WELCOME

Dear Colleagues,

We are pleased to welcome you at the Black Sea Neurogenesis 2025 international conference. This year's event is the fourth Neurogenesis meeting taking place at the Black Sea in the last 8 years. We hope that thanks to the high-profile Speakers, the conference shall be able to sustain as a regular event on the neuroscience calendar in the future.

As in the previous meetings, the 2025 event includes topics that cover the process of neurogenesis during the development of the brain, and in adulthood, including under conditions of injury. The complex mechanisms that lead to the proper assembly of the cells that build the brain during development intersect with the mechanisms that govern the production of new brain cells in distinct cellular niches of the adult brain. We hope that the attendance of Black Sea Neurogenesis by both developmental neurobiologists and clinicians will not only advance the translational impact of the fundamental discoveries on neural stem cells and their progeny, but will also built a bridge between the understanding of developmental and adult neurogenesis.

As in previous meetings, we hope that the event will promote building new partnerships between the participating researchers.

Yours sincerely,

Anton B. Tonchev, Organizer
Wieland B. Huttner, Programme Advisor

Varna, Bulgaria, June 2025

Demetrius
2022

BLACK SEA NEUROGENESIS 2025

Varna, Bulgaria, 05 - 07 June 2025

PROGRAMME

WEDNESDAY, JUNE 4 – ARRIVAL

20:00 **Speakers' Welcome Dinner**

DAY 1: THURSDAY, JUNE 5

08:30 Registration, poster placement

09:00 Welcome address and opening

SESSION I Developmental neurogenesis (09:30 - 12:00)
Chair: Anton Tonchev

09:30 - 10:15 **Extended Lecture: Magdalena Götz** (Munich, Germany)
Novel mechanisms of neurogenesis

10:15 - 10:45 COFFEE BREAK

10:45 - 11:15 **Boyan Bonev** (Munich, Germany)
3D epigenome evolution underlies divergent gene regulatory programs in primate neural development

11:15 - 11:45 **Michael Heide** (Göttingen, Germany)
Human-specific genes and their ancestors in brain development and evolution

11:45 - 12:00 Short talk - **Kamela Nikolla** (Lyon, France)

12:00 - 16:00 LUNCH AND FREE TIME

SESSION II Developmental neurogenesis (16:00 - 19:45)
Chair: Magdalena Götz

16:00 - 16:45 **Extended lecture: Zoltán Molnár** (Oxford, UK)
Shadows of the subplate – the link between early cortical development to Autism Spectrum Disorders

16:45 - 17:15 **Wieland Huttner** (Dresden, Germany)
Role of neural stem cell metabolism in neocortex expansion in development and human evolution

17:15 - 17:30 Short talk - **Yiling Li** (Munich, Germany)

17:30 - 18:30 COFFEE BREAK + POSTERS

18:30 - 18:45 Short talk - **Linus Kordt** (Bochum, Germany)

18:45 - 19:00 Short talk - **Ana Villalba Requena** (Vienna, Austria)

19:00 - 19:45 **Extended lecture: Victor Borrell** (Alicante, Spain)
Evolutionary convergence and divergence of cortical neurogenesis

20:00 Opening reception

DAY 2: FRIDAY, JUNE 6

SESSION III Developmental neurogenesis (09:00 - 12:15) *Chair: Victor Borrell*

09:00 - 09:45 **Extended Lecture: John Rubenstein** (San Francisco, CA, USA)
Dlx1&2 directly promote expression of Cxcl14 to control synapse development and interneuron survival

09:45 - 10:15 **Chiaki Ohtaka-Maruyama** (Tokyo, Japan)
Mechanisms of subplate expansion in primates

10:15 - 10:30 Short talk - **Vincenzo De Paola** (Singapore)

10:30 - 11:00 **COFFEE BREAK + POSTERS**

11:00 - 11:30 **Kazunobu Sawamoto** (Nagoya, Japan)
From birth to repair: Transformation of neural stem cells and neuronal migration for brain regeneration

11:30 - 12:15 **Extended Lecture: Hiroshi Kawasaki** (Kanazawa, Japan)
How folds of the brain form and what they mean

Conference photograph

12:15 - 16:30 **LUNCH AND FREE TIME**

SESSION IV Adult and developmental neurogenesis (16:30 - 19:30) *Chair: John Rubenstein*

16:30 - 17:00 **Nicolas Toni** (Lausanne, Switzerland)
The emergence of individuality: from the embryo to the adult

17:00 - 17:30 **Thorsten Döppner** (Emden, Germany)
Translational limitations/perspectives of adult neurogenesis and transplantation of cell products in clinical stroke settings

17:30 - 17:45 Short talk - **Lidiia Tynianskaia** (Göttingen, Germany)

17:45 - 18:30 **COFFEE BREAK + POSTERS**

18:30 - 18:45 Short talk - **Tokiharu Takahashi** (Tokyo, Japan)

18:45 - 19:30 **Extended lecture: Ryoichiro Kageyama** (Kobe, Japan)
Activated neurogenesis improves amyloid-β pathology and cognition in Alzheimer's disease model mice

20:00 **DINNER** – **"RAI"** BEACHSIDE RESTAURANT

SCAN QR code for location

DAY 3: SATURDAY, JUNE 7

SESSION V Adult neurogenesis (09:00 - 12:00)
Chair: Zaal Kokaia

09:00 - 09:45 **Extended Lecture: Arturo Alvarez-Buylla** (San Francisco, CA, USA)
A relay from apical to basal neuronal stem cells is key for adult neurogenesis

09:45 - 10:15 **Benedikt Berninger** (London, UK)
Engineering inhibitory neurogenesis in vivo via lineage reprogramming

10:15 - 10:30 Short talk - **Martin Ivanov** (Varna, Bulgaria)

