СТОВБУРОВІ КЛІТИНИ МОЗКУ:

РЕГЕНЕРАЦІЯ

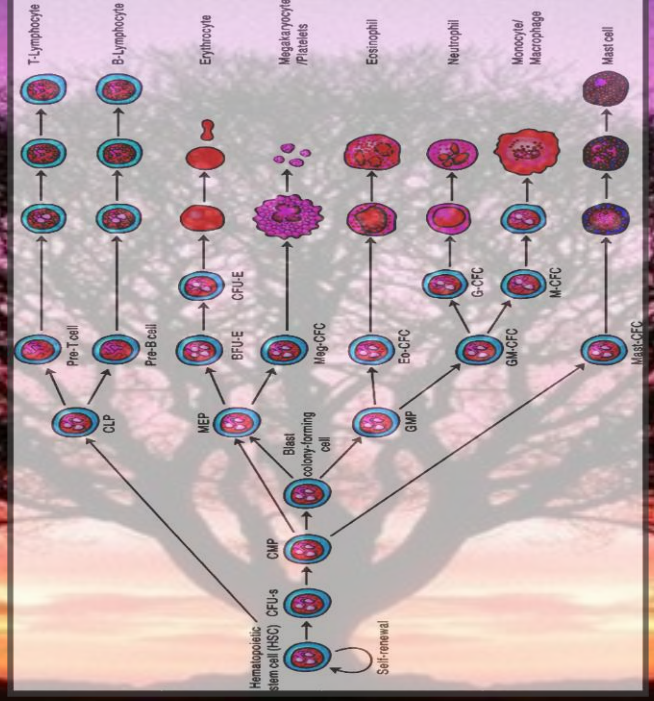
функція

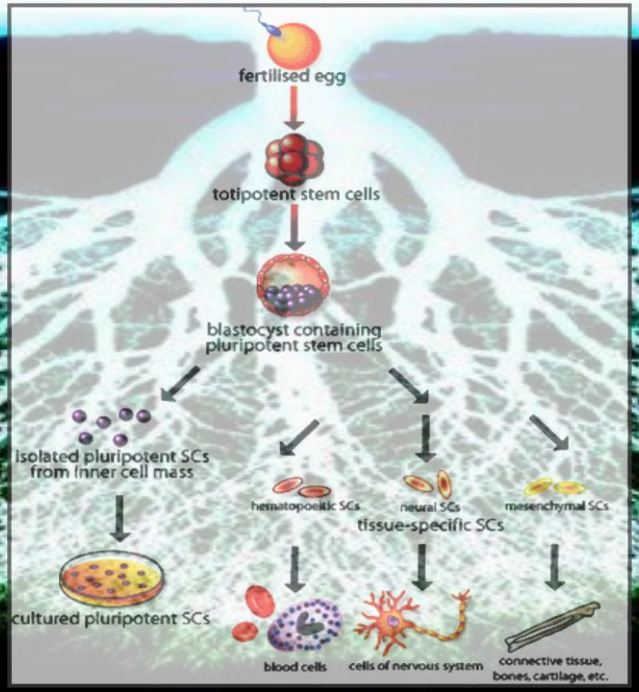
vs

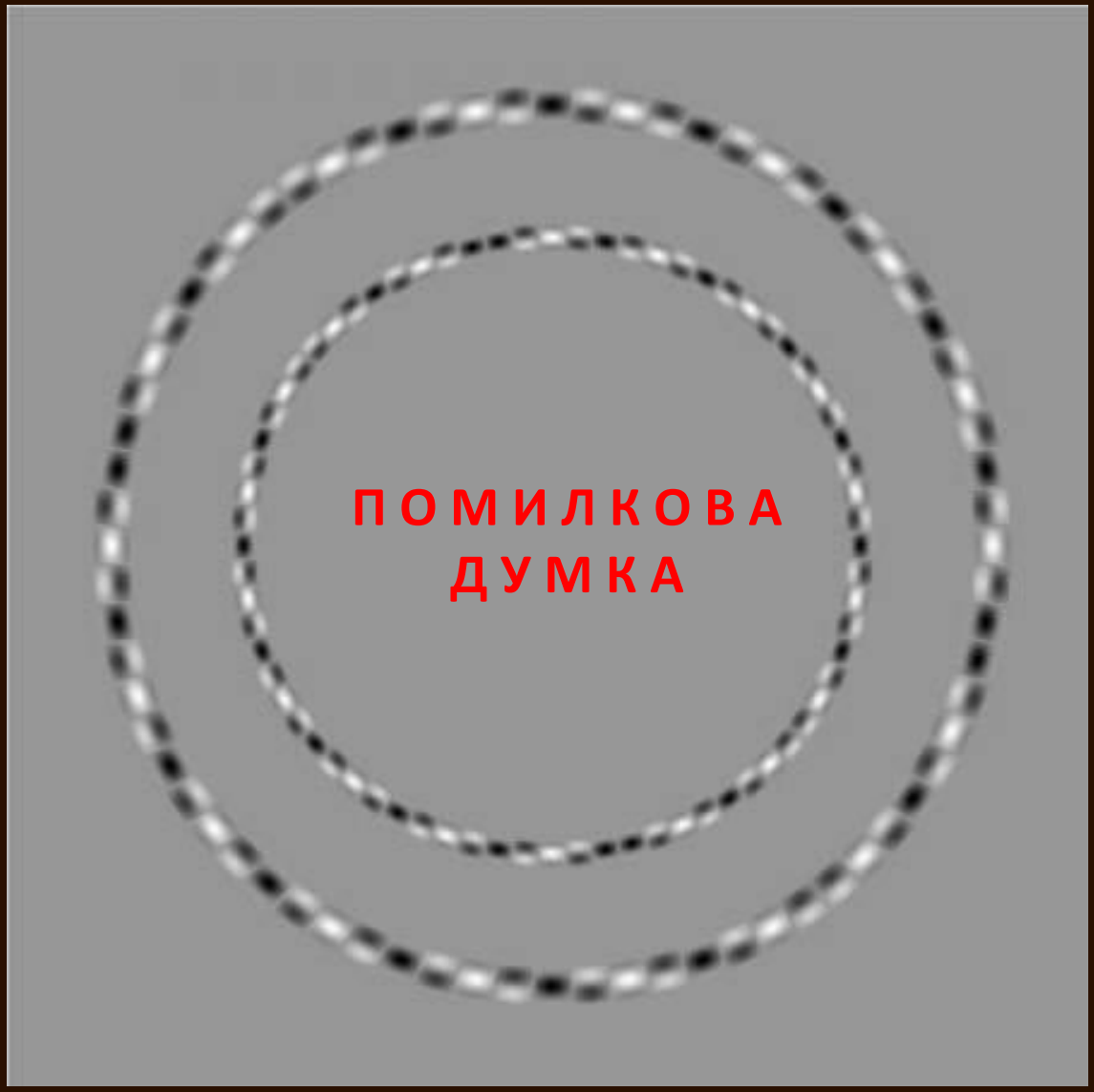
В. МЕДВЕДЄВ

стовбури

і клітини







головна властивість стовбурової клітини – відсутність властивостей

ТРИ ОСНОВНІ ВЛАСТИВОСТІ СТОВБУРОВИХ КЛІТИН:

1. ЗДАТНІ ДО ПОДІЛУ.
2. ЗДАТНІ ДО САМОВІДТВОРЕННЯ.
3. ДАЮТЬ ПОЧАТОК КЛІТИНАМ, ЩО ВИЙШЛИ З КЛІТИННОГО ЦИКЛУ.

ПОТЕНТНІСТЬ

Рівень потентності визначається кількістю видів диференційованих клітин серед загального числа нащадків стовбурової.

