WELCOME

Dear Colleagues,

We are pleased to welcome you at the Black Sea Neurogenesis 2022 international conference. It is held five years after the Varna Primate Neurogenesis symposium, and it not only expands the number of speakers, but also enlarges the topics covered: from *neocortical neurogenesis in development and evolution* to *adult neurogenesis in animal models and humans,* to *brain plasticity and neurogenesis in a clinical context.*

During the last five years, there has been a significant progress understanding how neural stem cell generate neurons at the single cell level, both in the developing and adult brain. The emerging principles have important implications for developmental neurobiology, stem cell biology and start to reveal mechanisms underlying neurological and mental disorders as well as those of the repair following brain injury.

We are happy to welcome leading experts in developmental and adult neurogenesis as well as speakers who will present a translational view on how the process of neural stem cell proliferation would impact recovery after injury or the formation of brain tumors.

We hope that the presentations and discussions during the meeting will not only shed new light on this exciting topic in the field of neurosciences, but will also foster new partnerships between the participating researchers.

Yours sincerely,

Wieland B. Huttner
Anton B. Tonchev

Varna, Bulgaria, May 2022

BLACK SEA NEUROGENESIS 2022
PROGRAMME

THURSDAY	MAY 26 – GRAND HALL, FLAMINGO GRAND HOTEL (2ND FLOOR)
15:00	**Registration, poster placement**
18:00	Welcome address and opening Organizers Officials from MU-Varna
18:30	**Keynote lecture:** Wieland B. Huttner – Neural stem cells, human-specific genes, and neocortex expansion in development and human evolution
19:30	Bulgarian folk dance
19:45	Art Exhibition "Memory and the Black Sea" by Demetrius
20:15	Conference photograph
20:30	Evening reception: drinks and hors d'oeuvres (Flamingo Grand Hotel, Piano Bar Bailando)

FRIDAY	MAY 27 – GRAND HALL, FLAMINGO GRAND HOTEL

Session "Neocortical neurogenesis in development and evolution" **(9:00-11:00)**
Chair: Anton B. Tonchev

9:00-9:30	**Victor Borrell** Evolution of cortical progenitor cells: much to gain, much to lose
9:30-10:00	**Boyan Bonev** 3D epigenome evolution and the expansion of the neocortex in primates
10:00-10:30	**Tran Tuoc** Epigenome regulation in neocortex expansion and generation of neuronal subtypes

10:30-11:30 COFFEE BREAK AND POSTERS

Session "Mechanisms of lineage development in developing cortex" **(11:30-13:30)**
Chair: Victor Borrell

11:30-12:00 **Simon Hippenmeyer**
Mechanisms of neural stem cell lineage progression in developing cerebral cortex

12:00-12:30 **Olivier Raineteau**
Single cell analysis of the dorsal V-SVZ reveals differential quiescence of postnatal pallial and subpallial neural stem cells driven by TGFβ/BMP

12:30-13:00 **Stavros Taraviras**
Determining the ependymal lineage

13:00-13:30 **Irina Stoyanova**
Does ghrelin stimulate neurogenesis in the cerebral cortex?

13:30-14:30 LUNCH

Session "Adult hippocampal neurogenesis" **(15:00 – 16:30)**
Chair: Hagen B. Huttner

14:30-15:00 **Federico Calegari**
Making brains with more neurons, from the womb to the grave

15:00-15:30 **Ionut Dumitru**
Neurogenesis in the human hippocampus

15:30-16:00 **Juan Manuel Encinas**
Neuronal hyperexcitation unveils the proinflamatory action of hippocampal neural stem cells

16:00-16:30 **Manlio Vinciguerra**
Histone variants, hippocampal plasticity and social behavior

16:30-17:00 COFFEE BREAK AND POSTERS

Session "Brain plasticity and neurogenesis in a clinical context" **(17:00 – 19:30)**

Chair: Federico Calegari

17:00-17:30 **Hannelore Ehrenreich**
Introducing the brain erythropoietin circle to explain adaptive brain hardware upgrade and improved performance

17:30-18:00 **Thorsten Döppner**
Therapeutic implications of extracellular vesicles in preclinical stroke models

18:00-18:30 **Pavle Andjus**
Studies on neurogenesis in rodent models of pathophysiology and plasticity of the CNS

