the dentate gyrus is critically involved in cognitive functions such as learning and memory and is implicated in neurological disorders, including temporal lobe epilepsy and depression. Investigating the development of hippocampal networks is fundamental to understanding both physiological and pathological mechanisms. This study examines age-related morphological changes in perforant path–granule cell synapses in the outer molecular layer (OML), considering their potential functional relevance.

We used focused ion beam scanning electron microscopy (FIB-SEM) to generate nanoscale 3D image stacks of the OML in C57BL/6 mice at three developmental stages: juvenile (2 weeks), adolescent (8 weeks), and fully mature (16 weeks). These enabled detailed morphological analysis of postsynaptic granule cell structures. To assess the potential functional impact of these structural alterations, we implemented a computational spine model based on our FIB-SEM reconstructions and applied fluorescence recovery after photobleaching (FRAP) experiments using two-photon microscopy to measure diffusion dynamics in granule cell spines.

Our data indicate that granule cell spine shape changes during development. Particularly the region of the spine neck reshapes significantly. The experimental diffusion measurements obtained via FRAP were consistent with simulated calcium gradients, supporting a link between spine morphology and diffusion properties.

The observed postsynaptic changes in the murine dentate gyrus reveal a potential new mechanism contributing to increased synaptic plasticity. Our combined experimental and computational findings support the hypothesis that spine neck remodeling is associated with improved memory function during hippocampal maturation.

Poster 123:

Title:
Neuroprotective role of Endothelin signaling in the retina

Authors:
Andrea E. Dillinger (Hamburg, Germany)
Sabrina I. Schmitt (Regensburg, Germany)
Ernst R. Tamm (Regensburg, Germany)
Anja Schlecht (Hamburg, Germany)
Barbara M. Braunger (Hamburg, Germany)
a.dillinger@uke.de

Abstract:
Vision loss in retinal degenerations is caused by a progressive loss of retinal photoreceptors. Endothelin-receptor-b (ETb) and Endothelin-(ET)-2 are upregulated after light-induced photoreceptor degeneration. In this study we analyzed the role of ETb in photoreceptor degeneration.

Etb expression was studied in wildtype retinae by RT-PCR and in-situ-hybridization. α-Cre;ETbfl/fl mice (ETbΔOC), harboring a deletion of ETb in retinal neurons and Müller cells, were analyzed. ETbfl/fl littermates were used as controls. Etb-deficiency was verified by RT-PCR, Western-Blot and in-situ-hybridization. Morphometric analyses of the inner nuclear layer (INL) and outer nuclear layer (ONL) and immunohistochemistry of Glial-Fibrillary-Acidic-Protein, glutamine-synthetase and ionized-calcium-binding-adaptor-molecule-1 were performed. Light exposure (5000 lux, 1 h) was used to induce photoreceptor degeneration. TdT-mediated-dUTP-biotin-nick-end-labeling and morphometric analysis of the ONL-thickness were performed to determine cell death. Endothelin signaling, pro- and antiapoptotic molecules, and neuroprotective factors were analyzed by RT-PCR.

Retinal expression of ETb is higher than ETa. ETb is highly expressed in the ganglion cell layer, INL and ONL. ETb was significantly reduced in ETbΔOC. Semithin sections of ETbΔOC mice showed no alterations in ONL- and INL-thickness. There were no signs of retinal glia reactivity. Light exposure resulted in a significantly higher number of apoptotic cells and a thinner ONL in ETbΔOC. Components of the endothelin signaling pathway were significantly increased in ETbΔOC retinae, concomitant with a significant increase Caspase 8, Fgf2 and Lif expression following light exposure.

We identified ETb-mediated signaling as novel neuroprotective pathway in photoreceptor degeneration, which may provide a molecular basis for future therapeutic approaches in retinal degeneration.

Poster 124:

Title:
CD44 Expression as a Marker in Alzheimer’s Disease and Multiple Sclerosis Models

Authors:
Luisa Burkert (Rostock, Germany)
Lars-Ove Brandenburg (Rostock, Germany)
Markus Kipp (Rostock, Germany)
Newshan Behrangi (Rostock, Germany)
Tanja Hochstrasser (Munich, Germany)
luisa.burkert@med.uni-rostock.de

Abstract:
CD44 is a cell surface receptor for hyaluronate that is broadly expressed across diverse cell types and is involved in cell-matrix adhesion, cell-cell interactions, proliferation, apoptosis, and the regulation of tissue architecture and inflammation. Dysregulation of CD44 has been implicated in various inflammatory and neurodegenerative diseases. In this study, we tested the hypothesis that CD44 expression is upregulated in response to neuroinflammation and neurodegeneration, with a focus on multiple sclerosis (MS) and Alzheimer’s disease.

We analyzed tissues from two murine models: experimental autoimmune encephalomyelitis (EAE) and APP/PS1 transgenic mice. Immunohistochemical staining followed by densitometric analysis was performed to assess CD44 expression.

In the EAE model, CD44 was markedly upregulated in the spinal cord white matter relative to controls, correlating with elevated inflammation demonstrated by CD3 immunoreactivity. In APP/PS1 mice, CD44 expression was increased in the hippocampus and localized to focal lesions, as also visualized by APPA4 staining.

These findings support the conclusion that neuroinflammation and neurodegeneration are associated with increased CD44 expression in the central nervous system (CNS), suggesting a potential role for CD44 in the pathophysiology of MS and Alzheimer’s disease.

Poster 125:

Title:
IOP but not age increases loss of optic nerve axons in mice with a deficiency in macroglial intermediate filaments

Authors:
Magdalena Gebert (Regensburg, Germany)
Leni Schnappauf (Regensburg, Germany)
Yao Zhang (Regensburg, Germany)
Johanna Heimbucher (Regensburg, Germany)
Ernst Tamm (Regensburg, Germany)
magdalena.gebert@ur.de

Abstract:
Glial fibrillary acid protein (GFAP) and Vimentin (VIM) are major intermediate filament components of astrocytes and contribute significantly to their biological functions. In human and experimental glaucoma, astrocytes show signs of reactivity, distinct morphological changes and an increased immunoreactivity for GFAP and VIM. Several studies in animal models of glaucoma reported on an association of the reactive phenotype with severe axon loss. In this study we aimed to investigate how a complete deficiency in GFAP and VIM influences retinal ganglion cell (RGC) survival under ocular hypertension and with increasing age.

GFAP and VIM deficiency was confirmed by Western Blot. Ocular morphology was analyzed using semi-thin sections. Ocular hypertension was induced by injection of magnetic microbeads. Intraocular pressure (IOP) was monitored weekly. Myelinated ON axons were counted in PPD-stained ON cross-sections, RGC somata were quantified in retinal wholemounts.

Deficiency in GFAP and VIM had no observable effects on ocular morphology, axon number or IOP. After 6 weeks of ocular hypertension axon and RGC loss was significantly higher in GFAP-/- VIM-/-. Axon and RGC numbers declined with increasing age in all animals. No statistically significant differences were found between GFAP-/- VIM-/- and wildtype animals at 6 and 12 month of age.

Our findings strongly indicate, that macroglial intermediate filaments play a crucial role in the eyes ability to cope with ocular hypertension. It remains to be elucidated if the observed negative effect is caused by biomechanical alterations at the optic nerve head or by altered astrocyte and/or Müller cell homeostatic functions.

Poster 126:

Title:
Profiling neuronal mechanisms counteracting progressive mitochondrial dysfunction

Authors:
Lisa Wolters (Mannheim, Deutschland)
Femke Groeneweg (Mannheim, Germany)
Marcin Luzarowski (Heidelberg, Germany)
Nina Bonekamp (Mannheim, Germany)
Christian Schultz (Mannheim, Germany)
lisa.wolters@medma.uni-heidelberg.de

Abstract:
Mitochondrial diseases, primarily caused by disruption of mitochondrial gene expression, often manifest as neurological disease. The clinical phenotype may differ markedly between mitochondrial disease patients, resulting in an extremely varied genotype-phenotype relationship. Thus, certain genetic factors and/or signalling pathways likely modify the clinical range of mitochondrial disorders. Furthermore, different cell types exhibit different resilience towards mitochondrial dysfunction. Neurons show a surprising ability to sustain mitochondrial dysfunction for prolonged periods. To understand neuronal resilience, we employed a time-course assay using inhibitors of mitochondrial transcription (IMTs) in primary cortical neurons to profile cellular responses to progressive mitochondrial dysfunction.

Dose/time-responses of primary cortical neurons on IMT were analyzed by RT-qPCR, immunoblotting, viability assay, and immunofluorescence. Proteomic changes upon IMT treatment were analyzed by quantitative proteomics.

Although mitochondrial transcripts were rapidly depleted at all concentrations, mitochondrially-encoded protein depletion only occurred at the highest concentration after prolonged IMT treatment. Cell viability remained unaffected up to 14 days post-treatment (dpt). However, the synaptic density was depleted 7 dpt, which is in line with a progressive neuronal phenotype. Quantitative proteomics revealed a dysregulation of proteins linked to neurodegeneration, mitochondrial translation, and energy metabolism. Furthermore, we found alterations of the proteolytic/autophagic system, such as the E3 ubiquitin ligase Nedd4l.

Nedd4l downregulation has been reported to contribute to an increased mitochondrial metabolism and induction of autophagy via stabilization of Ulk1, an important regulator of autophagy. We hypothesize that the dysregulation of proteins involved in the proteolytic/autophagic system contributes to the resilience of neurons towards mitochondrial dysfunction, warranting further investigation.

Poster 127:

Title:
Modulation of SAPK/JNK Signaling and Cell-Cell Interactions of Brain Microvascular Endothelial Cells: Effects of Lipocalin 2

Authors:
Franziska Pfeiffer (Bonn, Germany)
Fabio Zillmer (Bonn, Germany)
Natalie Tillmann (Bonn, Germany)
Tim Clarner (Bonn, Germany)
franziska_pfeiffer@uni-bonn.de

Abstract:
Lipocalin 2 (LCN2), released by reactive astrocytes during neuroinflammation, is thought to influence endothelial cells of the blood–brain barrier (BBB), potentially stabilizing permeability and limiting immune cell infiltration. This study aims at exploring the underlying molecular mechanisms.

In a first step, we confirmed the expression of the LCN2 receptor, 24p3R, in cultured human brain endothelial cells (hCMEC/D3). Then, to mimic brain inflammatory processes, cells were exposed to TNF-ALPHA or IFN-GAMMA in combination with LCN2. SAPK/JNK pathway activity was analyzed using phosphorylation blots for JNK and c-Jun. As additional readout, changes in tight junction gene expression were assessed using a RT-qPCR array addressing endothelial cell-cell interactions. Functional consequences of LCN2 signaling were further examined via transendothelial electrical resistance (TEER) measurements.