- **тотіпотентні**
- **плюрипотентні**
- **мультипотентні**

Мультипотентні стовбурові клітини

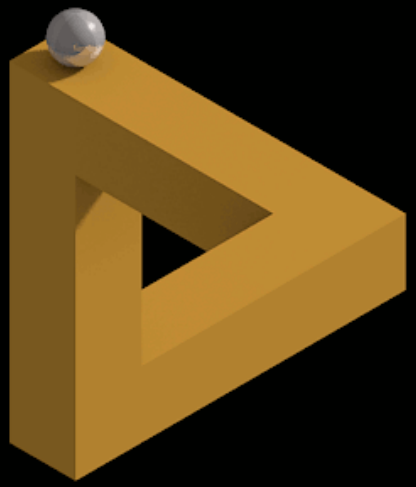
- Дають початок клітинам, які відповідають за основну функцію однієї ткани.

- Не здатні відтворити тканину в цілому (*функціональну архітектоніку*), що для нервової тканини має ключове значення.

Види мультипотентних стовбурових клітин

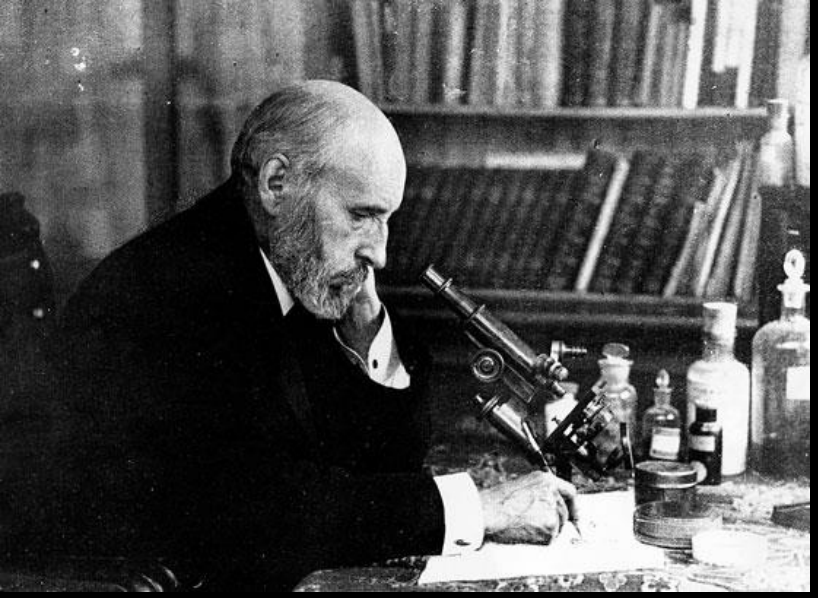
- мезенхимальні;
- гемопоетичні;
- міогенні;
- остеогенні;
- одонтогенні;
- ентодермальні;
- ектодермальні;
- **нейрогенні**;
- Стовбурові клітини нервового гребня

нез’ясовано



- потентний статус СК зрілого організму
- пластичність потентного статусу СК
- природа мезенхімальних СК
- СК і еквігеномність організму
- СК у функції і патології