18:30-19:00 **David Lutz**
Ghrelin-mediated neuroplasticity and neurogenesis

19:00-19:30 **Janine Gronewold**
Imaging markers for neuroplasticity in clinical stroke studies

20:30 **Dinner (Maritim Paradise Blue Hotel, Sea Hall)**

SATURDAY MAY 28 – GRAND HALL, FLAMINGO GRAND HOTEL

Session "Progenitors and neurogenesis in adult primates" **(9:00 – 11:30)**

Chair: Hannelore Ehrenreich

9:00-9:30 **Elly M. Hol**
Single-cell analysis of progenitors in the human subventricular zone

9:30-10:00 **Hagen B. Huttner**
Studying adult neurogenesis in humans: insights from C14-birthdating and 15N-thymidine methodology

10:00-10:30 **Anton B. Tonchev**
Transcriptional landscape of postischemic primate subventricular zone

10:30-11:00 **Loïc Chareyron**
Life and death of immature neurons in the primate amygdala

11:00-11:30 COFFEE BREAK AND POSTERS

Session "Neurogenesis – further aspects" **(11:30 – 13:00)**

Chair: Thorsten Döppner

11:30-12:00 **Fadel Tissir**
The delicate balance between proliferation of neural stem cells and neoplasia:
Implication of DIAPH3 in neurogenesis and glioblastoma

12:00-12:30 **Bogdan Draganski**
In vivo and ex vivo assessment of adult neurogenesis

12:30-13:00 **Dimitre Staykov**
Neurological consequences of COVID-19

Wieland B. Huttner

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Neural stem cells, human-specific genes, and neocortex expansion in development and human evolution

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

- The human-specific gene ARHGAP11B amplifies basal progenitors.
- This ability of ARHGAP11B depends on a single C-to-G base substitution.
- ARHGAP11B protein is imported into mitochondria and promotes glutaminolysis.
- ARHGAP11B can expand the primate neocortex.
- ARHGAP11B-mediated neocortex expansion increases cognitive performance.
- Neandertal apical progenitors (APs) exhibit a shorter metaphase than modern humans.
- Neandertal APs make more chromosome segregation errors than modern humans.
- Modern human vs. Neandertal transketolase-like 1 (TKTL1) differ by only 1 amino acid.
- Modern human, but not Neandertal, TKTL1 increases basal radial glia and neurons.
- In fetal modern human neocortex, TKTL1 is most highly expressed in the frontal lobe.

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

Víctor Borrell

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Evolution of cortical progenitor cells: much to gain, much to lose

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

- Cortical progenitor cell diversity increased in mammalian evolution.
- Cortical size and folding were reduced secondarily in the rodent lineage.
- The differential regulation of conserved genetic mechanisms was important in the evolution of neurogenesis.
- MIR3607 is highly expressed in cortical progenitors of primates and carnivores.
- MIR3607 promotes apical Radial Glia Cell (aRGC) amplification and polarity.
- MIR3607 strongly blocks APC expression and activates beta-Catenin signaling.
- MIR3607 expression in aRGCs was secondarily lost in the rodent lineage.
- Loss of MIR3607 expression led to increased APC, smaller progenitor pools and reduced cortex size.
- Differential regulation of MIR3607 expression was key in cortex size evolution.

https://vborrelllab.wixsite.com/website

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3D epigenome evolution and the expansion of the neocortex in primates

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

- Physical proximity between regulatory elements is a novel epigenetic layer with important consequences for development and evolution.
- Neurog2 coordinates the remodeling of the epigenetic landscape across multiple layers in both mouse and human cortical development.
- Large-scale identification and validation of human cell-type specific enhancers in apical and basal progenitors.
- Structural variations during primate evolution alter the 3D epigenome local environment and contribute to enhancer activity and specificity.
- In vitro models for cortical development (2D and cerebral organoids) from primate iPSCs represent a powerful way to dissect and perturb the epigenetic landscape associated with brain development and evolution.

www.bonevlab.com

Tran Tuoc

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Epigenome regulation in neocortex expansion and generation of neuronal subtypes