10:30 - 11:00 **COFFEE BREAK + POSTERS**

11:00 - 11:30 **Bogdan Draganski** (Bern, Switzerland)
In vivo brain histology using non-invasive Magnetic Resonance Imaging (MRI)

11:30 - 12:00 **Janine Gronewold** (Essen, Germany)
Improvement of neurogenesis and slowing of neurodegeneration through healthy lifestyle

12:00 - 16:30 **LUNCH AND FREE TIME**

SESSION VI Adult neurogenesis (16:30 - 19:45)
Chair: Arturo Alvarez-Buylla

16:30 - 17:15 **Extended Lecture: Hideyuki Okano** (Tokyo, Japan)
Applications of human induced pluripotent stem cells (hiPSCs) in treatment of Amyotrophic lateral sclerosis (ALS)

17:15 - 17:45 **Anton B. Tonchev** (Varna, Bulgaria)
Transcriptomic signatures of adult macaque monkey stem cell niche domains

17:45 - 18:00 Short talk - **Anna Chantzara** (Patras, Greece)

18:00 - 18:30 **COFFEE BREAK**

18:30 - 19:00 **Dirk Hermann** (Essen, Germany)
Opportunities and challenges of stem/ precursor cell-based therapeutics in the ischemic brain

19:00 - 19:45 **Extended Lecture: Zaal Kokaia** (Lund, Sweden)
Reprogrammed neurons and glia for brain repair after stroke

CONFERENCE CLOSING

FAREWELL DINNER

Medical University Varna "Prof. Dr. Paraskev Stoyanov"

TranStem

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2022

Magdalena Götz

Helmholtz Center, Munich, Germany

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Novel mechanisms of neurogenesis

I will focus on the new concept of organelle heterogeneity and how protein localization matters. I will discuss:

- Centrosome composition of human neural stem cells and neurons
- Most proteins are exchanged at the centrosome in this differentiation
- Changes occur at different places of the centrosome
- Protein localization at the centrosome matters for disease
- Example: the ubiquitous splicing factor PRPF6 and its role in periventricular heterotopia
- Many nuclear proteins are at the centrosome – what about centrosome/cytoskeleton proteins in the nucleus?
- Proteomic profiling highlights microtubule interacting proteins in nucleus
- Nuclear function of Map1b and its role in periventricular heterotopia

https://www.mcn.uni-muenchen.de/members/regular/goetz/index.html

Boyan Bonev

Helmholtz Center, Munich, Germany

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3D epigenome evolution underlies divergent gene regulatory programs in primate neural development

The expansion of the neocortex is a hallmark of human evolution and is closely linked to neural stem cell biology. Yet, the epigenetic mechanisms driving divergent gene regulation during primate neurogenesis remain elusive.

Here, we comprehensively mapped 3D genome organization, chromatin accessibility and gene expression in induced pluripotent stem cells and derived neural stem cells from human, chimpanzee, gorilla and macaque. We identified human-specific epigenetic signatures including cis-regulatory regions (CREs) and enhancer-promoter interactions, linking them to gene regulatory dynamics. While human-specific structural variants and HARs are associated with gene expression and epigenomic changes during evolution, they alone do not fully explain these differences. Instead, deep learning models revealed complex regulatory grammar at CREs, including transcription factor binding sites, local context and higher-order chromatin organization as key elements of species-specific regulatory evolution. High-resolution Hi-C uncovered unexpected global shifts in 3D genome architecture while topologically associating domains remain remarkably conserved. Notably, species-specific genes interacted with multiple differentially accessible regions, suggesting that synergistic enhancer activation is a key mechanism driving epigenome evolution.

These findings provide new insights into the epigenetic basis of primate brain evolution and lay the groundwork for the identification of novel human-specific molecular mechanisms and targets that can be further validated to deepen our understanding of the evolution of human traits.

www.bonevlab.com

Michael Heide

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Human-specific genes and their ancestors in brain development and evolution

In the field of neocortical neurogenesis in development and evolution, I will discuss the following topics:

- NOTCH2NLB and NBPF14 are two human-specific genes that are genomic neighbors and likely exert a combinatory effect.
- NBPF14 increases basal progenitor (BP) abundance by promoting delamination through elevated oblique cleavage plane orientation.
- NOTCH2NLB promotes apical progenitor (AP) proliferation, leading to an expansion of the AP pool.
- Co-expression of NBPF14 and NOTCH2NLB enhances BP production while maintaining self-renewal of APs.
- Double knockout of ARHGAP11A and ARHGAP11B results in elevated BP levels.
- ARHGAP11A orchestrates neuroepithelial organization by regulating cytoskeletal dynamics and extracellular matrix (ECM) composition.
- ARHGAP11A preserves the apical progenitor pool by controlling mitotic spindle orientation via RHOA–ROCK signaling

https://www.dpz.eu/en/brain-development-and-evolution

Zoltán Molnár

University of Oxford, Oxford, UK

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Shadows of the Subplate – the link between early cortical development to Autism Spectrum Disorders

In the field of neurodevelopment, I will report the following:

- The lowermost cell layer of the cerebral cortex that contains interstitial white matter cells in humans has great clinical relevance. These neurons are very abundant during development and express higher proportions of susceptibility genes linked to human cognitive disorders than any other cortical layer and their distribution is known to be altered in schizophrenia and autism.
- Despite these clinical links, our current knowledge on the adult layer 6b is limited. The adult population that remains in all mammals to form interstitial white matter cells in human or layer 6b in mouse display unique conserved gene expression and connectivity.
- Members of my laboratory recently identified intracortical and thalamic projections from a subpopulation of layer 6b cells that might regulate both cortical and thalamic arousal of cortical areas that are involved in higher cortical functions.
- Our data suggest that 6b is not just a developmental remnant cell population in the adult, but a layer that plays a key role in cortical state control, integrating and modulating information processing (Messore et al., 2024).