LCN2 reduced cytokine-induced phosphorylation of JNK, indicating inhibition of the pro-inflammatory SAPK/JNK signaling cascade. Co-treatment with LCN2 and inflammatory cytokines led to distinct, context-dependent regulation of tight junction-related genes, including TJAP1, HCLS1 and ICAM1. Despite these molecular changes, TEER measurements did not reveal significant alterations in barrier integrity at least at the investigated time points.

LCN2 modulates the inflammatory response of endothelial cells to inflammatory cytokines and changes gene expression patterns of cell-cell contacts. These findings support a role for LCN2 in preserving BBB stability during inflammation through selective, context-dependent signaling.

Poster 128:

Title:
Regional oligodendrocyte vulnerability and microglial CD83 expression in the cuprizone-model

Authors:
Sophia Meien (Rostock, Deutschland)
Hannes Kaddatz (Rostock, Germany)
Markus Kipp (Rostock, Germany)
sophia.meien@uni-rostock.de

Abstract:
Demyelination, characterized by oligodendrocyte loss and myelin damage, is a hallmark of neurological diseases like multiple sclerosis (MS). MS lesions exhibit a heterogeneous topographic distribution, with certain CNS regions more susceptible than others. Similarly, the cuprizone mouse model induces region-specific demyelination, affecting areas such as the corpus callosum and cortex, while regions like the spinal cord and fimbria hippocampi remain largely unaffected ('pseudoresistant'). This study investigates regional differences in oligodendrocyte vulnerability and the potential modulatory role of microglial CD83 expression. We hypothesize that regional oligodendrocyte stress resistance and elevated microglial CD83 in pseudoresistant areas reflect protective processes contributing to the spatial lesion patterns in MS.

C57BL/6 mice were intoxicated with cuprizone. Immunohistochemistry and immunofluorescence assessed myelin integrity (PLP), microglia density (IBA1), oligodendrocyte stress (ATF3, SerpinA3N) and oligodendrocyte density (CA2). Quantitative PCR measured mRNA levels of Atf3, Serpina3n and Cd83 in region-specific CNS samples.

PLP staining revealed pronounced demyelination in vulnerable regions, whereas pseudoresistant areas exhibited no significant demyelination. Despite myelin preservation, ATF3 and SerpinA3N were expressed in oligodendrocytes within pseudoresistant regions, indicating sublethal cellular stress without oligodendrocyte cell loss. qPCR analyses showed increased Cd83 expression in the spinal cord compared to the corpus callosum.

Our findings suggest that oligodendrocytes in pseudoresistant regions experience cellular stress without substantial cell loss. The elevated Cd83 expression in the spinal cord relative to the corpus callosum supports the hypothesis that region-specific microglial CD83 may protect against demyelination. Clarifying CD83’s role in microglial-oligodendrocyte interactions could reveal new therapeutic strategies for demyelinating diseases.

Poster 129:

Title:
Neuroprotection of Progesterone for the Enteric Nervous System Cells on the Model of Parkinson's Disease

Authors:
Alexandra Yurchenko (Bochum, Germany)
Sarah Stahlke (Bochum, Germany)
Carsten Theiss (Bochum, Germany)
Alexandra.Yurchenko@edu.ruhr-uni-bochum.de

Abstract:
Parkinson's disease (PD) is increasingly recognized as a systemic disorder with early pathology in the enteric nervous system (ENS). Considering age and sex as important risk factors in PD progression, we aimed to investigate the neuroprotective effects of progesterone on primary ENS cells. Special attention was given to the progesterone receptor membrane component 1 (PGRMC1), potentially involved in mitochondrial regulation and cell survival.

We established a protocol for isolating and cultivating primary ENS cells from male and female Wistar rats at different life stages: postnatal day 30 (prepubescent), over 65 days to under 16 months (sexually mature), and over 16 months (aged/postmenopausal). To model PD-related mitochondrial dysfunction, cells were treated with rotenone, with or without co-treatment with progesterone and the PGRMC1 antagonist AG-205. For all age groups, RT-qPCR was used to assess expression profiles of progesterone receptors, including PGRMC1.

Progesterone significantly improved neuronal survival in rotenone-treated ENS cultures. This protective effect was abolished when AG-205 was added, suggesting that the PGRMC1-mediated non-genomic pathway plays a key role. RT-qPCR confirmed the presence of key progesterone receptors across all age groups, with PGRMC1 showing the highest expression levels.

These results indicate that progesterone may protect ENS neurons through PGRMC1-dependent mechanisms, relevant to age- and sex-related PD vulnerability. This experimental model supports further investigation into human ENS cultures and mitochondrial pathways involved in the gut-brain axis.

Poster 130:

Title:
Mismatch repair protein MSH3 and somatic instability in human Huntington brain

Authors:
Marie-Louisa Schoebel (Bochum, Germany)
Paul Günther Scheunemann (Bochum, Germany)
Juan Azad (Bochum, Germany)
Marie-Luise Kuemmel (Bochum, Germany)
Carsten Saft (Bochum, Germany)
Huu Phuc Nguyen (Bochum, Germany)
Eckart Foerster (Bochum, Germany)
Larissa Arning (Bochum, Germany)
Elisabeth Petrasch-Parwez (Bochum, Germany)
Marie-Louisa.Schoebel@ruhr-uni-bochum.de

Abstract:
The expanded cytosine-adenine-guanine (CAG) trinucleotide repeat in the Huntingtin (HTT) gene, which causes Huntington´s disease (HD), has a high tendency for further expansion in somatic cells, driving the onset and course of the disease. The DNA mismatch repair protein MSH3, also linked to somatic CAG expansion, may have an important impact on HD neuropathology. To further elucidate the regional expression pattern of MSH3 in HD, so far only studied in cell cultures and animal models, we here investigated the distribution in human HD brain areas with known somatic instability.

Postmortem brains from clinically well-characterized HD patients with different CAG repeat lenghts were analyzed by MSH3 peroxidase- and fluorescence immunohistochemistry. Additional markers were used for regional and cellular identification; electron microscopy was used to evaluate the degeneration state.

In most HD patients, investigated MSH3 immunostaining was localized in a layer-specific manner in nuclei of cortical pyramidal neurons, suggesting selective vulnerability of certain cell types, confirmed by electron microscopy. In the less affected cerebellum consistent nuclear Purkinje and granule cell staining was observed across all patients. Striatal neurons show individually varying MSH3 immunoreactivity, partly reflecting the heterogenous neurodegeneration. A positive glial reaction may reflect to which extent MSH3 is involved in degeneration.

This study shows for the first time a complex cell-type specific nuclear localization of MSH3 protein in various HD brain areas, partly reflecting the vulnerability of the affected neurons. A consistent correlation between regional MSH3 expression and CAG instability has not yet been established and needs to be further investigated.

Poster 131:

Title:
Cell non-autonomous proteostasis crosstalk in neurodegeneration models

Authors:
Lara Aletta Gruijs da Silva (Cologne, Germany)
Elke Franke (Cologne, Germany)
Irina Dudanova (Cologne, Germany)
lara.gruijs-da-silva@uk-koeln.de

Abstract:
Protein aggregates, a result of proteostasis failure, are initially present in neurodegenerative diseases in a subset of neurons, then gradually spread to other brain regions along neuronal projections, suggesting involvement of cell non-autonomous mechanisms. How cell-cell interactions disturb proteostasis in initially unaffected neurons that later develop pathology is unknown. Our hypothesis is that spreading of aggregates in the brain involves prior cell non-autonomous proteostasis crosstalk between different neuronal populations.

Proteostasis disturbances were analyzed using EGFP-fused firefly luciferase (Fluc-EGFP) as a proteostasis sensor. Alterations in Fluc-EGFP were studied through immunofluorescence to discern Fluc-EGFP foci that form due to reduced solubility of the sensor, and luciferase assay to detect variations in its enzymatic activity. Our previous observations indicate that Fluc-EGFP reacts to proteostasis defects caused by protein misfolding before the formation of protein aggregates. Furthermore, stable cell lines expressing Fluc-EGFP as well as lines propagating tau aggregates were established.

We show that the Fluc-EGFP stable cell line reliably detects proteostasis disturbances caused by different types of proteotoxic stress, including proteins known to aggregate in neurodegenerative disorders. Furthermore, preliminary findings indicate cell non-autonomous proteostasis alterations in Fluc-EGFP cell lines in response to aggregated tau.

Our new tools set the stage for further detailed mechanistic dissection of cell-to-cell proteostasis crosstalk in neurons. Overall, this project will uncover cell non-autonomous effects of protein misfolding on the proteostasis of initially intact neurons that become affected as a result of protein aggregate spreading, and pinpoint molecular pathways that could be targeted to prevent pathology spreading.

Poster 132:

Title:
Interplay between TDP-43 and PGRN in human microglia

Authors:
Buse Özbaykent (Munich, Germany)
buse.oezbaykenr@bmc.med.lmu.de

Abstract:
To investigate how progranulin (GRN) deficiency alters human iPSC-derived microglial stress responses, with a focus on cytoplasmic TDP-43 aggregation, organelle stress and metabolic reprogramming.

Human iPSC-derived microglia (WT and GRN KO) were generated and characterized by phagocytosis assay and immunostainings for Iba1, TMEM119, CD68, and P2RY12. Cells were exposed to different stressors to induce activation. TDP-43 aggregation, including localization to ER and mitochondria, was assessed by immunostaining. ER-Golgi integrity was analyzed via microscopy. Single-cell RNA-seq profiled transcriptional signatures. Translation rates were measured post-activation via OPP assay. Metabolic states were assessed using FLIM imaging.

GRN KO i-microglia showed increased baseline CD68 expression, suggesting a primed reactive state. Upon H_2O_2 treatment, GRN KO cells formed TDP-43 aggregates localized to both ER and mitochondria, while WT formed only stress granules. Microscopy revealed uncoupled ER-Golgi networks in GRN KO even before stress, with further fragmentation upon activation. Single-cell analysis showed upregulated UPR genes and a shift from oxidative phosphorylation to glycolysis in GRN KO microglia. Translation rates globally decreased after activation, consistent with ER stress responses.

Our data demonstrate that GRN deficiency predisposes human microglia to aberrant stress responses characterized by TDP-43 aggregation at both ER and mitochondria, ER stress, disrupted organelle interactions, and metabolic reprogramming. These findings highlight GRN’s essential role in maintaining microglial homeostasis and suggest mechanisms linking GRN mutations to TDP-43 proteinopathies in neurodegeneration.