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

- Epigenetic profiling reveals epigenetic divergence between human basal progenitors (BPs) during corticogenesis.
- H3K9 acetylation (H3K9ac) is low in murine BPs and high in human BPs.
- Increased H3K9ac specifically induces BP proliferation and gyrification of the murine cortex.
- H3K9ac drives BP amplification by increasing expression of the evolutionarily regulated gene, TRNP1, in the developing cortex.
- Pax6 expression is required for H3K9ac–induced genesis of BPs and cortical expansion.
- H3K9 acetylation activates interneuronal fate in cortical progenitors in developing mouse cortex.
- H3K9 acetylation promotes proliferation of interneuronal progenitor-like cells in cortex of *Pax6* mutant mouse models.

https://www.ruhr-uni-bochum.de/mhg/

Simon Hippenmeyer

Institute of Science and Technology Austria (ISTA)

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Mechanisms of neural stem cell lineage progression in the developing cerebral cortex

In the field of *mechanisms of lineage development in developing cortex,* I will report the following:

- Clonal analysis using MADM (Mosaic Analysis with Double Markers) technology.
- Quantitative framework of radial glial progenitor (RGP) lineage progression.
- Genetic dissection of cell-autonomous candidate gene function and tissue-wide effects.
- Single cell genetic MADM analysis of EED/PRC2 function in RGP lineage progression.
- Genetic profiling in sparse versus global Eed/PRC2 deletion paradigm.
- Role of PRC2 function in cortical astrocyte production.
- Model of PRC2 function in RGP lineage progression.

https://ist.ac.at/en/research/hippenmeyer-group/

Olivier Raineteau

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Single cell analysis of the dorsal V-SVZ reveals differential quiescence of postnatal pallial and subpallial neural stem cells driven by TGFβ/BMP-signalling

In the field of *mechanisms of lineage development in developing cortex*, I will report the following:

- The postnatal dorsal V-SVZ domain contains an intermix of NSCs expressing pallial or subpallial markers.
- Activated NSCs are heterogeneous and are primed for lineage differentiation.
- Quiescent NSCs emerge at postnatal stages within the dorsal V-SVZ.
- Pallial/subpallial NSCs enter distinct states of quiescence that parallel their different contribution to postnatal neurogenesis.
- Bmpr1a integrates TGFβ/BMP-signalling to synchronize quiescence induction and blockade of neuronal differentiation to rapidly silence pallial germinal activity after birth.

https://www.sbri.fr/team/4b-cellular-programming-brain

Stavros Taraviras

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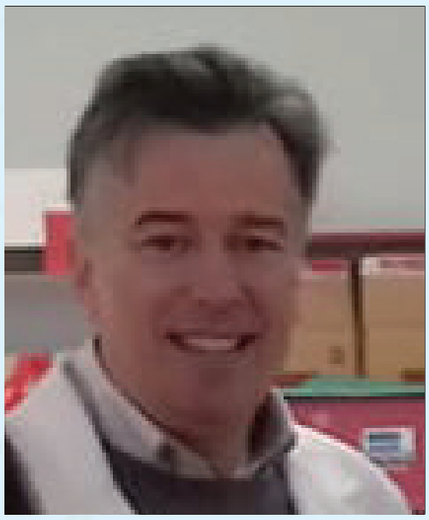
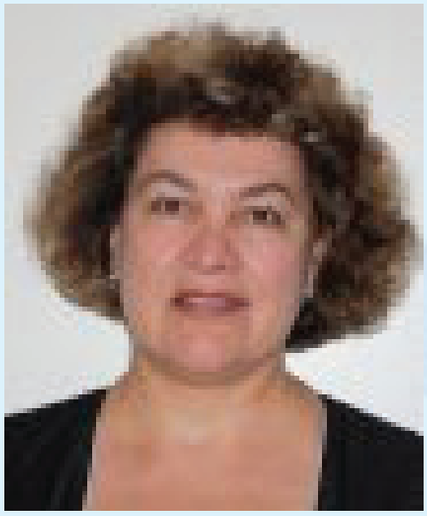


Determining the ependymal lineage

In the field of *mechanisms of lineage development in developing cortex,* I will report the following:

- GemC1 and McIdas are expressed during late embryogenesis in radial glial cells lying next to the lateral ventricles.
- Lack of GemC1 leads to hydrocephalus in mouse and humans.
- GemC1 is crucial for the commitment of radial glial cells towards the ependymal lineage and acquire adult neural stem cells characteristics.
- McIdas is required for differentiation of multiciliated ependymal cells.
- GemC1 and McIdas transcriptionally activate key transcription factors of ependymal cell differentiation.
- GemC1 and McIdas can reprogram cells towards the ependymal lineage.

http://stemcellslab.upatras.gr/



Irina Stoyanova

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Does ghrelin stimulate neurogenesis in the cerebral cortex?