Messore F et al., (2024) Biorxiv preprint doi: https://doi.org/10.1101/2024.07.25.605138

https://www.dpag.ox.ac.uk/team/zoltan-molnar

Wieland B. Huttner

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Role of neural stem cell metabolism in neocortex expansion in development and human evolution

In the field of neocortical neurogenesis in development and evolution, I will focus on the role of neural stem cell metabolism and discuss the following:

- ARHGAP11B, a human-specific protein which expands the primate neocortex, is imported into mitochondria and promotes glutaminolysis in basal progenitors.
- There is functional synergy of ARHGAP11B and GLUD2 (glutamate dehydrogenase), an ape-specific metabolic regulator.
- Transketolase-like 1 (TKTL1), which increases cortical neurogenesis via increased abundance of basal radial glia, operates in the pentose phosphate pathway and promotes fatty acid synthesis.

https://www.mpi-cbg.de/research/researchgroups/alumni-emeriti/wieland-huttner/group-leader

Víctor Borrell

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Evolutionary convergence and divergence of cortical neurogenesis

In the field of neocortical neurogenesis in development and evolution, I will report the following:

- Cortical expansion during evolution involved increasing neurogenesis, mediated by Intermediate Progenitors (IPCs) and Indirect Neurogenesis.
- In mouse, Indirect neurogenesis predominates at early stages, and progressively switches to Direct neurogenesis which predominates at late stages, in contrast to the established dogma.
- Distinct subclasses of apical and basal Radial Glia Cells co-exist in ferret and human, but not mouse.
- Subclasses of RGCs and IPCs are specifically involved in distinct parallel cell trajectories and lineages, amplifying neuronal amount and diversity.
- Basal progenitors in ferret are much more diverse than in mouse, including IPCs, bRGCs and subapical RGCs.
- Basal progenitors are absent in reptiles but present in chick, suggesting an ancient evolutionary origin.
- Basal progenitors in chick display a diversity in morphology, dynamics and marker expression greater to mouse and similar to ferret.
- Transcriptomic analyses suggests that cortical progenitors and neurogenesis are subject to different molecular regulation in birds than other amniotes.
- Our results suggest that cortical progenitor cells were already diverse in ancestral amniotes, and this complexity was expanded in large mammals but lost in small rodents.

https://vborrelllab.wixsite.com/website

John Rubenstein

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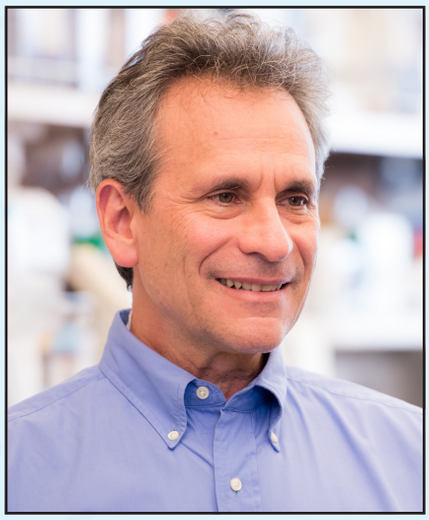

Dlx1&2 directly promote expression of Cxcl14 to control synapse development and interneuron survival

My lab has worked on the characterization and function of the Dlx genes for over thirty years. These homeobox transcription factors are the core regulators of the development of forebrain GABAergic neurons, sequentially controlling most steps in their formation, specification, migration, maturation, survival and function (reviewed in Rubenstein et al., doi: 10.1242/dev.202684). While we have a deep understanding of Dlx prenatal functions, so far, nothing is known about the molecular mechanisms how the Dlx genes control postnatal steps in cortical interneuron development, including synapse formation and survival.

Here we identify transcriptomic changes in Dlx-mutant interneurons and demonstrate that one of the genes, Cxcl14, is critical in cortical interneuron synaptogenesis and survival. Also noteworthy, our transcriptomic analysis of wild type neonatal cortical interneurons, provide clear evidence that, as early as postnatal day 3, we can identify evidence for at least 6 types of MGE- and CGE-derived cortical interneurons.

We have gone on to define cis-regulatory elements (candidate enhancers; pREs) that are bound by DLX2 at various stages during cortical interneurons development. We discovered three class of pREs: 1) constitutively bound, beginning in progenitors of telencephalic GABAergic neurons, and in maturing cortical interneurons; these pREs are implicated in regulating genes encoding core components of telencephalic GABAergic development; 2) pREs regulating genes that are expressed in prenatal and neonatal immature cortical interneurons; 3) pREs regulating genes in neocortical interneurons. We demonstrate that two of these pREs (from the *Cxcl14* and *Pnoc* loci) have activity in young adult murine CINs. Thus, we have identified genomic, epigenomic, transcriptomic, mouse genetic, and synaptic evidence of how the Dlx genes and proteins control postnatal cortical interneuron development.

https://rubensteinlab.ucsf.edu/

Chiaki Ohtaka-Maruyama

Tokyo Metropolitan Institute of Medical Science, Japan

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Mechanisms of subplate expansion in primates

In the field of neocortical neurogenesis in development and evolution, I will discuss the following topics:

• Subplate Layer in Cortical Development

The subplate (SP) layer facilitates thalamocortical connections and radial neuronal migration, playing a critical role in establishing cortical architecture during embryonic development.

• Primate Subplate Layer Expansion

In primates, the SP layer is significantly expanded during mid-gestation, but its precise biological roles and underlying mechanisms remain poorly understood.