Poster 133:

Title:
Differential Expression of Histone Modifiers in Microglia: A Response to TGfbeta1

Authors:
Leonie Johanna Lancelle (Bielefeld, Germany)
Phani Sankar Potru (Bielefeld, Germany)
Björn Spittau (Bielefeld, Germany)
leonie.lancelle@uni-bielefeld.de

Abstract:
Microglia, the immune cells of the CNS, play crucial roles in brain development, maintaining balance, and responding to immune challenges. The multifunctional protein TGfbeta1 is important for both their function and regulation of homeostasis. and their reaction to neurodegenerative processes. Impairment of the signaling through TGfbeta1 leads to inflammatory reactions and impaired homeostasis of the brain.

In our study we utilized micro-array data of microglia treated with TGfbeta1 for 1 h and control microglia without treatment. R was used to analyze differences in the expression levels of histone modifiers and differences of the overall gene expression between both sample groups. Further, the data was analyzed through Pathway analysis and Co-expression analysis to find co-regulated genes and potential regulators.

Transcriptomic data were generated through a micro-array of microglia samples treated with a solution containing Tgfbeta1 for 1 h and control samples of untreated microglia. The generated data shows distinct differences between microglia that were treated with Tgfbeta1 compared to the untreated ones. We were able to gain further insight into changes in the gene expression and potential changes in the downstream effect of genes on the chromatin level through clustering, differential gene expression analysis and Co-expression network analysis.

This study provides new insights into the effect of Tgfbeta1 on microglia, their gene expression pattern, and possible downstream effects altering chromatin accessibility and gene regulation.

Poster 134:

Title:
Cholecystokinin attenuates Inflammation-induced Impairments of Purkinje Cell Dendritogenesis

Authors:
Fabienne Lülsberg (Bonn, Germany)
Mohammed Bahaaeldin (Bonn, Germany)
Carolin Bülte (Bonn, Germany)
Karl Schilling (Bonn, Germany)
Stephan Baader (Bonn, Germany)
fabienne.luelsberg@ukbonn.de

Abstract:
Neuroinflammation during early brain development is increasingly recognized as a contributing factor to psychiatric disorders such as autism spectrum disorder (ASD) and schizophrenia. Cholecystokinin-8 (CCK-8), a gastrointestinal peptide, is an important mediator of the gut-brain axis and was suggested to function as a modulator of neurotransmission affecting ASD. Currently, no morphological correlates are known, and signalling mechanisms have not been addressed experimentally. The cerebellum has been frequently implicated in autism-related pathology, with Purkinje cells (PCs) playing a central role in cerebellar development and function. This project investigates how lipopolysaccharide (LPS)-induced inflammation affects PC dendritogenesis and whether CCK-8 can modulate these effects.

To analyze PC dendritogenesis and microglia interactions under in-vivo-like conditions, we used immunohistochemically stained slice cultures treated with various agonists and antagonists. 3D images of these slices were semiautomatically analyzed using Imaris.

We show that LPS-treatment reduced PC dendritogenesis. PCs displayed shorted dendrites, less branches and less complex dendritic arbors. The culture medium contained elevated levels of tumor necrosis factor alpha (TNF-α) following LPS stimulation. Blocking TNF-α receptor signalling and microglial proliferation rescued dendritic growth. CCK-8 application reduced inflammation-induced dendritic deficits in wild-type mice. This anti-inflammatory effect of CCK could be diminished in CCK receptor knockout mice, suggesting a receptor- mediated neuroprotective effect of CCK-8.

Our findings show that LPS-induced inflammation disrupts PC dendritogenesis, with microglia and TNF-α as key mediators. CCK-8 exerts a protective effect via CCK receptors, highlighting its potential role in modulating neuroinflammation during brain development.

Poster 135:

Title:
Preventing Oligodendrocyte Loss in Demyelinating Diseases: Insights into Ferroptosis

Authors:
Coram Guevara (Bonn, Germany)
Olida Aydin (Bonn, Germany)
Natalie Tillmann (Bonn, Germany)
Fernando Ortiz (Santiago de Chile, Chile)
Tim Clarner (Bonn, Germany)
guevara@uni-bonn.de

Abstract:
Multiple sclerosis (MS) is a chronic central nervous system disease characterized by demyelination, inflammation, oligodendrocyte (OL) loss, and neurodegeneration. These pathological changes are consequence of disruptions in redox homeostasis and chronic inflammation, where the Nrf2 pathway plays a central regulatory role. While TGF-β1 is well known for its immunomodulatory functions in MS, recent findings suggest it also contributes to redox regulation through Nrf2 activation. However, the mechanisms and relevance of this TGF-β1/Nrf2 crosstalk in MS remain unclear. Disturbances in redox balance have been implicated in ferroptosis, an iron-dependent form of cell death increasingly recognized as a key driver of OL loss during demyelination. We hypothesize that Nrf2 activation protects OLs from ferroptosis, potentially under the influence of TGF-β1. We aim to investigate the interplay between TGF-β1 signaling and Nrf2 activation, and the potential of Nrf2 activity to protect OLs from ferroptosis.

We employed the cuprizone model of demyelination and pharmacological inhibition of TGF-β receptor 1 (Galunisertib) to assess effects on Nrf2 signaling. Behavioral assessments included memory and locomotor tests. An in vitro ferroptosis model (erastin-treated Oli-Neu cells) was used to evaluate Nrf2-dependent protection.

Galunisertib reduced TGF-β1 and Nrf2 protein levels in brain tissue, accompanied by impaired memory and locomotor performance. These deficits were reversed by pharmacological Nrf2 activation. In vitro, Nrf2 activation significantly attenuated erastin-induced ferroptosis in OL cells.

Our findings suggest that TGF-β1 contributes to Nrf2 pathway activation, with protective effects against OL loss and functional impairment in demyelination. Targeting this axis may offer a therapeutic strategy in MS.

Poster 136:

Title:
New Insights into ALS Pathology: Histomorphological Analysis of Intestinal Structures in the Wobbler Mouse

Authors:
Jonathan Paul Langer (Bochum, Germany)
Veronika Matschke (Bochum, Germany)
Carsten Theiss (Bochum, Germany)
jonathan.langer-v4j@ruhr-uni-bochum.de

Abstract:
Amyotrophic lateral sclerosis is a progressive neurodegenerative disorder traditionally considered to primarily affect the central nervous system. Recent investigations, particularly in transgenic mice model, suggest that systemic alterations, especially in the gastrointestinal tract, may play a critical role in disease progression. However, for the Wobbler mouse, an animal model for sporadic ALS, data on such changes are still scarce. In order to contribute to closing this knowledge gap, we aim to morphologically and functionally characterize the gastrointestinal tract to elucidate its role in ALS pathogenesis during age progression in the Wobbler mouse.

Intestinal samples from Wobbler and wild-type mice were collected at three developmental stages (p20, p40, and p60), processed using the Swiss-roll technique, paraffin-embedded, stained (HE, Goldner), and analysed morphometrically. Variable linear parameters such as villus height and muscularis thickness, as well as quantitative parameters like the number of immune cell infiltrates, goblet cells, and Paneth cells, were assessed.

Analyses reveal structural differences in the gastrointestinal tract between Wobbler and wild-type mice that vary across developmental stages. Morphometric parameters such as villus height, crypt depth, and muscularis thickness show genotype-dependent deviations, with Wobbler mice generally exhibiting reduced values compared to wild-type-controls. In contrast, quantitative parameters tend to show no consistent significant differences between genotypes.

These findings suggest that structural alterations occur in the GI tract of Wobbler mice, potentially indicating functional impairments such as reduced peristalsis or absorption capacity. The results support the hypothesis that pathological processes within the intestine may be relevant to ALS pathogenesis.

Poster 137:

Title:
CPI-17 as a Protective Factor in Neuroinflammation

Authors:
Lennart Meinke (Rostock, Germany)
lennart.meinke@uni-rostock.de

Abstract:
CPI-17 is an inhibitor of myosin light chain phosphatase expressed in various cell types, including oligodendrocytes, where it has shown protective effects in demyelination. Its expression in Purkinje cells, however, remains uncertain. We investigated whether CPI-17 confers protective effects in neuroinflammation (e.g., multiple sclerosis) and whether it is expressed in Purkinje cells.

Wild-type and CPI-17 knockout mice were subjected to an experimental autoimmune encephalomyelitis (EAE) model. Paraffin-embedded spinal cord and cerebellar sections from non-EAE mice were analyzed using immunohistochemistry (IHC).

CPI-17 knockout mice exhibited more severe clinical EAE symptoms. However, IHC did not reveal increased densities of T cells or microglia. Purkinje cells showed a CPI-17–positive signal in both wild-type and knockout mice at high antibody concentrations, but this signal was lost at lower concentrations and considered nonspecific.

We found no evidence of increased inflammatory cell infiltration in CPI-17 knockout mice. Our results suggest that CPI-17 is not expressed in Purkinje cells.

Poster 138:

Title:
ZP2-mediated signaling cascade in capacitated spermatozoa

Authors:
Lorena Sunjic (Essen, Germany)
Caroline Wiesehoefer (Essen, Germany)
Jong-Nam Oh (New Haven, CT, US)
Marc Wiesehoefer (Essen, Germany)
Jean-Ju Chung (New Haven, CT, US)
Gunther Wennemuth (Essen, Germany)
lorena.sunjic@uk-essen.de

Abstract:
Zona pellucida protein 2 (ZP2) is an important glycoprotein of the extracellular matrix of the ovum. ZP2 serves as a primary sperm receptor and induces a change in sperm motility (Wiesehoefer et al., 2022), which is necessary for successful fertilization of the oocyte. The aim of this study is to investigate the signaling cascade of the ZP2-induced change in sperm motility.

The induction of the acrosome reaction by ZP2 was assessed using three independent approaches: FITC-PSA staining, CTC staining, and spermatozoa from a transgenic SU9/Acr3 mouse line with GFP-tagged acrosin and RFP labelled mitochondria. To evaluate Ca2+ influx upon ZP2 stimulation, [Ca2+]i changes were measured using the fluorescent indicator Fluo-4 AM. Protein–protein interactions between ZP2 and the sperm-specific Ca2+ channel (CatSper) were analyzed by immunoprecipitation.

Across all three experimental models, ZP2 induces the acrosome reaction in capacitated murine spermatozoa. This effect correlates with a significant increase of [Ca2+]i. Using CatSper1-deficient sperm confirmes that CatSper is involved in the Ca2+ response of spermatozoa to ZP2. Biochemical assays revealed that ZP2 directly binds to CatSper, suggesting a dual role as both ligand and channel activator.

Our findings demonstrate that ZP2 serves as a potent physiological trigger of the acrosome reaction in capacitated murine sperm via CatSper-mediated Ca2+ influx. The identification of a direct interaction between ZP2 and CatSper provides novel mechanistic insight into sperm activation and highlights a crucial step in mammalian fertilization.