In the field of *mechanisms of lineage development in developing cortex*, I will report the following:

- Dissociated cortical neurons were used as an in vitro model of stroke.
- On day 1 cultures were subjected to hypoxia for 6 hours.
- Before hypoxia half of them were supplemented with ghrelin, the other half of the cultures were used as a control.
- At the end of the first week both experimental groups were stained immunocytochemically for detection of Ki67, doublecortin (DCX), and TF Zbtb20.
- The quantitative analysis showed that expression of Ki67 a week after exposure to hypoxia was downregulated significantly, while ghrelin supplementation led to high proliferation rate.
- Ghrelin also elevated the expression of DCX and Zbtb20 compared with the control groups.

https://scholar.google.com/citations?hl=en&user=le5HhTIAAAAJ

Federico Calegari

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Making brains with more neurons, from the womb to the grave

In the field of *adult neurogenesis and cognitive function,* I will report the following:

- Genetic expansion of neural stem cells is used to increase neurogenesis over the course of life.
- Increased neurogenesis improves sensory and cognitive performance rescuing age-related cognitive deficits.
- Newborn neurons differentially modulate parallel streams of information processing within the hippocampus.
- A new model for different computational functions of newborn neurons will be proposed.

https://tu-dresden.de/cmcb/crtd/forschungsgruppen/crtd-forschungsgruppen/calegari?set_language=en

Ionut Dumitru

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Neurogenesis in the human hippocampus

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

- We identified and characterized neural progenitors in the childhood human hippocampus by single nucleus RNA sequencing
- The childhood human hippocampal progenitors resemble mouse neural progenitors
- Human childhood neural stem cells express canonical neurogenic markers as: Nestin, GFAP, PAX6, SOX6, HOPX, etc.
- Human childhood intermediate progenitors express canonical neurogenic markers as: TFAP2C, EOMES, SOX2, ASCL1, etc.
- Human childhood neuroblasts express canonical neurogenic markers as: EOMES, IGFBPL1, SOX4, SOX11, etc.
- We validated by RNAscope EZH2 as human intermediate progenitor marker, GLRA2 as neuroblast marker and KCNH7 as immature granule neuron marker.
- Using the transcriptional information obtained from childhood hippocampal progenitors, we identified in adult human hippocampal samples analyzed by single nucleus RNA sequencing nuclei originating from cells that express neural progenitor markers and resemble human childhood and mouse neural progenitors.

https://ki.se/en/cmb/jonas-frisens-group

Juan M. Encinas

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The differential effects of neuronal hyperexcitation levels on hippocampal neurogenesis

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

- Lower levels of neuronal hyperexcitation, mimicking interictal epileptiform activity, increase neurogenesis.
- Medium levels of neuronal hyperexcitation, mimicking mesial temporal epilepsy, trigger aberrant neurogenesis.
- High levels of neuronal hyperexcitation, mimicking mesial temporal lobe epilepsy with hippocampal sclerosis ablate neurogenesis.
- High levels of neuronal hyperexcitation, mimicking mesial temporal lobe epilepsy with hippocampal sclerosis induce the conversion of neural stem cells (NSCs) into pro-inflammatory reactive NSCs (React-NSCs).
- ATP and purinergic 2X receptors (P2XR) mediate the effects of neuronal hyperexcitation of NSCs and neurogenesis.

https://www.achucarro.org/en/research/group/laboratory-of-neural-stem-cells-and-neurogenesis

Manlio Vinciguerra

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Histone variants, hippocampal plasticity and social behavior

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

- Epigenetic mechanisms: focus on histone variants.
- Histone variants modulate human neurogenesis. They have important roles in the memory and switching of gene expression states during brain development.
- Histone variants MacroH2A1 (present in two isoforms, macroH2A1.1 and macroH2A1.2) is the largest histone variant, with pleiotropic roles in cell differentiation and plasticity.
- MacroH2A1.1$^{-/-}$ mice exhibit an enhancement both of sociability and of active stress-coping behavior.
- MacroH2A1.1$^{-/-}$ mice also display an increased hippocampal synaptic plasticity, accompanied by significant neurotransmission transcriptional networks changes.