• Transcriptomic Analysis of SP Layer

Visium and Xenium spatial transcriptomics revealed primate-specific genes in the SP layer, distinct from mice, highlighting its evolutionary expansion. SP neurons likely regulate thalamocortical projections via retinoic acid signaling.

https://www.igakuken.or.jp/regeneration/english.html

Kazunobu Sawamoto

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From birth to repair: Transformation of neural stem cells and neuronal migration for brain regeneration

- Birth orchestrates the transition of radial glia into long-term neural stem cells by modulating intrinsic programs and niche interactions.
- Following brain injury, newborn neurons derived from neurogenic niches extend a specialized growth cone to navigate complex environments and migrate toward lesion sites.
- Engineered artificial scaffolds mimic extracellular structures and promote directional migration and integration of newborn neurons into damaged brain regions.
- Modulation of cell adhesion enables chain migration of neurons, providing a therapeutic target to enhance regenerative outcomes after brain injury.

https://k-sawamoto.com/

Hiroshi Kawasaki

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How folds of the brain form and what they mean

In the field of the development and evolution of cortical folds, I will discuss the following topics:

- We introduced in utero electroporation techniques to ferrets.
- Gene expression not only in neurons but also in astrocytes and oligodendrocytes can be manipulated in ferrets.
- FGF signaling and sonic hedgehog signaling mediate cortical folding.
- Not only neurogenesis but also astrogenesis crucial is for cortical folding.
- Cortical folds are important for enhancing glymphatic circulation.
- Sulci serve as hot spots for cerebrospinal fluid infiltration into the brain parenchyma.

https://square.umin.ac.jp/top/kawasaki-lab/e-top.html

Nicolas Toni

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The emergence of individuality: from the embryo to the adult

In the field of adult neurogenesis, I will discuss the following:

- Adult mice show drastic natural variations in hippocampal neurogenesis despite identical genetic background and housing conditions.
- The position of the embryo in the uterine horn influences brain development and has a particularly strong effect on the development of the dentate gyrus of the hippocampus.
- These differences are maintained throughout development and contribute to the natural variations observed on adult neurogenesis and may underlie inter-individual differences in susceptibility to stress.

www.tonilab.org

Thorsten R. Döppner

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Translational limitations/ perspectives of adult neurogenesis and transplantation of cell products in clinical stroke settings

With the introduction of systemic thrombolysis and the advent of endovascular thrombectomy, recanalization of the formerly occluded vessel is the gold standard in modern stroke therapy. Such recanalization strategies, however, are limited to the very first hours after stroke onset, leaving a therapeutic gap during the subacute and chronic phase of the disease. In light of neurogenesis persisting throughout adulthood, preclinical stroke studies focused on fostering the therapeutic potential of neurogenesis. As such, stem/ precursor cells and their products have been used in a variety of translational studies as well as in some clinical trials to help stimulate endogenous neurogenesis and regeneration under stroke conditions.

In this presentation the following topics will be discussed:

- Relevance of neurogenesis in preclinical (rodent) stroke studies.
- Translational stroke studies focusing on stem/ progenitor cell transplantation and their cell products such as extracellular vesicles.
- Clinical trials using poststroke stem cell transplantation.
- Feasibility of neuroregenerative-based therapeutic strategies in clinical work flows and algorithms.

As such, the presentation will provide an overview of cell-based therapeutic strategies in stroke settings from a strong clinical point of view.

https://www.anevita.de/kliniken/klinik-emden/klinik-emden-medizin/klinik-emden-neurologie/

Ryoichiro Kageyama

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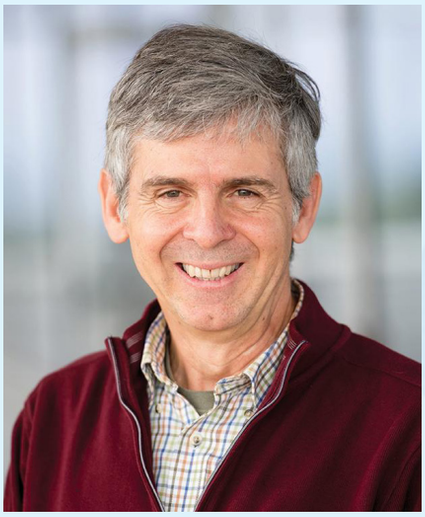
Activated neurogenesis improves amyloid-β pathology and cognition in Alzheimer's disease model mice

Adult hippocampal neurogenesis declines with aging and neurological disorders, leading to cognitive impairment. We previously demonstrated that inducing Plagl2, a zinc finger transcription factor gene, and inhibiting Dyrk1a, a gene associated with Down syndrome, can functionally rejuvenate aged neural stem cells (NSCs), thereby promoting neurogenesis and improving cognition in aged mice. Here, we found that this treatment effectively activated neurogenesis, reduced amyloid- deposition, and enhanced cognition in Alzheimer's disease model mice. Downstream of this treatment, many genes were globally upregulated or downregulated in the hippocampus. The upregulated genes include those involved in activation of astrocyte and microglia for amyloid- clearance, while the down-regulated genes include Prkag2 (encoding protein kinase AMP-activated non-catalytic subunit gamma 2) and Maml2 (encoding mastermind-like 2 co-activator), whose knockdown alone stimulated neurogenesis and reduced amyloid- deposition. These results suggest that activating neurogenesis by inducing Plagl2 and anti-Dyrk1a activity can mitigate age-related neurological disorders, including Alzheimer's disease, by regulating a broad network of downstream genes, which may serve as promising therapeutic targets.

https://www.riken.jp/en/research/labs/bdr/



Arturo Alvarez-Buylla

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A relay from apical to basal neuronal stem cells is key for adult neurogenesis

The continual birth of neurons in the postnatal vertebrate brain depends on the long-term persistence of primary precursors, referred to as neural stem cells (NSCs). In the Ventricular-Subventricular Zone (V-SVZ), the most extensive germinal niche in the adult mammalian brain, a population of astroglial cells (B1 cells) with apical contact with the lateral ventricle functions as the NSCs for generating neurons and glia. B1 cells, however, cannot explain the protracted neurogenesis destined for the olfactory bulb in adult mice.