Poster 139:

Title:
The impact of mitochondrial defects on female fertility in PARL deficient mice

Authors:
Laura Andric (Bonn, Germany)
Sarah Schumacher (Bonn, Germany)
Florian Renziehausen (Bonn, Germany)
Lars Fester (Bonn, Germany)
laura.andric@uni-bonn.de

Abstract:
The mitochondrial protease PARL regulates key cellular functions. PARL deficiency ($Parl^{-/-}$) in mice causes neurodegeneration, muscle and gonadal atrophy, and infertility in both sexes. Based on preliminary observations of uterine atrophy and anovulation in $Parl^{-/-}$ mice, we hypothesize that PARL deficiency disrupts ovulation and uterine maturation. This may result from impaired mitochondrial respiration, dysregulated sex hormone synthesis, and reduced MFN1-dependent mitochondria–ER contact sites. Our aim is to elucidate how mitochondrial dysfunction caused by PARL loss affects female reproductive capacity through altered endocrine signaling and organ development.

Ovaries, uteri, and duodena from 8-week-old $Parl^{-/-}$ and WT mice (n=4 per group) are collected and processed for H&E staining, immunofluorescence, Western blot, and transmission electron microscopy. Serial ovarian sections are 3D-reconstructed to analyze follicular structures and corpora lutea. Steroid hormone synthesis and MFN1 localization are assessed to investigate mitochondrial-endocrine interactions and folliculogenic arrest.

$Parl^{-/-}$ ovaries show reduced size and absence of corpora lutea despite preserved folliculogenesis, with increased atretic follicles. Uteri display smaller diameter and a thinner myometrial layer. Immunofluorescence confirms CL absence and elevated ERα receptor density. Western blot supports increased ERα, while ERβ remains unchanged, suggesting altered estrogen signaling contributes to reproductive dysfunction in $Parl^{-/-}$ mice.

Increased ERα expression in $Parl^{-/-}$ ovaries may reflect compensation for impaired steroidogenesis. Absent corpora lutea and atretic follicles indicate ovulatory failure. Future studies will assess MFN1, mitochondria-ER contacts, and steroidogenic enzymes across developmental stages (P20 and 4 weeks) to distinguish primary versus secondary defects, elucidating mitochondrial contributions to ovarian development, hormone production, and female fertility.

Poster 140:

Title:
Using PCL fibres to guide neural and endothelial growth in biofabricated tissue constructs

Authors:
Kornelia Kenst (Würzburg, Germany)
Peter Sperling (Würzburg, Germany)
Saskia Roth (Bayreuth, Germany)
Leonid Ionov (Bayreuth, Germany)
Süleyman Ergün (Würzburg, Germany)
Philipp Wörsdörfer (Würzburg, Germany)
kornelia.kenst@uni-wuerzburg.de

Abstract:
In addition to their use in disease modeling and drug testing, organoids can serve as modular building blocks for tissue engineering. To assemble multiple organoids into larger, millimeter-scale tissue constructs, scaffold materials are needed that not only support but also direct the growth of tissue components such as nerves and blood vessels. Here we investigate the use of touch-spun polycaprolactone (PCL) fibre meshes with defined fibre orientations for this purpose.

Human iPSC-derived neural and vascularized mesenchymal organoids were generated and cultured on matrigel-coated, touch-spun PCL fibres. PCL fibres were aligned either in a parallel or grid-like manner using an angle of 45 or 90°. Vascular and neural sprouts were assesed using immunofluorescence analyses.

Human iPSC-derived neural organoids that contain both central nervous and neural crest cells were placed on parallel-aligned PCL fibres. Peripheral nerve fibers began sprouting within a few days, partially accompanied by Schwann cell precursors. These neural sprouts followed the fibre orientation and extended up to 8 mm within two weeks.

Moreover, vascularized mesenchymal organoids, containing an intrinsic endothelial network, were also cultured on PCL fibres. The orientation of endothelial sprouts aligned with the fibre orientation, both in parallel and grid-like constructs.

Different types of organoids adhere well to PCL fibre meshes and can be used to assemble larger tissue constructs. The orientation of the fibres directs both neural and endothelial sprouting, offering a promising strategy for guided tissue engineering.

Poster 141:

Title:
Real-Time Monitoring of Pancreatic Tumor Dynamics in the Chick Chorioallantoic Membrane Model

Authors:
Andreas Ettner-Sitter (Regensburg, Deutschland)
Benedikt Wagner (Regensburg, Deutschland)
Agata Montagner (Regensburg, Deutschland)
Thia Aung (Deggendorf, Germany)
Christina Hackl (Regensburg, Deutschland)
Silke Haerteis (Regensburg, Deutschland)
andreas.ettner-sitter@ur.de

Abstract:
Pancreatic ductal adenocarcinoma (PDAC) is among the most lethal cancers, with a 5-year survival rate of just 6%. Its progression is closely tied to aberrant angiogenesis and vascular compression. The chorioallantoic membrane (CAM) model offers a versatile in vivo platform to engraft human, as well as primary PDAC tissue. The CAM is a highly vascularized membrane of the chick embryo. In this study, we use advanced imaging and histology to quantitatively assess PDAC progression and angiogenesis on the CAM.

The CAM is applied as a drug testing platform to study tumor biology and the effect of potential therapeutic agents on angiogenesis, tumor metabolism, proliferation, and metastasis. Human tumor tissue from PDAC patients was grafted onto the CAM and therapeutics were intravenously injected. Ultra-high frequency ultrasound at 71 MHz as well as Indocyanine green angiography were conducted. Immunohistopathological evaluation was performed by HE, CD31, cytokeratin7, and cytokeratin19 staining, and molecular analysis was performed.

The 3D ultrasound technique allowed for reproducible quantification of tumor volume and surface area across multiple scans. Our results demonstrated reductions in tumor size observed over time (n = 10), indicating a dynamic response of the tumor to the microenvironment. Through 3D ultrasound we were able to quantify tumor volume and surface area across multiple scans. These results were also confirmed through immunohistological staining.

The growth of PDAC tumor tissue can be evaluated in the CAM model, which is a promising model for the future. This will help shed light on future therapies individually, including personalized therapy.

Poster 142:

Title:
Exploring wound healing dynamics of human skin using an innovative CAM-based in vivo wound healing model

Authors:
Sandra Maurer (Regensburg, Germany)
Valeria Mei (Regensburg, Deutschland)
Charlotte Wagner (Regensburg, Deutschland)
Thiha Aung (Regensburg, Deutschland)
Silke Härteis (Regensburg, Deutschland)
Vera Rötzer (Regensburg, Deutschland)
sandra.maurer@vkl.uni-regensburg.de

Abstract:
Chronic wounds are a growing clinical and economic challenge, with limited treatment options and high healthcare costs. Yet, our understanding of wound healing in human skin remains limited. A key barrier is the lack of suitable models, since findings from animal and in vitro models often fail to translate.

To address this issue, we established a 3D-in-vivo wound healing model based on the chorionic allantois membrane (CAM), allowing the cultivation of human skin and the observation of wound healing processes. This 3R-compliant model helps to reduce conventional animal models and thus animal suffering.

By analyzing cell-cell-contacts (desmosomes, gap- and adherens junctions), as well as cytokeratins using digital PCR, Western blotting, immunohistochemistry and transmission electron microscopy, we analyse the dynamics of wound healing. Additionally, we assess the skin's regenerative character by quantifying Ki67-stained proliferating cells with AI-assisted image analysis.

The CAM-model is a suitable model to analyze human wound healing. HE- and Ki67-staining, confirmed skin tissue vitality for up to seven days. and the AI-tool has proven itself a useful tool for large-scale quantification. Additionally, the dynamics of cell-cell-contacts and the cytoskeleton were observed, giving insights to their expression changes during cutaneous wound healing in humans, which we found slightly distinct from findings in mouse models.

By exploring human wound healing mechanisms, especially those associated with cell-cell contacts, this approach and our novel wound healing model will contribute to the development of therapeutic strategies for chronic wounds, especially by enabling the translation from bench to bedside.

Poster 143:

Title:
AI-driven image analysis to complement translational research of dermal wound healing in the CAM-based 3D in-vivo model

Authors:
Valeria Mei (Regensburg, Germany)
Sandra Maurer (Regensburg, Germany)
Philipp Kainz (Altenrhein, Switzerland)
Andreas Ettner-Sitter (Regensburg, Germany)
Thiha Aung (Deggendorf, Germany)
Silke Härteis (Regensburg, Germany)
Vera Rötzer (Regensburg, Germany)
valeria.mei@stud.uni-regensburg.de

Abstract:
Artificial intelligence (AI) is increasingly relevant in our society and evolves rapidly. However, its application in translational research remains limited. In this project, we aim to apply AI-driven image analysis to gain deeper insights into human wound healing, which, besides growing clinical relevance, remains poorly understood.

We cultivate wounded human skin samples in our 3R-compliant 3D in-vivo wound healing model based on the chorionic allantois membrane (CAM). In this model we test potentially promoting wound healing compounds, like Hydroxytyrosol, directly on human skin. To assess the tissue's regenerative capacity, we perform immunohistological staining for the proliferation marker Ki-67 and trained a deep-learning model on the IKOSA platform to determine proliferating cells, complemented by a Python script. In parallel, we quantify angiogenesis on the CAM utilizing an AI-driven application on IKOSA.

We found AI-based algorithms to be a versatile tool for effective large-scale quantification of proliferation and angiogenesis. It also proved useful to confirm vitality of cultivated human skin for at least seven days, validating our experimental setup, and to evaluate possible treatments: Initial experiments with Hydroxytyrosol suggest that it may support wound closure and influence angiogenesis on the CAM, while its effect on cell proliferation appears limited.

Our AI-assisted evaluation approach reduces confounders like subjective bias and enables consistent, reproducible analysis. It offers many advantages compared to manual analysis, is easy to implement and complements our translational wound healing research. Altogether, this method contributes to testing new therapeutics and may ultimately improve clinical outcomes for patients with wound healing disorders.

Poster 144:

Title:
In vivo 3D tumor model to study human cholangiocarcinoma tissue

Authors:
Agata Montagner (Regensburg, Germany)
Andreas Ettner-Sitter (Regensburg, Germany)
Benedikt Wagner (Regensburg, Germany)
Thiha Aung (Deggendorf, Germany)
Christina Hackl (Regensburg, Germany)
Silke Härteis (Regensburg, Germany)
montagneragata@gmail.com

Abstract:
Cholangiocellular carcinoma (CCA) is characterized by a dismal 5-year-survival rate of 20%. The chorioallantoic membrane (CAM) is an extraembryonic membrane of the chick embryo and it constitutes a multifaceted model. The CAM model has been utilized to study many different malignancies and it is slowly attracting interest for investigating CCA. However, it has not previously been applied to human CCA tissues. Our objective is to establish the CAM model for the cultivation of CCA tissue.