http://transtem.org/en/news/

Hannelore Ehrenreich

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Introducing the brain Erythropoietin circle to explain adaptive brain hardware upgrade and improved performance

In the field of *brain plasticity and neurogenesis in a clinical context,* I will report the following:

- My previous work - from clinical studies first (!) to rodents - demonstrated hematopoiesis-independent EPO effects on neuroprotection & cognitive performance.
- All this suggested that brain-expressed EPO serves fundamental, previously overlooked physiological functions in mammals.
- Focusing on murine CA pyramidal neurons, I introduced the brain EPO circle as model of enduring neuroplasticity through enhanced dendritic spine density and swift generation of new functional neurons without proliferation: Brain 'hardware upgrade'.
- The brain EPO circle can be entered anywhere, starting either with mild to moderate inspiratory hypoxia, with recombinant human (rh)EPO treatment or – intriguingly - with motor-cognitive challenge: Principle of 'brain doping' through functional hypoxia.
- To describe the relative hypoxia that arises upon enhanced neuronal activity and mediates brain EPO expression, I coined the term 'functional hypoxia'.
- The entire precursor cell lineage in adult CA, ready to differentiate towards pyramidal neurons in response to brain-expressed or rhEPO, remains 'in flow', characterized by transient waves of non-proliferating precursors that rise at particular time windows.
- In this process, neuron-microglia counterbalance plays a pivotal role with both microglial and pyramidal EPOR being critical for neuronal differentiation upon EPO.
- Hypothesizing that the brain EPO system enables escalation of performance on demand, I suggest to exploit 'brain doping' for treatment of brain diseases.

https://www.mpinat.mpg.de/de/ehrenreich

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Therapeutic implications of extracellular vesicles in preclinical stroke models

In the field of *brain plasticity and neurogenesis in a clinical context,* I will report the following:
- Causal stroke therapy is limited to recanalizing strategies.
- Poststroke neuroprotection has failed until recently.
- Transplantation of adult stem cells helps boost poststroke endogenous neurogenesis.
- Extracellular vesicles derived from such stem cells induce pleiotropic effects stimulating neurological recovery.

https://www.ukgm.de/ugm_2/deu/ugi_neu/index.html

Pavle R. Andjus

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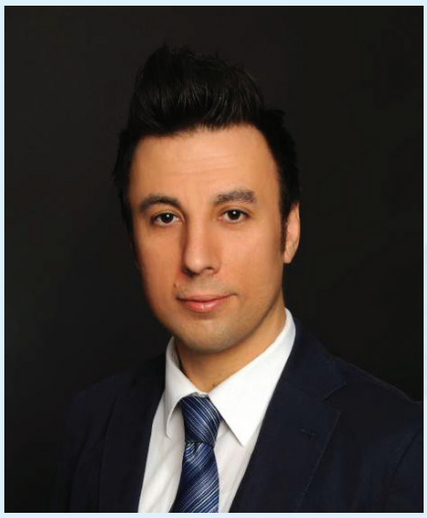
Studies on neurogenesis in rodent models of pathophysiology and plasticity of the CNS

In the field of *adult neurogenesis in health and disease*, I will report the following:
- The animal model of long term (1year) survival after cardiac arrest ischemia.
- MRI and immunocytochemistry of immune cell infiltration and BBB compromise.
- Microglial markers of neuroinflammation.
- Neurogenesis in the subventricualar zone and DCX+ neuroblast migration.
- ECM protein Tenascin C in proliferation and differentiation.
- TnC KO mouse model in standard vs enriched environment (EE).
- Proliferation in the dentate gyrus and SVZ is augmented in EE regardless of phenotype.
- Proliferation in the TnC KO d. gyrus and SVZ is augmented regardless of rearing.
- Differentiation (BrdU/NeuN+ cells) in the hippocampus/d. gyrus is augmented in EE regardless of phenotype.
- Differentiation in the SVZ is suppressed in EE regardless of the phenotype.
- Differentiation in the TNC KO hippocampus/d. gyrus is augmented regardless of rearing.

http://clm.bio.bg.ac.rs/laboratory/staff/



David Lutz

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Ghrelin-mediated neuroplasticity and neurogenesis