I will discuss:

- B1 cells in mice are largely depleted during juvenile and early adult life, but neurogenesis continues at a relatively higher rate.
- The location of the primary cilia and basal body allows for the objective identification of a second population of basal V-SVZ astroglial (B2) cells without epithelial anchorage.
- As B1 cells decline during juvenile development, B2 cells increase in numbers.
- B2 cells possess transcriptomic properties of both quiescent and activated NSCs and can generate new neurons and glial cells well into adulthood.
- In the human brain, B1-like cells are depleted soon after birth, but a population of B2-like cells persists into adulthood.

The work suggests that non-apical NSCs serve as a relay to maintain neurogenesis in the adult and aged mouse brain. This process could help understand the extent to which gliogenesis and neurogenesis continue in the postnatal human brain.

https://ablab.ucsf.edu/

Benedikt Berninger

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Engineering inhibitory neurogenesis *in vivo* via lineage reprogramming

Lineage reprogramming of resident glia into induced neurons emerges as an experimental strategy to remodel diseased brain circuits. A key challenge to the approach consists in the generation of neurons with neuronal subtype-specific features.

- Our previous work has highlighted the possibility of converting postnatal cortical astrocytes into induced neurons with hallmarks of parvalbumin-expressing, fast-spiking interneurons. I will discuss our recent efforts using single cell transcriptomics to uncover the changes in gene expression that underpin the conversion of early cortical astrocytes into interneuron-like cells.
- Endogenous cortical interneurons die by apoptosis during a critical window of development. Is this regulation recapitulated by induced interneuron-like cells?
- Can induced interneuron-like cells integrate into functional cortical circuits? Here, I will present data from two-photon live imaging that show that induced interneuron-like cells can become recruited into active cortical circuits and respond to sensory stimuli.

https://www.kcl.ac.uk/people/benedikt-berninger

Bogdan Draganski

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In vivo brain histology using non-invasive Magnetic Resonance Imaging (MRI)

In the field of imaging neuroscience, I will discuss the following points:

- What is the concept of computational anatomy using MRI?
- Current state-of-the-art of in vivo brain histology applications
- Use cases in cross-sectional and longitudinal studies of the brain in health and disease
- Outlook: future developments in the field

https://neurologie.insel.ch/de/

Janine Gronewold

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Improvement of neurogenesis and slowing of neurodegeneration through healthy lifestyle

Neurogenesis persists throughout lifetime. In particular, in the dentate gyrus of the hippocampus, a high number of immature neurons at different stages of development are observed in adults without neurological diseases. Yet, neurogenesis appears to decline with increasing age as a part of normal ageing. In Alzheimer's disease (AD), however, neurogenesis is much more restricted with a significant reduction in the number and degree of maturity of new neurons. The pharmacological treatment options of AD as a highly prevalent neurodegenerative disease are still limited comprising mainly acetylcholinesterase inhibitors, memantine and recently approved monoclonal antibodies. As these pharmacological treatments have various side effects, they are not suitable for everyone. Since they might have only a moderate effect on quality of life, non-pharmacological options should be considered for the treatment and prevention of neurodegeneration. Recent studies have shown that a healthy lifestyle including exercise, social activities and specific nutritional aspects can promote neurogenesis in healthy ageing and slow neurodegeneration and cognitive decline in dementia. In this presentation, the following topics will be discussed:

- Neurogenesis in healthy aging
- Neurogenesis in AD and other dementias
- The effects of a healthy lifestyle on neurogenesis in healthy aging
- The effects of a healthy lifestyle on neurogenesis in dementia patients
- Inclusion of healthy lifestyle aspects in current guidelines about dementia prevention, intervention and care

https://neurosciencelab.uk-essen.de/members/

Hideyuki Okano

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Applications of human induced pluripotent stem cells (hiPSCs) in treatment of Amyotrophic lateral sclerosis (ALS)

In the field of iPSC-based disease modeling and drug discovery for neurodegenerative diseases such as ALS, I will present the following findings:

- Human iPSCs enable disease modeling and drug screening for ALS, a neurodegenerative disorder affecting motor neurons (MNs).
- ALS pathology involves “dying forward” excitotoxicity and “dying back” axonal degeneration.
- iPSC-derived MNs from familial (FUS/TDP-43) and sporadic ALS showed axonal degeneration, supporting the “dying back” hypothesis.
- A screen of FDA-approved drugs identified Ropinirole, a D2 receptor agonist, as a strong therapeutic candidate.
- In the Phase I/IIa ROPALS trial, Ropinirole was safe and slowed ALS progression and respiratory decline over one year.
- iPSCs from ROPALS participants suggested Ropinirole inhibits SREBP2-dependent cholesterol biosynthesis.
- Cholesterol metabolism may be a key pathway in ALS pathogenesis, as supported by polygenic risk analysis.
- These results highlight iPSC-based approaches for identifying new ALS treatments.

http://www.okano-lab.com/

Anton B. Tonchev

Medical University – Varna, Bulgaria

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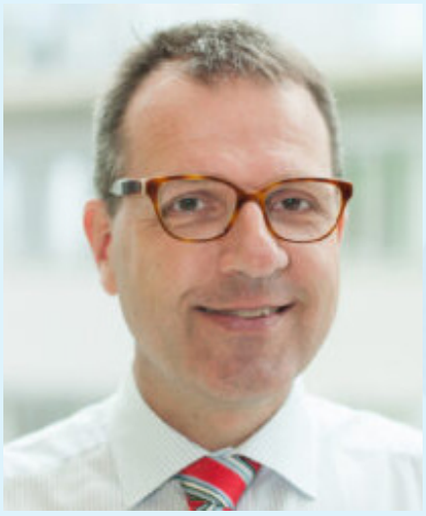
Transcriptomic signatures of adult macaque monkey stem cell niche domains