We cultivated human CCA tissue (samples from 13 patients) on the CAM model. Its growth was observed through daily photos utilizing a microscope. Moreover, vascularization of the tumor tissue was studied through two different methods: ultra-high frequency ultrasound and laser speckle contrast imaging (LSCI) measurements. The histological structure and proliferation of the engrafted tumor was compared to the untreated primary tumor through HE and Ki67 staining.

We were successful in growing human CCA in the CAM model. Further, LSCI and ultra-high frequency ultrasound measurements confirmed the vascularization of CCA engrafted on the CAM. The typical abnormal bile duct and dense tumor stroma were preserved even after cultivation in the CAM model and proliferation of the tumor tissue on the CAM was confirmed through Ki67 staining.

The CAM model demonstrates significant potential as a preclinical platform for investigating CCA biology and drug efficacy as it is possible to culture tumor tissue. Its suitability for personalized medicine applications, enabling patient-specific therapeutic screening, represents a promising future direction.

Poster 145:

Title:
WNT7A/7B assemble a GPR124-RECK-LRP5/6 co-receptor complex to activate WNT/β-catenin signaling in brain endothelial cells

Authors:
Robin Heiden (Würzburg, Germany)
Süleyman Ergün (Würzburg, Deutschland)
Laura Hannig (Hamburg, Germany)
Calvin Kuo (Stanford, CA, USA)
Barbara Braunger (Hamburg, Germany)
Mario Vallon (Würzburg, Germany)
robin.heiden@uni-wuerzburg.de

Abstract:
Developmental CNS angiogenesis and integrity of the blood-brain barrier critically rely on WNT7A/7B signaling in endothelial cells, requiring the classical WNT receptors FZD and LRP5/6 as well as the WNT7-specific co-receptor complex GPR124-RECK. While the formation of a ternary GPR124-RECK-WNT7 complex is well-established, the suggested WNT7-induced FZD-LRP5/6-GPR124-RECK multi-receptor complex lacks biochemical evidence. This study was designed to elucidate WNT7-induced co-receptor complexes and signaling mechanisms.

bEnd.3 brain endothelial cells were stably transfected with a WNT/β-catenin signaling reporter. CRISPR/Cas9 was used to knock out the WNT7 receptors GPR124, RECK, FZD or LRP5/6. The cells were stimulated with WNT7A, WNT7B, or classical WNT3A and reporter activity was measured. RECK-containing protein complexes were isolated from cells with or without GPR124 knockout and with or without WNT7A stimulation using a combined chemical crosslinking and streptavidin pulldown approach.

Knockout of GPR124, RECK, or LRP5/6 fully blocked WNT7 signaling. In contrast, knockout of all FZD isoforms preserved partial WNT7 signaling but completely abrogated classical WNT3A signaling. Mass spectrometry analysis of RECK-associated proteins revealed WNT7-induced formation of a GPR124-RECK-LRP5/6 co-receptor complex lacking FZD. In GPR124 knockout cells, WNT7 stimulation still induced RECK-LRP5/6 heterodimerization, yet this complex lacked signaling activity and LRP5/6 phosphorylation. Dimeric GPR124 ectodomain- or antibody-mediated higher-order clustering of RECK-WNT7-LRP5/6 complexes restored WNT/β-catenin signaling

Our data demonstrate that WNT7-induced β-catenin signaling in CNS endothelial cells is partially driven by a FZD-independent GPR124-RECK-LRP5/6 co-receptor complex with GPR124 potentially playing a role in LRP5/6 activation rather than recruitment.

Poster 146:

Title:
Fasciola hepatica eludes biliary tuft cells in rat

Authors:
Adriano Sanna (Gießen, Germany)
Janine Koepke (Gießen, Germany)
Simone Häberlein (Gießen, Germany)
Uwe Pfeil (Gießen, Germany)
Svenja Gramberg (Gießen, Germany)
Christos Samaklovis (Gießen, Germany)
Jasmin Ballout (Gießen, Germany)
Martin Speckmann (Gießen, Germany)
Gabriela Michel (Gießen, Germany)
Martin Diener (Gießen, Germany)
Wolfgang Kummer (Gießen, Germany)
adriano.sanna95@gmail.com

Abstract:
Besides canonical cholangiocytes, extrahepatic bile ducts harbour rare cell types with ill-defined function, resembling ionocytes and tuft cells found in other mucosal epithelia of endodermal origin. Based on data from tuft cells in the small intestine, we hypothesized their involvement in the host response to parasitic worms and addressed this hypothesis in the common bile duct (CBD) of the rat, a common animal model for fascioliasis, a parasitic worm disease

Epithelial cell populations were characterized in controls and CBD infected with Fasciola hepatica by unbiased sc-mRNAseq analysis, transmission electron microscopy, and by immunohistochemistry using antibodies against tuft cell markers

Electron microscopy and sc-mRNAseq experiments revealed at least 4 differentepithelial clusters: goblet-like cells (Galnt12, Cldn2, Pah), ion transporting cells (Cftr,Car2, Atp1a1), secreting/endocrine cells (Ppy, Chgb, Pip) and tuft cells (Trpm5,Pou2f3, Avil).In chronic fascioliasis, the CBD showed massive enlargement and epithelial remodelling with elongated, straight peribiliary glands, reduced immunoreactive tuft cell density (ALOX5AP, DCLK1) and suppression of cytokine-cytokine receptor interaction, calcium-signalling, linoleic acid metabolism andarachidonic acid signalling pathways in tuft cells

This study identifies canonical tuft cells in the rat CBD that are distinct from microvillus-bearing and ion transporter expressing cells described before as “brushcells”. Related cells in the small intestine proliferate in response to protists and the helminth Nippostrongylus brasiliensis and are essential for clearing this infection. Although expressing a receptor for the helminth metabolite propionate, rat CBD tuft cells, however, are negatively regulated by F. hepatica, which might reflect amechanism of evasion from the hosts worm defence.

Poster 147:

Title:
MTSS1 regulates planar cell polarity and function of ependymal cells

Authors:
Britta Eiberger (Bonn, Germany)
Barbara Kalthoff (Bonn, Gerrmany)
Clemens Wagner (Bonn, Germany)
Andrea Christ (Bonn, Germany)
Gregor Kirfel (Bonn, Germany)
Karl Schilling (Bonn, Germany)
Tim Clarner (Bonn, Germany)
britta.eiberger@uni-bonn.de

Abstract:
Planar cell polarity (PCP), the coordinated orientation of cells within the tissue plane, plays a critical role in neural development, including neurogenesis and the patterning of polarized brain structures such as the ependyma. A key function of PCP proteins is the regulation of cytoskeletal organization and actomyosin dynamics to ensure proper tissue architecture. Metastasis Suppressor 1 (MTSS1) is a cytoplasmic adaptor protein that acts at the interface between the plasma membrane and the actin cytoskeleton. It stabilizes cell–cell junctions, shapes cell morphology, and promotes the formation of membrane protrusions like filopodia and lamellipodia. MTSS1 has also been observed at basal bodies of primary cilia, suggesting a potential role in cilia function.

Based on these known roles, we hypothesized that MTSS1 regulates motile cilia in the mouse brain by influencing PCP through cytoskeletal and membrane remodeling. To test this, we studied the development and function of wild-type and Mtss1-deficient ependymal cells using primary cultures, advanced optical imaging, and fluorescent particle tracking in ventricular explants.

We found that MTSS1 deficiency reduces cilia density and alters cell size. Analysis of basal body patch positioning—an anatomical marker of ependymal PCP—revealed that MTSS1 is required for proper translational, but not rotational, polarity. Functionally, loss of MTSS1 in ciliated ependymal cells disrupts the directional flow of cerebrospinal fluid (CSF) in the lateral ventricles.

Overall, our findings suggest that MTSS1 coordinates ependymal PCP by regulating basal body positioning, thereby ensuring proper cilia-driven cerebrospinal fluid flow in the brain ventricles.

Poster 148:

Title:
In-situ analysis of drug and metabolite distribution in a 3D in vivo cyst model of autosomal dominant polycystic kidney disease using desorption electrospray ionization-mass spectrometry (DESI-MS)

Authors:
Jan Schueler (Regensburg, Germany)
Petra K. Pallmann (Regensburg, Germany)
Antonia Hehemann (Regensburg, Germany)
Simon Heckscher (Regensburg, Germany)
Verena Gross (Straubing, Germany)
Matthias May (Straubing, Germany)
Barbara Nuebel (Erlangen, Germany)
Bernd Wullich (Erlangen, Germany)
Bjoern Buchholz (Regensburg, Germany)
Jens M. Werner (Regensburg, Germany)
Jonathan Jantsch (Cologne, Germany)
Wolfram Gronwald (Regensburg, Germany)
Zoltan Takats (Regensburg, Germany)
Peter J. Oefner (Regensburg, Germany)
Katharina M. Schmidt (Regensburg, Germany)
Katja Dettmer (Regensburg, Germany)
Silke Haerteis (Regensburg, Germany)
Petra.Pallmann@stud.uni-regensburg.de

Abstract:
Autosomal dominant polycystic kidney disease (ADPKD) is the most common monogenic kidney disorder. Recently, we demonstrated that human ADPKD kidney tissue can be cultured and analyzed for up to seven days on the chorioallantoic membrane (CAM), a three-dimensional in-vivo model that holds promise as platform for preclinical studies. Here, we demonstrate its usefulness in studying absorption and distribution of topically applied active substances by means of mass spectrometry imaging (MSI) using benzbromarone as example.

Renal cyst tissue obtained from nephrectomies was cultured for one week on the CAM, a well vascularized membrane. Perfusion was visualized by ultra-high frequency ultrasound (UHF). We topically applied benzbromarone, a potential therapeutic agent for inhibiting cyst growth, daily. One hour after the final treatment, the tissue was removed from the CAM, snap-frozen, and cryosectioned. The cryosections were first analyzed by DESI-MS, and afterwards histologically examined.

Renal cyst tissue was clearly identifiable in MSI images and could be distinguished from the surrounding CAM tissue due to its distinct lipid and metabolite content. Additionally, benzbromarone was successfully detected within the renal cyst tissue. The MSI images revealed not only how benzbromarone penetrated the renal cyst tissue, but also its accumulation around the cyst epithelium.

Using mass spectrometry imaging, we demonstrated convincingly the potential of the CAM model as platform for drug testing in human tissue. MSI provided detailed insights into the behavior of the applied drug within the tissue, confirming not only tissue penetration by the active substance but also allowing characterization of distribution, accumulation and metabolism.