In the field of *brain plasticity and neurogenesis in a clinical context*, I will report the following:
- Pleotropic functions of ghrelin.
- Ghrelin and neurogenesis.
- Ghrelin-mediated neuroprotection in experimental models of neurotrauma and neurodegeneration.
- GHSR1 signalling in neurogenesis and neuroregeneration.

https://loop.frontiersin.org/people/509320/editorial

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Imaging markers for neuroplasticity in clinical stroke studies

In the field of *brain plasticity and neurogenesis in a clinical context*, I will give an overview about:

- Structural and functional imaging markers for neuroplasticity.
- Structural markers include: lesion volume, total brain volume, ROI volume, white matter integrity (fractional anisotropy, mean diffusivity, axial diffusivity, radial diffusivity).
- Functional markers include: task-based functional MRI, resting-state functional MRI, task-based PET/PET-MRI.
- Chances and challenges of imaging markers for neuroplasticity in clinical stroke studies.
- Chances include: surrogate endpoint for therapeutic studies, public data repositories, translation from animal to human.
- Challenges include: comparability of brain lesions in animals and humans, comorbidity profiles, brain complexity, multidimensional stroke influences, study design, standardization of data collection and analysis, clinical utility.

https://www.uni-due.de/neurologie/neurolab/bio_jgronewold.php

Elly M. Hol

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Single-cell analysis of progenitors in the human subventricular zone

In the field of *progenitors and neurogenesis in adult primates*, I will report the following:

- A splice-variant of GFAP, GFAP*delta*, is a marker for human neural progenitors in the human developing and adult subventricular zone.
- Neural stem cells from elderly individuals and Parkinson patients form neurospheres and can be differentiated into neurons and glia in vitro.
- Ventricular cerebrospinal fluid increases neural progenitor proliferation and continues to stimulate neural progenitors throughout aging.
- Parkinson neural progenitors show significant changes at both transcriptome and proteome compared to neurological controls.
- Our transcriptomic and proteomic data suggest that the progenitors transit into a primed-quiescent state, that is in an “alert” non-proliferative phase in Parkinson.
- Single cell profiling of adult human neural progenitors identifies SFRP1 as a target to re-activate progenitors.

www.translationalneuroscience.nl

Hagen B. Huttner

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Studying adult neurogenesis in humans: insights from C14-birthdating and 15N-thymidine methodology

In the field of *progenitors and neurogenesis in adult primates,* I will report the following:

- Concept of neuroregeneration in humans.
- Adult neurogenesis studied by C14 retrospective carbon dating focussing on findings for the human healthy hippocampus and healthy amygdala.
- Adult neurogenesis studied by C14 retrospective carbon dating focussing on findings on neuronal cell turnover in cortical stroke, basal ganglia stroke, as well as effects on hippocampus.
- Introduction of 15N-thymidine infusion to study human cell tunrover *in vivo* including results from a first-in-CNS-tissue.

https://www.ukgm.de/ugm_2/deu/ugi_neu/index.html

Anton B. Tonchev

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Transcriptional landscape of postischemic primate subventricular zone

In the field of *progenitors and neurogenesis in adult primates,* I will report the following:

- The adult macaque monkey subventricular zone (SVZ) contains different subdomains, including anterior (SVZa) and inferior (SVZi).
- The monkey SVZa is capable of enhanced neurogenesis after global brain ischemia.
- The monkey SVZi shows enhanced progenitor proliferation without neurogenesis following ischemia.
- Transcriptomic analyzes demonstrate that global brain ischemia elicits differential gene expression response in SVZa and SVZi.
- Novel gene markers for primate SVZ including the apelin receptor are strongly expressed in the primate SVZa niche upon ischemic insult.
- An interactive open online image database (www.monkey-niche.org) shows *in situ* data of gene expression in monkey SVZ for hundreds of genes.