In the field of adult primate neurogenesis, I will report the following:

- The subventricular zone (SVZ) of the adult macaque monkey brain contains different domains: anterior (SVZa) and inferior (SVZi).
- While SVZa maintains neurogenesis, SVZi is not capable of generating new neurons.
- Brain ischemia triggers neural progenitor proliferation in both SVZa and SVZi.
- The transcriptomic response to brain ischemia in SVZa and SVZi shows both similar and distinct features.
- An open virtual histological atlas (www.monkey-niche.org) shows gene expression patterns in macaque SVZa, SVZi, and in the dentate gyrus.
- Novel gene markers for monkey SVZa or SVZi may help tracking precursor cells and their progeny in primates.

https://www.mu-varna.bg/EN/anton-tonchev



Dirk M. Hermann

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Opportunities and challenges of stem/ precursor cell-based therapeutics in the ischemic brain

Stem/ precursor cells of different cellular sources are capable of inducing striking therapeutic responses in neurological disease models. Extracellular vesicles (EVs) are extremely versatile naturally occurring membrane particles that convey complex signals between cells and recapitulate the effects of their parental stem/ precursor cells. When applied in vivo in ischemic stroke models, small EVs obtained from mesenchymal stromal cells (MSCs) exhibit remarkable effects on immune responses, vascular integrity, brain remodeling and plasticity. This multimodal response helps to set the stage for neurological recovery and long-term brain tissue survival, as we previously showed. In this presentation

- The current understanding of the biological effects of EVs will be presented, describing how EVs released from various subcellular sources identify their cellular targets and convey signals to recipient cells.
- Mechanisms of action in ischemic stroke contexts will be defined. A major target of systemically administered EVs are immune cells in the blood. Their contribution to the restorative effects of EVs will be outlined.
- Effects of vascular risk factors on the responses to systemically administered stem/ precursor cells and EVs will be evaluated. These factors compromise their therapeutic responses.
- Considerations about the planned clinical translation of EVs will be presented. With the data meanwhile obtained, stem/ precursor cell-derived EVs will shortly enter randomized clinical trials. We will have to circumvent pitfalls putting at risk this promising therapeutic strategy for failure.

The goal is to provide a steppingstone that can be used to critically discuss cell-based therapies as next generation therapies in brain diseases.

https://neurosciencelab.uk-essen.de/

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Reprogrammed neurons and glia for brain repair after stroke

In the area of neurodegeneration and neuroregeneration, I will present the following key findings:

- Cortical progenitor cells derived from human skin can differentiate into cortical projection neurons.
- These progenitors successfully integrate into stroke-affected cortical networks in rats, establishing incoming and outgoing synaptic connections.
- Transplantation of these cortical neurons into the stroke-lesioned cortex prevents the thalamus from secondary, delayed degeneration.
- They also demonstrate functional integration into adult human cortical organotypic slice cultures.
- When transplanted, the cortical neurons respond to sensory input in live animals, and their optogenetic inhibition leads to measurable changes in spontaneous behavior.
- We developed an efficient and rapid protocol for generating functional myelinating oligodendrocytes from human skin-derived long-term neuroepithelial-like cells.
- These oligodendrocytes exhibit structural, molecular, and functional features consistent with mature human oligodendrocytes.
- Following transplantation into stroke-injured rat somatosensory cortex and adult human cortical organotypic models, the derived oligodendrocytes are capable of myelinating both human graft-derived and host axons.
- Collectively, these results suggest that transplantation of reprogrammed cells may offer a viable strategy to repair damaged neural circuits in stroke patients, with potentially significant therapeutic implications.

https://www.stemcellcenter.lu.se/research-groups/kokaia

Short talk abstracts

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MECP2 role in cell-cycle kinetics and mode of division of cortical progenitors in the developing primate brain

MECP2 (methyl-CpG binding protein 2) is an X-linked gene associated with two main severe neurological disorders, Rett syndrome (RTT) and MECP2 duplication syndrome (MDS), both characterized by a postnatal onset of symptoms. While most functional studies of MECP2 have focused on later stages of cortical development, its role during early development of the cerebral cortex in humans and non-human primates remains largely unknown.

First, by using a combination of single-cell RNA-seq with immunohistochemistry we provide a detailed description of MECP2 expression patterns and timetable in the developing cortex of both non-human primates and humans. We report a rostral-caudal gradient of increasing MECP2 expression reminiscent of the rostral-caudal maturation gradient of cortical areas as well as an apical-basal gradient in the developing cortical wall.

Second, so as to investigate the role of MECP2 at early stages of neurogenesis, we implemented a targeted overexpression of MECP2 in cortical progenitors generating infragranular (E65) in the macaque cortex. We report significant alterations in the cell-cycle parameters of apical progenitors, affecting their proliferation kinetics, division mode, and cellular features such as interkinetic nuclear migration in the ventricular zone . Additionally, we observed consequences of MECP2 overexpression on the dynamics of radial migration of newborn neurons and their maturation.

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TGIF2 is a major regulator of neural stem cell fate and neurogenic priming

During brain development, neural stem cells (NSCs) must balance self-renewal with differentiation and ensure lineage progression. To identify novel regulators of NSCs during neurogenesis, we isolated NSCs by FACS from the mouse cerebral cortex and ganglionic eminence at mid-neurogenesis, and at birth, when gliogenesis starts in both, but neurogenesis only continues in the latter region. RNA-seq and ATAC-seq revealed major transcriptional and chromatin changes between these stages and identified TGFB-Induced Homeobox Factor 2 (TGIF2) as a key candidate factor in neurogenic NSCs. In vitro and in vivo experiments demonstrated a potent role of TGIF2 controlling NSC fate maintenance mediated by its interaction with the SIN3A/HDAC repressor complex suppressing neuronal differentiation genes. Multiomic comparison of NSC and neuron gene expression allowed the comprehensive analysis of neurogenic priming in cortical NSCs, identifying TGIF2 as its major regulator by restraining neuronal differentiation gene activation in NSCs.