Poster 149:

Title:
Impairment of mucociliary clearance after experimental stroke

Authors:
Claudia Dames (Berlin, Germany)
Alexander Pernis (Boston, USA)
Aysegül Demiral (Giessen, Germany)
Uwe Pfeil (Giessen, Germany)
Wafaa Mahmoud (Irbid, Jordan)
Renate Paddenberg (Giessen, Germany)
Daniel Berchtold (Berlin, Germany)
Tamara Papadakis (Giessen, Germany)
Ioannis Alexopoulos (Giessen, Germany)
Katarzyna Winek (Jena, Germany)
Maryam Keshavarz (Augsburg, Germany)
Gabriela Krasteva-Christ (Homburg/Saar, Germany)
Andreas Meisel (Berlin, Germany)
Wolfgang Kummer (Giessen, Germany)
Christian Meisel (Berlin, Germany)
Ayseguel.Demiral@anatomie.med.uni-giessen.de

Abstract:
Bacterial pneumonia is a major complication in patients with acute stroke. We addressed in an experimental stroke model whether mucociliary clearance is affected after cerebral ischemia.

Mice (C57BL/6J; Adrb2-/-) were subjected to middle cerebral artery occlusion (MCAO), sham-operated and naïve animals served as controls. Activity of tracheal sympathetic innervation was silenced by transection of the cervical sympathetic trunk (decentralization) 5 d before MCAO surgery. Ciliary activity was assessed by measuring particle transport speed (PTS) at explanted tracheas, cellular composition of the tracheal epithelium by confocal imaging of whole mounts, expression of adrenergic receptors by RT-qPCR, ultrastructural alterations by electron microscopy. Catecholaminergic nerve fibers were visualized by immunohistochemistry.

Baseline PTS was reduced 1 and 3 d after MCAO and returned to control values at d14. The frequency of ciliated cells was reduced by 48% 3 d after MCAO and was back to control at d14. Ciliated cells showed abnormal morphology, shortening and loss of cilia 1 d after MCAO. RT-qPCR revealed Adrb2 as the dominantly expressed adrenergic receptor in the tracheal epithelium, and tracheas of Adrb2-/- mice showed no significant decline in PTS after MCAO. There was a dense subepithelial network of catecholaminergic nerve fibers, and its functional silencing by decentralization prevented impairment of PTS at d1 after MCAO.

Stroke induces rapid changes in cellular composition and function of the respiratory epithelium resulting in impaired cilia-driven clearance function. This effect is mediated by sympathetic innervation and beta2-adrenergic signaling.

Poster 150:

Title:
Peroxisomes in Anucleate Cells: Evidence from Human and Murine Platelets

Authors:
Heike Bömmel (Würzburg, Germany)
Anggi Muhatar Pratama (Würzburg, Germany)
Jürgen Kößler (Würzburg, Germany)
Süleyman Ergün (Würzburg, Germany)
Markus Bender (Würzburg, Germany)
Srikanth Karnati (Würzburg, Germany)
srikanth.karnati@uni-wuerzburg.de

Abstract:
Peroxisomes are essential metabolic organelles involved in lipid metabolism and detoxification of reactive oxygen species (ROS). The presence and function of peroxisomes in anucleate cells such as platelets remain largely unexplored. Therefore, This study aimed to (1) confirm the presence of peroxisomes in human and murine platelets, and (2) elucidate their functional significance using a platelet-specific PEX5 knockout (PS-PEX5KO) mouse model.

Peroxisomal proteins were detected in MEG-01 megakaryocytic cells using immunofluorescence, qRT-PCR, and Western blotting. Donor-derived human platelets, stored concentrates, and murine platelets were analyzed for comparison. To assess function, PS-PEX5KO mice were generated via Cre-LoxP. Blood was analyzed for hematological parameters and platelet morphology. Glycoprotein receptor expression and activation were evaluated by flow cytometry. Catalase activity was quantified enzymatically. Platelet spreading was assessed by phase-contrast microscopy.

Peroxisomes were clearly identified in both human and murine platelets. PS-PEX5KO mice were viable but exhibited significantly larger platelets compared to wild-type (WT) controls. Erythrocyte counts remained unchanged, while lymphocytes were elevated and granulocytes/eosinophils were reduced in PS-PEX5KO mice. KO platelets showed slightly increased glycoprotein receptor expression with no change in activation. Catalase activity was significantly higher in KO platelets. At 30 minutes, KO platelets exhibited a significant increase in spreading, indicating accelerated spreading kinetics.

This study confirms the presence of functional peroxisomes in platelets and reveals that loss of PEX5 in platelets alters their morphology, ROS, and spreading behavior without affecting activation. These findings uncover a previously unrecognized role for peroxisomes in platelet biology and suggest implications for immune and hematological regulation.

Poster 151:

Title:
Fibronectin is essential for lymphvasculogenesis in the kidney

Authors:
Kristina Hermann (Regensburg, Germany)
Silke Seibold (Regensburg, Germany)
Kathrin Skoczynski (Regensburg, Germany)
Björn Buchholz (Regensburg, Germany)
Ernst R. Tamm (Regensburg, Germany)
Leonie Herrnberger-Eimer (Regensburg, Germany)
Kristina.Hermann@vkl.uni-regensburg.de

Abstract:
As a prominent component of the fibrillar extracellular matrix (ECM), fibronectin (FN) is abundantly expressed in the renal interstitium. Using mice with inducible and systemic FN deletion, this study examines its functional relevance during postnatal kidney maturation.

Fnfl/fl mice carrying a tamoxifen-inducible CAGG-Cre-ERTM transgene were treated with tamoxifen eye drops (2.5mg/mL) on postnatal day 0-2. FN deletion was confirmed by RT-PCR, Western blotting, and immunofluorescence. Kidney morphology, ECM composition, and lymphatic development were analyzed through histological analysis, in situ hybridization, and marker-based staining.

By postnatal day 3-4, FN levels were reduced by approximately 90% in kidney and liver, resulting in near-complete loss of fibrillar FN in the renal interstitium. FN-deficient mice developed rapidly expanding spherical fluid-filled structures at the corticomedullary junction, a region normally enriched in FN and its mRNA. Although morphologically resembling cysts, these structures lacked tubular epithelial cells and contained loose matrix and hyaluronan. While blood capillaries remained intact, LYVE-1+ lymphatic endothelial precursors were significantly reduced, suggesting impaired lymphatic vessel development. In contrast, FN deletion after postnatal day 10 had no effect on lymphatic architecture, indicating a critical early postnatal window for FN-dependent lymphvasculogenesis.

FN is essential for providing interstitial scaffolding that supports the migration and organization of lymphatic endothelial precursors during early postnatal kidney development. Its absence disrupts lymphvasculogenesis, resulting in insufficient fluid drainage, progressive interstitial edema, and ultimately cyst-like tissue degeneration. The findings reveal a previously unrecognized role for FN in the formation of the lymphatic network and the maintenance of fluid homeostasis in the kidney.

Poster 152:

Title:
Intrinsic physiology and synaptic connectivity of genetically specified VIP-expressing GABAergic neurons in L2/3 of mouse barrel cortex.

Authors:
Hala Nam (Göttingen, Germany)
hala.nam@med.uni-goettingen.de

Abstract:
Neocortical VIP interneurons (~12% of GABAergic INs) form disinhibitory circuits with SST and PV cells in the barrel cortex. Recent studies suggest VIP-INs comprise CR-, CCK-, and ChAT-expressing subtypes, but their detailed connectivity remains unclear.

In our study, we now focused on VIP+/ChAT+, VIP+/CR+, VIP+/CCK+ cells while performing intralaminar L2/3 paired patch-clamp recordings and optogenetics to detect their postsynaptic target cells, along with post-hoc morphological characterization. Additionally, we analysed the electrophysiological properties of each VIP subtype.

We show that VIP-ChAT INs preferentially target SST cells, whereas VIP-CR INs target both PV and SST cells, and VIP-CCK INs preferentially target excitatory cells. Each of the three VIP subtypes exhibits a wide-ranging electrophysiological and morphological profile, which shows over the subgroups no statistically significant differences, except for the distribution of action potential firing patterns. Overall, this makes the entire VIP cell population diverse in molecular identity, electrophysiological properties, morphology, and connectivity.

Our findings draw attention to the significant importance of this varied population of VIP INs in maintaining E/I balance and their contribution in somatosensory information processing. Notably, in light of evidence showing that their dysfunction is linked to various neurological and psychiatric disorders, affecting cognitive functions like schizophrenia and epilepsy, highlighting its crucial role in maintaining normal brain dynamics.

Poster 153:

Title:
Does gender influence learning, perceptions and retention in regional anatomy dissection courses?

Authors:
Veronica Antipova (Graz, Österreich)
Martin Siwetz (Graz, Österreich)
Franz A. Fellner (Linz, Österreich)
Simone Manhal (Graz, Österreich)
Julian F. Niedermair (Linz, Österreich)
Benjamin Ondruschka (Hamburg, Germany)
Amélie J. Poilliot (Basel, Switzerland)
Monika Wimmer-Röll (Linz, Österreich)
Andreas Wree (Rostock, Germany)
Niels Hammer (Graz, Österreich)
veronica.antipova@medunigraz.at

Abstract:
Medical educators should understand the differences within the medical student population, including gender. Research on gender effects in learning and skill acquisition has yielded contradictory results, particularly in anatomy learning among undergraduate medical students.

This study examined gender differences in several aspects of anatomy education. First, the ability of undergraduate medical students to identify anatomical structures was assessed to determine gender differences in learning gains. Second, learning outcomes were compared between a 3-month and a 1-month regional anatomy course, with attention to gender. Third, knowledge retention was evaluated at 6- and 12-month follow-ups. Finally, student perceptions of different embalming methods for anatomy learning were assessed with a focus on gender.

Minor differences were observed between genders across different regions in learning gains. Scores from the 3-month course were higher than those from the 1-month course, regardless of gender, particularly for the neck, thorax, and abdomen. A significant knowledge gain was noted in abdominal anatomy at the 6- and 12-month follow-up: scores improved by 56% in females and by 68% in males. The comparison of Thiel- versus ethanol-based embalming revealed gender differences regarding tissue pliability and the utility of tissues for achieving learning objectives.

In conclusion, subtle differences appear to exist between female and male students in terms of perceptions and learning outcomes in gross anatomy dissection courses. These findings will be useful in developing new curricula for undergraduate medical students, taking into account gender differences in the context of regional anatomy dissection courses.

Poster 154:

Title:
Social Stress Response: tailored by Microglia–OPC Crosstalk?