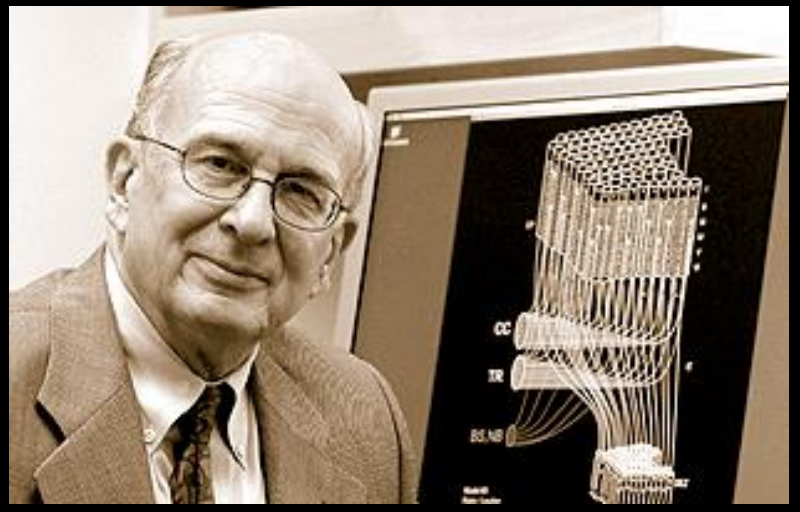
Нейрогенні стовбурові клітини



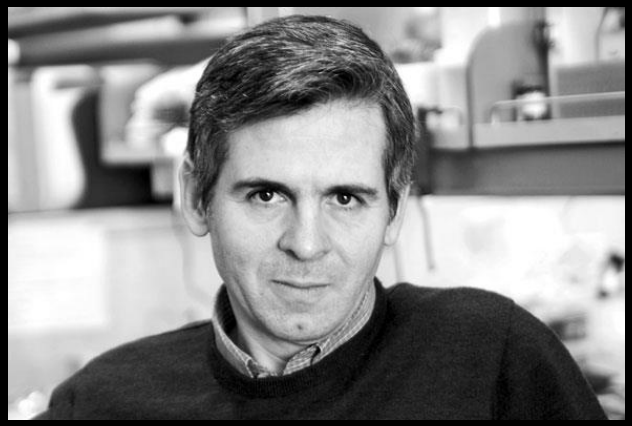
Santiago Ramon-y-Cajal

Joseph Altman

Fernando Nottebohm



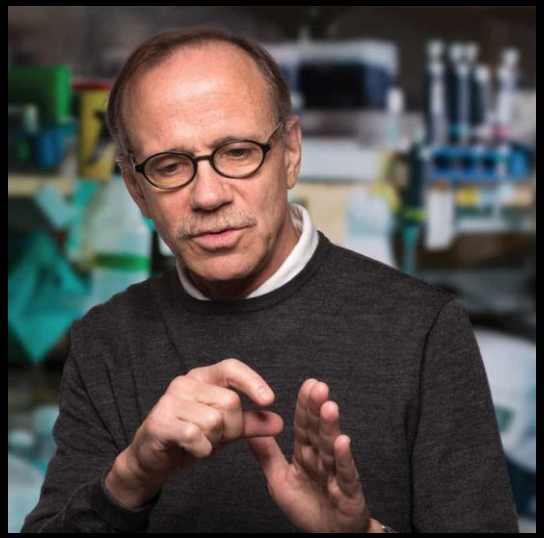
Pasco Rakic



Arturo Alvarez-Buylla

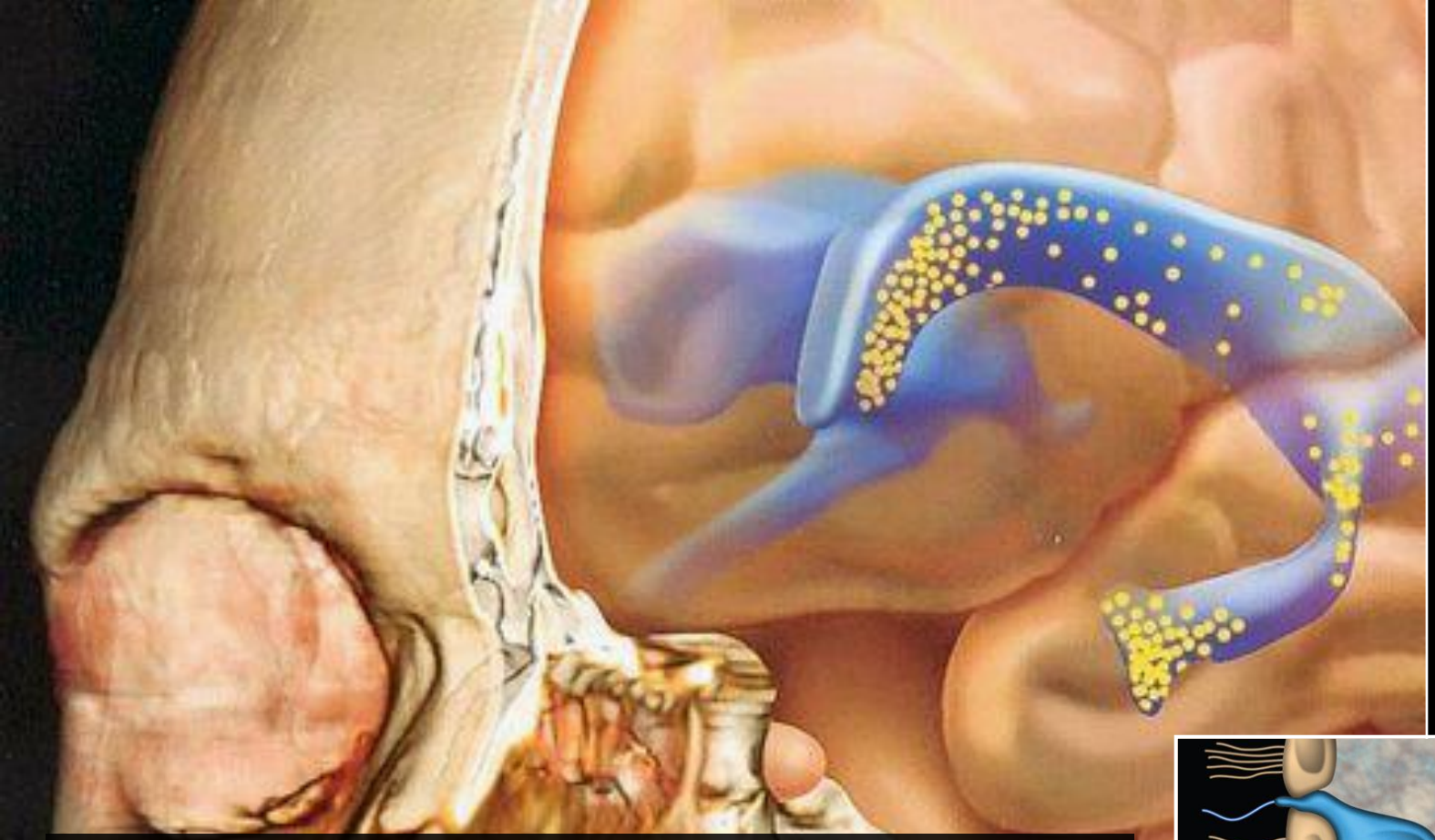