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Modeling Reelin functions in human cerebral organoids

Reelin, a large extracellular glycoprotein encoded by the RELN gene, plays a fundamental role in cortical development and has been associated with a range of human neurodevelopmental and neurodegenerative disorders including Alzheimer's disease and epilepsy (Frotscher et al., 2003; Hamad et al., 2021, 2024; Weninger et al., 2021). To investigate human cerebral Reelin function, we are generating cerebral organoids from human induced pluripotent stem cells (iPSCs), combining this approach with CRISPR/Cas9-mediated gene editing to produce RELN knockout lines and introduce the gain-of-function RELN-COLBOS mutation, which has been associated with resilience to Alzheimer's disease (Lopera et al., 2023).

For this purpose, several single guide RNAs targeting sequences in five exons of RELN have been successfully cloned into CRISPR/Cas9 expression vectors. Transfection of human iPSCs yielded transfection efficiencies of 20–40%, and puromycin-based selection led to the survival and

expansion of individual clones. These clones have been undergoing PCR and sequencing analysis to confirm the presence of indel mutations and the targeted COLBOS variant. In parallel, we have initiated cerebral organoid differentiation according to (Lancaster et al., 2013) in several iPSC lines and can report their initial histological evaluation.

The focus of the project lies in the maturation and comparative analysis of wild-type, RELN-knockout, and RELN-COLBOS organoids. Planned experiments include histological and immunohistochemical characterization of cortical architecture, assessment of Reelin-dependent signaling (e.g., Dab1 phosphorylation), and evaluation of molecular signatures using single-cell RNA sequencing. In addition, organoids are planned to be exposed to elevated temperatures as well as oxygen and glucose-deprived conditions to model fever induced and ischemic stress, examining Reelin's role in neuroprotection and regeneration.

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Rptor/mTORC1 function in radial glia progenitor cell lineage progression

The cerebral cortex is composed of an extraordinary number of neuronal and glial cell types assembling into cortical circuits that account for cognitive abilities. Remarkable heterogeneity in the spectrum of cortical cell types has been described on the basis of global transcriptome analysis. How cell-type diversity is generated by radial glia progenitor (RGP) cells during development is however still poorly understood. Here we focus on the Rptor/mTORC1 signaling pathway and its role in RGP cell lineage progression and cortical neurogenesis. By using Mosaic Analysis with Double Markers (MADM) technology we ablated Rptor/mTORC1 either sparsely in single clones or in the entire tissue. Such paradigms enabled us to determine the relative contributions of cell-autonomous Rptor/mTORC1 function and their interaction with global tissue-wide mechanisms. We found that Rptor is not cell-autonomously required for cortical neurogenesis but rather for survival of defined populations of cortical excitatory neurons. Strikingly, RNA sequencing analysis unveiled differential requirement for Rptor function across distinct specific projection neuron classes, implying that mTORC1 signaling fulfills critical roles in the generation of cortical cell-type diversity.

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Single-cell multiomic atlas of human cortical development in Down Syndrome

Down syndrome (DS), caused by an extra copy of chromosome 21, is the most common genetic cause of intellectual disability, affecting approximately 1 in 700 live births. Despite decades of research, it remains unclear how the increased dosage of some or all of the ~200 protein-coding genes on chromosome 21 impacts brain development - particularly in the cerebral cortex, which is essential for higher-order cognitive functions.

To address this, we generated a single-cell atlas of the human fetal cortex, capturing both transcriptomic and chromatin accessibility landscapes during a critical window of cortical development, spanning the end of the first trimester through midgestation (11–20 weeks post-conception).

In parallel, I will present recent advances in modeling human brain circuit development and disease mechanisms using iPSCs derived from individuals with DS. These systems provide an ethically sustainable and virtually unlimited source of human neurons and glial cells. While animal models remain indispensable, faithfully capturing the complexity of DS requires a human genetic background. Nevertheless, conventional in vitro systems fall short in replicating key developmental processes such as interactions with the vasculature and immune system—both crucial for accurate disease modeling.

To overcome these limitations, we combine iPSC-derived neurons with a xenotransplantation platform and multiomic analyses to study how trisomy 21 drives DS-associated neurological features, including infant seizures, cognitive deficits, and early-onset Alzheimer's-like dementia. Our integrative approach—spanning transcriptomic, epigenomic, and proteomic data—provides new insights into DS pathophysiology and identifies candidate mechanisms and therapeutic targets for restoring neural circuit function.

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The role of transcription factor families in primate brain evolution: focus on humans

During primate evolution, the neocortex, a defining feature of mammals responsible for complex cognitive and motor functions, has undergone significant expansion in both size and folding. This process has been shaped by various families of transcription factors and their interplay. One key group is the Krüppel-associated box (KRAB) domain-containing zinc-finger proteins (KZFPs), which have rapidly expanded during primate evolution. Humans encode

approximately 350 KZFPs, many of which are primate-specific. These transcription factors regulate gene expression patterns critical for brain development and function, suggesting their involvement in the evolutionary expansion of the human brain. Here, we characterized the role of one such KZFP during brain development using ectopic expression and knockdown approaches in cerebral organoids and in in vitro differentiated neural progenitor cells.

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BMP signalling regulates embryonic Gfap-expressing neural stem cells in hippocampal development

In the hippocampus, neural stem cells (NSCs) persist into adulthood and generate neurons throughout life. Whilst adult hippocampal NSCs have been demonstrated to respond to BMP signalling, which controls their quiescence and proliferation balance, their developmental origins and regulation mechanisms remain elusive. Previously, we demonstrated that embryonic Gfap-expressing cells generate granule cells in the dentate gyrus.