Authors:
Giulia Poggi (Mainz, Germany)
Senta Gioia Reuss (Mainz, Germany)
Christopher Pryce (Zürich, Switzerland)
Marianne Müller (Mainz, Germany)
Michael Schmeisser (Mainz, Germany)
poggigiu@uni-mainz.de

Abstract:
Social experiences shape behaviour and brain circuits, with emerging evidence pointing to a critical role of the oligodendrocyte lineage (OL) and myelin in these processes – especially, with respect to aversive social experiences. Social stress is a key trigger for disorders of increased aversion responsiveness, such as depression and anxiety, and is linked to functional changes in cortico-amygdala circuits. These alterations might implicate changes in the OL and myelination. However, key features of these (mal)adaptive responses remain inconsistently reported across studies. Interestingly, this is paralleled by heterogenous inflammatory response. Under physiological conditions, microglia – the immune cells of the CNS – continuously monitor the CNS microenvironment and play a key role in maintaining OL and myelin homeostasis. We therefore hypothesised that microglia sense, decode, and respond to the social stress signals, thereby tailoring the OL adaptive response.

In male mice, we employed histological and semi-automated image analyses to examine the impact of social stress on: (1) the density and proliferation of microglia and oligodendrocyte precursor cells (OPCs); (2) their reciprocal localization; and (3) their morphological complexity (as an indication of their status).

We demonstrated that social stress alters myelin content and OL proliferation-maturation dynamics. Notably, the density of proliferative OPCs correlated with the density of microglia. The latter showed changes in morphology and in spatial proximity to OPCs, suggesting potential regulatory interactions.

These findings provide preliminary but integrative evidence that microglia-OPC crosstalk may mediate the OL response to social stress and may contribute to the heterogeneity of stress-related psychiatric disorders.

Poster 155:

Title:
Structural Brain Changes, Glial Cell Loss and Mitochondrial Dysregulation in a Mouse Model of Anorexia Nervosa

Authors:
Linda Frintrop (Rostock, Germany)
Annelie Zimmermann (Rostock, Germany)
Stephan Lang (Rostock, Germany)
Matthias Grothe (Greifswald, Germany)
Antje Vogelgesang (Greifswald, Germany)
Marcus Frank (Rostock, Germany)
Markus Kipp (Rostock, Germany)
linda.frintrop@med.uni-rostock.de

Abstract:
Anorexia nervosa (AN) is a psychiatric disorder associated with extreme weight loss, hyperactivity, and brain atrophy. Weight rehabilitation reversed these symptoms, although the underlying pathophysiological mechanisms are mostly unknown. Studies have indicated disrupted white matter integrity, particularly in the corpus callosum (CC), and changed mitochondrial metabolism. We hypothesize that starvation induces structural brain alterations through combined effects on neurons, glial cells and, subcellularly, on mitochondria.

A starvation-induced hyperactivity mouse model was used, in which female mice maintained a 25% body weight loss over two weeks, followed by a three-week refeeding phase. Brain sections and serum neurofilament light chain (NfL) levels were used to investigate brain volumes and neurodegeneration. Behavioral changes were evaluated using the elevated plus maze. Immunohistochemistry and transmission electron microscopy (TEM) were employed to evaluate glial alterations and mitochondrial morphology. Complex morphological analyses of microglia were performed using Neurolucida 360.

Chronic starvation induced AN-like symptoms, including hyperactivity and reduced brain volumes in the cortex and CC. These changes were accompanied by elevated serum NfL levels and reduced anxiety-like behavior. Starvation led to decreased oligodendrocyte density in the CC, which normalized after refeeding. TEM revealed decreased axonal calibers, increased mitochondrial density and decreased mitochondrial areas in the white matter. Additionally, microglial density in the cortex decreased, while soma size and process length increased.

Starvation leads to neurodegeneration, glial cell loss, and mitochondrial dysregulation, contributing to structural brain and behavioral alterations in AN. These interconnected alterations underscore the role of cellular and structural brain changes in the pathophysiology of AN.

Poster 156:

Title:
Histopathological aspects of the nucleus morphology in astrocytic tumors. Problems in differential diagnostic and the role of immunochemistry.

Authors:
Anca Sava (Iași, Romania)
Ana Maria Dumitrescu (Iasi, Romania)
Dragos Andrei Chiran (Iasi, Romania)
Stefanie Griebeler Oliveira (Pelotas, Brasil)
Ruxandra Teodora Stan (Iași, Romania)
Lucian Eva (Iasi, Romania)
dragos_chr@yahoo.com

Abstract:
Our study reveals the multiple aspects of the nucleus in the glioblastomas and other tumors with astrocyte origin, ranging from the small cells with monomorphic oval nuclei and minimal cytoplasm to the giant cells (large mononucleated and multinucleated cells with abundant cytoplasm).

Our study investigated 120 tumors from the patients that underwent surgery in the "Prof. Dr. N. Oblu" Hospital Iaşi, Romania. Tumors were fixed and embedded in paraffin, then stained with Hematoxylin-Eosin and several histochemical techniques (Van Gieson trichrome staining, silver impregnation staining, PAS staining). Immunohistochemical stainings were also performed with the following antibodies: anti-GFAP, anti-Synaptophysin, anti-Olig2, and the Ki-67 binding index was determined.

Glioblastomas with small cells are highly cellular, but rather monotonous. Tumor cells are poorly differentiated with brisk mitotic activity (3-5 mitosis/high power field–HPF). There is also prominent microvascular proliferation (2-3 mitosis/HPF in the endothelial tumor cells). Giant cell glioblastomas presented a low tumor cell density with numerous multinucleated giant cells. The mitotic activity is lower (2-3 mitosis/HPF) compared with small cell glioblastomas. Also, the mitotic activity in the endothelial tumor cells is lower (1 mitosis/ HPF). Glioblastomas and the astrocytic tumors derive from astrocytes and are the most malignant tumors of central nervous system. Glioblastomas have a great number of tumoral glial cell nuclei and a great nuclear pleomorphism.

The nuclear morphology and the mitotic activity explain the different prognosis between these different histological variants of astrocytic tumors and the two types of glioblastomas.

Poster 157:

Title:
Changes in reward-encoding meta-ensembles induced by alcohol dependency

Authors:
Janine Reinert (Göttingen, Germany)
Tianyang Zheng (Mannheim, Germany)
Botond Molnar (Cluj-Napoca, Romania)
Wolfgang Sommer (Mannheim, Germany)
Christoph Koerber (Göttingen, Germany)
janine.reinert@med.uni-goettingen.de

Abstract:
Cues associated with natural rewards as well as with drugs can trigger reward seeking behavior when presented. Reward-associated behavior is thought to be mediated by neuronal ensembles, sparsely distributed populations of neurons within a certain brain region, that are activated by a certain stimulus or context. Ensembles activated by drugs or natural rewards are are largely overlapping and of similar sizes in a given brain area, and only the co-activation of them into a meta-ensemble encodes the reward-specificity. Here, we asked how these meta-ensembles change in rats with a history of alcohol dependency.

Rats were trained to associate distinct cues with the natural reward saccharin (sweet) and the drug alcohol. One half of the cohort, subsequently underwent chronic intermittent alcohol exposure, to induce alcohol dependence. After a period of abstinence, neuronal ensembles active during seeking behavior for either reward were determined by reinstatement testing and analysis of the expression of the immediate early gene c-Fos. c-Fos expression then served as the basis for graph theory-based analysis of meta-ensembles.

Graph theory-based network analysis revealed that saccharin and alcohol recruited distinct meta-ensembles across brain regions. The network recruited by saccharin was highly modular whereas the alcohol-network showed no modularity but an increase in functional connectivity. Moreover, in the two meta-ensembles different brain regions were in control of information flow, depending on the identity of the reward.

We could show specific differences in the representation of natural and drug rewards within the brain’s reward system and thereby define a neuronal correlate underlying reward-specificity.

Poster 158:

Title:
Molecular Characterization of GALC Mutations Causing Krabbe Disease

Authors:
Piet Ankermann (Erlangen, Germany)
Eileen Socher (Erlangen, Germany)
piet.ankermann@fau.de

Abstract:
Krabbe disease (also called globoid cell leukodystrophy) is a rare lysosomal storage disorder affecting the white matter in the central and peripheral nervous systems. It causes progressive neurodegeneration, most commonly in its infantile form. As such, it is usually diagnosed within the first year of life and associated with high morbidity and mortality. The disease follows an autosomal recessive inheritance pattern and results from mutations in the GALC gene, which encodes the lysosomal hydrolase galactocerebrosidase (GALC). Our study investigates the structural effects of several missense mutations in GALC identified in patients with Krabbe disease.

To examine the consequences of these pathogenic mutations on the three-dimensional protein structure of the enzyme GALC, structural models were generated. These models served as the foundation for a series of molecular dynamics simulations assessing the structural stability of wild-type and mutant GALC.

Comparative analyses between wild-type GALC and variants with missense mutations revealed differences in protein flexibility, the intramolecular interactions between different amino acids within GALC and also the stability of secondary structure elements near the active site of the enzyme. Moreover, one mutations also altered the size and shape of the substrate-binding pocket, despite not being located directly within the pocket.

Overall, our MD simulations shed light on how GALC mutations may impact the protein structure of galactocerebrosidase in the lysosomal environment. These structural insights help to explain how such mutations could impair enzyme function, contributing to the pathology of Krabbe disease.

Poster 159:

Title (of your talk/poster, NOT your academic title) MECHANISMS OF CYST FORMATION IN A XENOPUS MODEL OF AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE

Contact Email taiyo.yamamoto@anatomy.uzh.ch

First Name Taiyo
Last Name Yamamoto

Department/Division Institute of Anatomy

Organization/University University of Zurich

Country (Format: e.g. "Austria", "Germany", "Switzerland") Switzerland

Email taiyo.yamamoto@anatomy.uzh.ch

Presenter Yes

Objective Autosomal Dominant Polycystic Kidney Disease (ADPKD) is a highly prevalent genetic disorder, primarily caused by mutations in PKD1 or PKD2. The disease is marked by the progressive formation of renal cysts, ultimately leading to end-stage renal disease. Effective treatment options remain lacking, largely due to incomplete understanding of the roles of PKD1/PKD2 gene products and the molecular mechanisms driving cyst formation.

Methods This study utilizes the Xenopus model to observe and manipulate renal tubules in vivo during their transformation from healthy to cystic. We compared pkd1 knockout kidneys to wild-type controls, particularly focusing on the period before and after the onset of luminal flow. To investigate the mechanisms involved, we employed physical and genetic interventions, small-scale compound screens, and a phosphoproteomic screen to identify molecular regulators of tubular behavior.