Elizabeth Gould

Fiona Doetsch

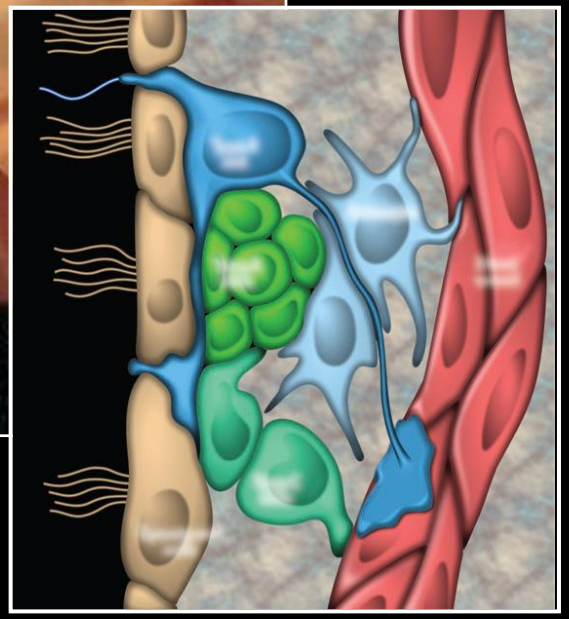


Fred Gage

in brevi



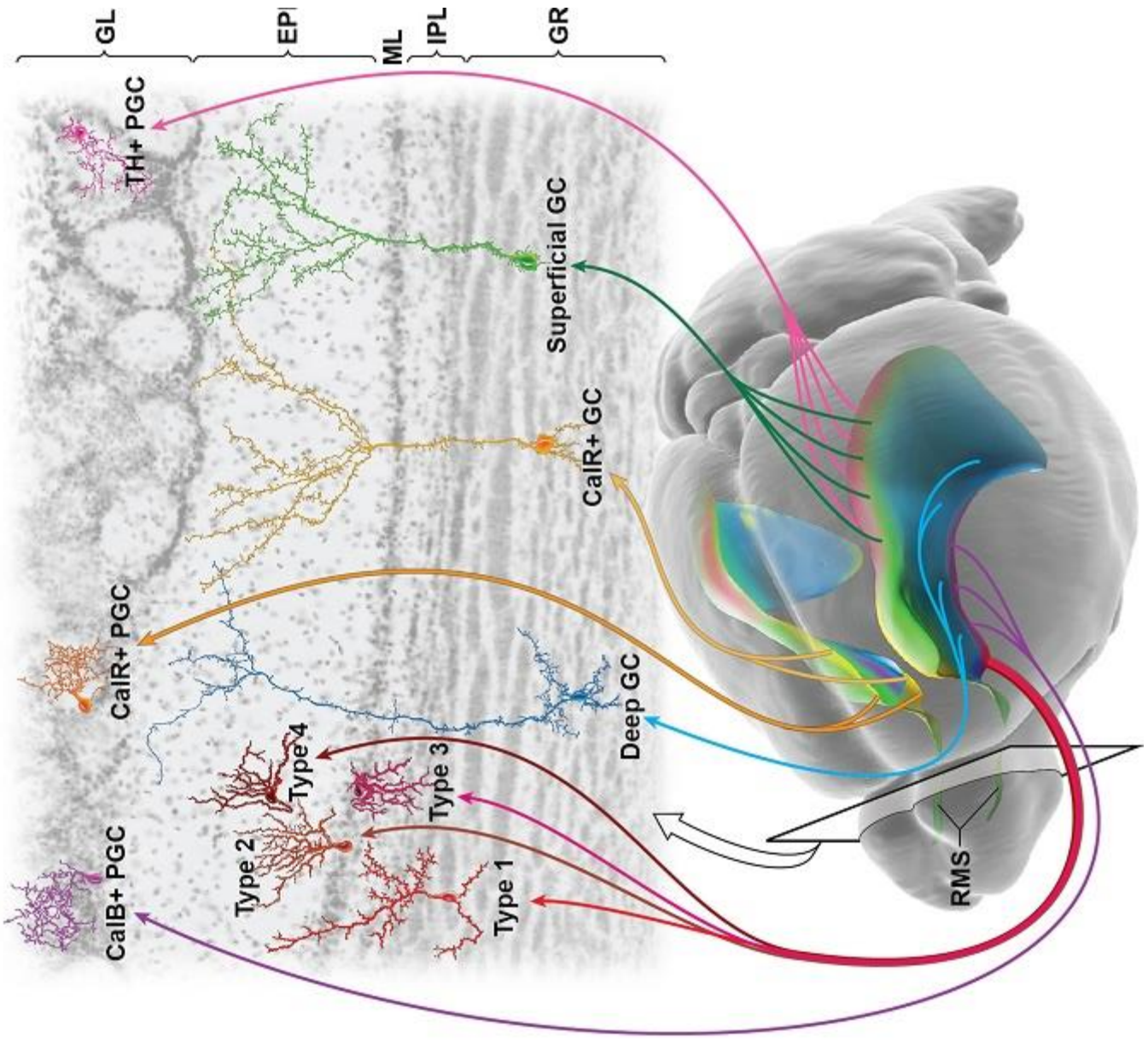
- низька мітотична активність
- чутливі до медіаторів і факторів росту
- широко мігрують
- функція мозку, а не регенерація.



Trends Neurosci. 2014 October ; 37(10): 563–571. doi:10.1016/j.tins.2014.08.006.

Adult neural stem cells stake their ground

Daniel A. Lim[1,2,3] and **Arturo Alvarez-Buylla**[1,2]



Trends Neurosci. 2014 October ; 37(10): 563–571. doi:10.1016/j.tins.2014.08.006.