Here, we establish that these Gfap-positive cells are not merely granule cell progenitors but may constitute bona fide NSCs that give rise to adult hippocampal NSCs, as supported by our BrdU lineage tracing experiments. In vitro neurosphere assays reveal that these embryonic Gfap-expressing cells display characteristics consistent with neural stem properties. Notably, our experiments reveal that around embryonic day 16.5, a population of actively proliferating embryonic NSCs already undergo cell cycle deceleration, ultimately developing into adult NSCs. Furthermore, we demonstrate that BMP signalling, in addition to its established role in adult NSC regulation, governs Gfap-expressing hippocampal NSCs throughout multiple critical developmental stages: embryonic hippocampal NSC specification and the transition from embryonic to adult NSCs. Using loss-of-function approaches with dominant negative constructs, we identify BMP pathway involvement in orchestrating sequential developmental steps necessary for proper hippocampal neurogenesis.

Remarkably, we successfully induced the conversion of a subset of embryonic cortical NSCs into hippocampal NSC-like cells through BMP signalling manipulation. This conversion was evidenced by the expression of dentate-specific markers, including the transcription factor Prox1. This work reveals the fundamental role of BMP in orchestrating hippocampal NSC development from embryonic origins to adult stages and offers insights into potential approaches for directing neural stem cell fate determination. We discovered that embryonic Gfap-positive cells are true NSCs that give rise to adult hippocampal NSCs and demonstrated that BMP signalling orchestrates hippocampal NSC development from embryonic specification through adulthood, enabling us to induce hippocampal NSC-like properties in a subset of cortical NSCs.

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Evidence of postnatal neurogenesis in the Septum Pellucidum of the adult human brain

The septum pellucidum is a thin midline structure situated between the lateral ventricles, traditionally considered to lack neurogenic potential. It encloses a cavity known as the cavum septi pellucidi. In this study, we provide morphological and immunohistochemical evidence supporting the presence of postnatal neurogenesis within this region. Anatomically, the septum pellucidum was delineated using Sudan Black B (SBB) staining, which revealed two distinct compartments: a dorsally located, wider part, and a ventrally located, narrower channel-like part. Nissl staining with cresyl violet confirmed the presence of cells within both compartments. Notably, immunostaining for neurofilament H did not reveal axonal or dendritic fibers, indicating the absence of mature neuronal processes within the structure.

To assess neuronal maturity, we employed NeuN immunostaining combined with lipofuscin autofluorescence. NeuN-positive neurons lacking lipofuscin granules were identified, suggesting an immature neuronal phenotype. Quantification across three subjects revealed that between 30.4% and 63.1% of neurons were devoid of lipofuscin. These findings were further corroborated by double immunostaining for doublecortin (DCX) and NeuN, revealing both DCX+/NeuN− and DCX+/NeuN+ cells, indicative of distinct stages of neuroblast maturation.

Given the presence of immature neurons within the structure, we next explored whether neural stem cells (NSCs) might also be present. To this end, we performed immunostaining for established NSC markers. Cells expressing these markers were observed lining and outlining the cavum septi pellucidi, further supporting the hypothesis of ongoing postnatal neurogenesis in this region.

Our data reveal the septum pellucidum as a previously overlooked site of postnatal neurogenesis in the adult primate brain. The presence of immature neurons at different maturation stages, along with quiescent neural stem cells, suggests that this region may represent a novel neurogenic niche with potential implications for brain plasticity and repair.

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Antagonistic roles of Geminin and GemC1 in lineage specification and subventricular zone niche cytoarchitecture

The subventricular zone (SVZ) is a dynamic neurogenic niche housing neural stem cells (NSCs) and multiciliated ependymal cells (ECs), which together sustain neural regeneration and niche integrity. NSCs give rise to neurons and glia, while ECs provide essential structural and trophic support. A finely tuned balance between these cell types is critical for SVZ function, yet the mechanisms guiding their fate decisions remain poorly defined.

Geminin plays a central role in regulating NSC dynamics and lineage commitment. Its loss increases EC production at the expense of the NSC pool, prompting enhanced NSC self-renewal and reduced differentiation, alongside elevated cell cycle re-entry and S-phase activity in NSC progenitors. Geminin-deficient SVZ colonies show a neuronal lineage bias, and transcriptomic analyses identify Geminin as a key regulator of NSC activation and neurogenesis. These findings position Geminin as a major determinant of the balance between NSC maintenance and differentiation.

Conversely, GemC1 is a key upstream regulator of ependymal cell (EC) differentiation. Loss of GemC1 disrupts the balance between cell populations of the SVZ, leading to a shift toward a NSC phenotype at the expense of EC generation. This transition is associated with increased NSC abundance, elevated proliferative activity, and enhanced neurogenesis in the postnatal SVZ. Furthermore, GemC1-deficient cells display altered chromatin architecture at multiple genomic loci, consistent with an NSC transcriptional identity. These findings underscore GemC1's critical role in regulating the equilibrium between NSC maintenance and EC differentiation within the SVZ niche.

Together, our findings reveal that the Geminin superfamily governs SVZ architecture by modulating the balance between NSCs and ECs. The antagonistic roles of Geminin and GemC1 define a coordinated regulatory axis that shapes NSC and EC fate, offering insights into NSC dynamics and brain regeneration.

Posters

Using brain organoids to reveal the roles of ZNF90 and OVOL2 in Hominid brain development and evolution

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Identity of cells expressing the transcription factor Zbtb20 in the developing murine cerebellum

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Transcription factor Zbtb20 in mice vestibular system

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Combined effects of enriched environment and memantine on adult hippocampal neurogenesis

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Expression of the transcription factors PRDM14 and PRDM16 in human fetal choroid plexus

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Zbtb20 expression in the developinvg ventral human telencephalon

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