Results Before the onset of luminal flow, pkd1 knockout kidneys are indistinguishable from their wild-type counterparts. Surprisingly, after flow begins, both wild-type and mutant tubules exhibit transient dilations. In wild-type kidneys, these dilations are followed by a rapid constriction that restores the tubule to its original diameter. In contrast, pkd1 knockout kidneys fail to initiate this constrictive response, leading to unchecked dilation. We identified actomyosin as a key driver of the constriction process and revealed molecular regulators that modulate both the dilation and constriction phases.

Conclusion Our findings support a novel hypothesis that insufficient tubular constriction is an early and critical contributor to cyst initiation in ADPKD. By uncovering molecular components involved in this process, this work opens new avenues for therapeutic exploration.

Poster 160:

Exploring the Parathyroid–Thymus Connection

Marius Valeriu Hînganu1, Maria-Paula Comanescu2, Delia Hînganu1

1 Department of Morpho-Functional Sciences I, Grigore T.Popa University of Medicine and

Pharmacy, Iasi, Faculty of Medicine, University Street No. 16, 700115, Iași, Romania,

hinganu.delia@umfiasi.ro, marius.hinganu@umfiasi.ro

2 Department of Surgery I, Grigore T.Popa University of Medicine and Pharmacy, Iasi, Faculty

of Medicine, University Street No. 16, 700115, Iași, Romania, mariapaula.

comanescu@umfiasi.ro

The parathyroid and thymus glands play crucial roles in the endocrine and immune systems, respectively, and share notable developmental and functional connections. Both originate from the third pharyngeal pouch during embryogenesis, suggesting a potential for physiological interaction and shared pathological mechanisms. This study is based on the hypothesis that their common origin underlies a deeper interdependence between these glands. We examined the developmental trajectories, anatomical proximity, and possible biochemical communication between the parathyroid and thymus. Emerging evidence indicates that parathyroid hormone (PTH) may influence thymic function, particularly T-cell maturation and immune modulation. Conversely, thymic-derived signaling molecules might affect calcium regulation and parathyroid activity.

Understanding the interplay between these glands may shed light on new mechanisms in endocrine-immunological disorders, including hypoparathyroidism, thymomas, myasthenia gravis, and thymic hypoplasia. This opens the door to novel therapeutic strategies. Given the limited existing data, further large-scale studies are critical to elucidate the molecular basis of this glandular crosstalk. This includes investigating receptor expression for key secreted molecules from each gland and analyzing the cervico-pericardial ligaments, which may house essential vascular and neural connections. These pathways could represent the primary route of communication between the parathyroid and thymus.

Keywords: thymic function, parathyroid glands, parathyroid-thymus interconnection, morfofunctional endocrine research

Poster 161:

Ultrastructural and Vascular Insights into Hip Joint Degeneration

Delai Hinganu1, Riana Maria Huzum2, Bogdan Huzum2, Marius Valeriu Hinganu1

31Department of Morpho-Functional Sciences I, Faculty of Medicine, “Grigore T. Popa”

University of Medicine and Pharmacy, 700115 Iasi, Romania,
marius.hinganu@umfiasi.ro,

hinganu.delia@umfiasi.ro

2Department of Surgery, Faculty of Medicine, “Grigore T. Popa” University of Medicine and

Pharmacy, 400347 Iasi, Romania,

Degenerative processes of the hip joint extend beyond articular cartilage, increasingly involving periarticular structures such as the joint capsule, acetabular labrum, and subchondral bone. This study investigates the structural, molecular, and vascular changes associated with advanced hip osteoarthritis, emphasizing the potential role of epiphyseal vascularization in disease progression.
Tissue samples from patients undergoing total hip arthroplasty were compared with controls presenting morphologically normal joints. A comprehensive methodology combining immunohistochemistry (IHC), scanning electron microscopy (SEM), micro-computed tomography (microCT), and hematoxylin-eosin staining was employed. Key molecular markers for proliferation (Ki67), inflammation (CD68), angiogenesis (CD31, ERG), chondrogenesis (SOX9), and lubrication (Lubricin) were analyzed. Degenerative samples showed increased expression of Ki67, CD68, CD31, ERG, and SOX9, with a marked reduction in Lubricin. SEM
revealed disorganized collagen networks, ultrastructural damage, and neovascular remodeling.
Additionally, persistent anastomoses between epiphyseal vessels were observed, even after bone resorption, suggesting that vascular impairment may precede and drive osteoarthritic changes.
These findings highlight the importance of endothelial signaling—particularly through the Notch
pathway—in skeletal aging and joint degeneration. The study supports an integrated view of vascular, molecular, and structural degeneration and introduces new perspectives on early detection, targeted therapies, and morphofunctional rehabilitation strategies for the hip joint.

Keywords: hip capsule; labrum; osteoarthritis; immunohistochemistry; SEM; SOX9; CD68;
ERG; lubricin; joint degeneration

Poster 162:

The boundaries of the sciatic gliding space

Hanno Steinke [a*], Vaclav Klicnik [b], Andreas Wolfgang Reske [b], Wolfgang Gruener [b], Dina Wiersbicki [c] and Karolina Böllinger [a]

[a] Institut für Anatomie, Universität Leipzig, Liebigstr. 13, 04103 Leipzig, F. R. of Germany

[b] Klinik für Anästhesiologie, Intensivmedizin, Notfallmedizin und Schmerztherapie Heinrich-Braun-Klinikum, Karl-Keil-Straße 35, 08060 Zwickau, F. R. of Germany

[c] Department of Orthopaedic, Trauma and Plastic Surgery, Liebigstr. 20, 04103 Leipzig, F. R. of Germany

[*] Corresponding author

Email addresses:
Karolina.Boellinger@medizin.uni-leipzig.de (Böllinger)
AndreasWolfgang.Reske@HBK-Zwickau.de (Reske)
Vaclavklicnik@gmail.com (Klicnik)
Wolfgang.Gruener@HBK-Zwickau.de (Grüner)
Dina.Wiersbicki@medizin.uni-leipzig.de (Wiersbicki)
Steinke@medizin.uni-leipzig.de (Steinke)

For anatomical teaching purposes, the sciatic nerve is dissected like a rope, as shown in the textbook. In order to do this, the tissue connecting the nerve to the surrounding area must first be removed. This tissue is referred to rather unspecifically as 'connective tissue'. Assuming that this tissue provides a gliding space to prevent pain during leg movement, we dissected the area and injected resin into it. We examined it using sonography in dead and living subjects. We found that the nerve is enclosed in a C-shaped sliding space with the convexity facing laterally. The dorsal space lies beneath the gluteal connective tissue. The ventral branch is constricted by three arches that are clearly related to the small rotators. The piriformis's limits the space cranially. Caudally, the biceps femoris divides the space to the knee. Our findings have been verified in several dozen legs to date. Clinically, using an arthroscopic approach, fibrous bands appear in this space when gluteal pain or sciatica occurs, and these can be released through therapy. We have rarely observed such fibres; our examinations appear to have been carried out on a healthy population.

Poster 163:

Exploring Age-Related Changes in the Mouse Brain: A Single-Cell Analysis of Male and Female Microglia

Natascha Vidovic[1], Julia Hassa[2], Anika Winkler[2], Yvonne Kutter[3], Tobias Busche[2], Björn Spittau[1]

[1] Bielefeld University, Medical School OWL, Anatomy and Cell Biology, Bielefeld
[2] Bielefeld University, Medical School OWL & Center for Biotechnology (CeBiTec), OMICS Core Facility Next-Generation Sequencing, Bielefeld
[3] Bielefeld University, Center for Biotechnology (CeBiTec), Technology Platforms Genomics, Bielefeld

Introduction

Novel technologies such as single-cell RNA and single-nucleus RNA sequencing have shed new light on the complexity of different cell populations in physiological and pathological states. This project aims to investigate the molecular and cellular alterations of microglia occurring in the brains of aging male and female mice at the single-cell and spatial level.

Methods

Utilizing advanced single-cell sequencing technologies Chromium and Visium from 10x Genomics, we profiled the transcriptomes of microglia from young (6 months-old) and aged (24 months-old) wildtype mice of both sexes for specific gene signatures. Sequencing was done with Illumina and data was analyzed using 10x Loupe Browser.

Results

In total we copmared and analyzed 4 different goups (N=3). First results show differences in the expression profile of female and male microglia-specific genes as well as between the two age groups. Phagocytic and activation markers are upregulated in the aged group compared to younger samples. Genes associated with the neurodegenerative phenotype of microglia are upregulated in the aged group as well. Moreover, male and female cells differ in their expression levels of classical activation markers and genes involved in TGFβ signaling.

Discussion

By comparing gene expression patterns and functional pathways between age groups and sexes, we identified different molecular signatures associated with aging. Insights gained from this project may pave the way for future comprehensive studies elucidating the mechanisms driving age-related brain dysfunction and may contribute to the development of novel therapeutic strategies for age-related cognitive impairment and neurodegenerative diseases.

Key words

Microglia/Aging/Singe-cell transcriptomics/Sex differences/TGFβ signaling

Poster 163:

3D Biofabrication of Vascular Structures using Human iPSC-derived Mesodermal Progenitor Cells

Leyla E. Dogan[1], Nathaly Chicaiza-C.[2], Florian Kleefeldt[1], Philipp Wörsdörfer[1], Jürgen Groll[2], Süleyman Ergün[1]
[1]Institute of Anatomy and Cell Biology, Würzburg, Germany
[2]Department for Functional Materials in Medicine and Dentistry, Würzburg, Germany

Objective:

This work aims to biofabricate hierarchically organized multilayered large blood vessels from human induced pluripotent stem cells-derived mesodermal progenitor cells (hiMPCs).

Methods:

An in-gel bioprinting system was used to extrude bioink into tubular geometry (2 cm length, lumen diameter up to 4 mm). As biomaterial FGX, a blend of cold-water fish skin GelMA (F), porcine skin GelMA (G) and xanthan gum (X) was combined with collagen type I (C) to formulate biomaterial (FGXC). After extrusion of hiMPCs within FGXC, cellular viability and morphogenesis were evaluated using life-dead staining, H&E staining, immunofluorescence analyses and tissue clearing.

Results:

Printed hiMPCs differentiated into vascular (endothelial (ECs) and smooth muscle cells (SMA) as well as non-vascular cell types (macrophage-like cells) and self-organized into structures resembling early embryonic vessels with intima, media, and adventitia like layers. The presence of immune and progenitor cell populations indicates that the printed tubular constructs not only support vascular morphogenesis and plasticity but also enable cell-driven processes essential for long-term remodeling of the vascular wall.

Conclusion:

hiMPCs have the potential to differentiate into vascular & non-vascular cell types (stem cells, immune cells) in an organized manner to form hierarchically organized large blood vessel structures after in-gel bioprinting process. It shows that hiMPCs are suitable cell sources for the biofabrication of hierarchically organized multilayered vascular structures.