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

Loïc J. Chareyron

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Life and Death of Immature Neurons in the Primate Amygdala

In the field of *progenitors and neurogenesis in adult primates*, I will report the following:

- A large population of immature neurons is present in the paralaminar nucleus of the primate amygdala.
- These immature neurons express the markers Bcl-2, NeuN and DCX.
- This population of immature neurons decreases by 40% between 1 and 5 years in the paralaminar nucleus of the rhesus monkey.
- In parallel, the number of mature neurons increases by 570% between 1 and 5 years.
- Immature neurons migrate from the caudally situated SVZ to the paralaminar nucleus after one year of age.
- The paralaminar nucleus receives direct input from the hippocampal formation.
- Early hippocampal injury is followed by an increase in the number of immature and mature neurons in the monkey amygdala.
- In contrast, an adult lesion of the hippocampus is followed by an increase in the number of mature neurons and a decrease in the number of immature neurons.

https://applicationspub.unil.ch/interpub/noauth/php/Un/UnPers.php?PerNum=1242298&LanCode=8

Fadel Tissir

College of Health and Life Sciences, Doha, Quatar, Université Catholique de Louvain, Belgium

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The delicate balance between proliferation of neural stem cells and neoplasia: Implication of DIAPH3 in neurogenesis and glioblastoma

In the field of *neurogenesis and glioblastoma*, I will report the following:
- DIAPH3 localizes to centrosome and is required for assembly of mitotic spindle.
- DIAPH3 deficiency causes a massive loss of neural progenitors.
- Cortex-specific ablation of Diaph3 disrupts neurogenesis.
- Downregulation of DIAPH3 alters expression and localization of several MAPs.
- Absence of DIAPH3 weakens the spindle assembly checkpoint.
- DIAPH3 secures nuclear division and its loss causes aneuploidy.
- Combined loss of DIAPH3 and p53 triggers glioblastoma.

https://www.researchgate.net/profile/Fadel-Tissir-2

Bogdan Draganski

University of Lausanne, Switzerland

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Adult hippocampal neurogenesis – how close are we to find in vivo correlates in humans?

In my presentation I will cover the following topics:

- Studying adult hippocampal neurogenesis in humans – current achievements and controversies.
- Readily available and novel brain imaging techniques tapping into human neurogenesis.
- Epileptic-seizure induced hippocampal neurogenesis.
- Electro-convulsive therapy induced hippocampal neurogenesis.
- Modulating hippocampal neurogenesis - candidate behavioral correlates and their implication for the clinics.
- Outlook – what can we expect from the future.

https://www.unil.ch/crn/en/home/menuinst/research-labs/research-in-neuroimaging-lren/bogdan-draganski.html

Dimitre Staykov

Department of Neurology, Hospital of the Brothers of St. John, Eisenstadt, Austria

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Neurological consequences of COVID-19

In my presentation I will cover the following topics:

- Overview of the mechanisms of damage to the nervous system caused by SARS-CoV-2 infection.
- Inflammatory complications affecting the central nervous system.
- Macro- and microvascular complications affecting the central nervous system.
- Peripheral neurological consequences of SARS-CoV-2 infection.
- Influence of SARS-CoV-2 infection on neurogenesis.
- Post-Covid-Syndrome.

https://www.barmherzige-brueder.at/site/eisenstadt/medizinpflege/abteilungeninstitute/neurologie/ueberuns/article/34081.html

POSTERS

Role of gut microflora in neuroinflammation and neurogenesis in stroke models: an experimental paradigm

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Role of mitochondrial biogenesis in ischemic stroke: an overview

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Reduced neuroinflammation and enhanced neurogenesis in models of melatonin deficit

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Evaluation of mitochondrial function in patients with Parkinson's disease

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Role of Pten in RGP lineage progression at single-cell resolution using Mosaic Analysis with Double Markers

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Interneuronal defects in the somatosensory cortex of Zbtb20$^{-/-}$ mice

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Histopathological aspects of SARS-CoV-2-induced hippocampal damage

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Pax2 expression in developing cerebellum of Zbtb20$^{-/-}$ mice

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3D-Visualization of the Enteric Nervous System by CUBIC-based tissue clearing of human colon

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Stem cells gene expression along the rostro-caudal axis of the primate subventricular zone

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Phenotype of the Apelin receptor-expressing cells in the subventricular zone and the rostral migratory stream of the adult mouse brain

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Microglial expression of HAM-56 is associated with neural progenitor density and proliferation in the human fetal telencephalon

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H3 acetylation selectively promotes basal progenitor proliferation and neocortex expansion by activating TRNP1 expression

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Mitochondrial dysfunction and NGF levels as predictive markers of Autism Spectrum Disorder

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