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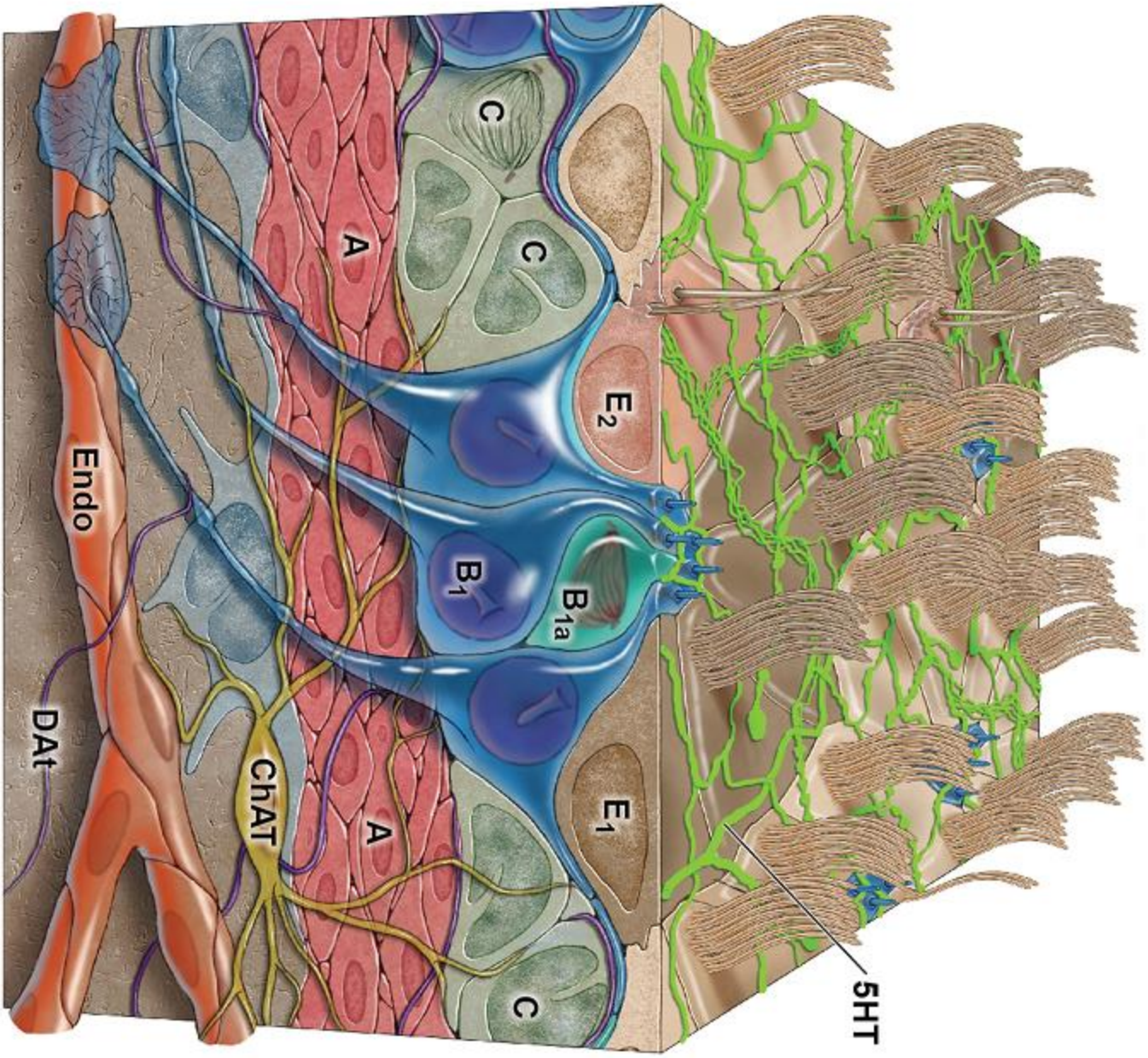
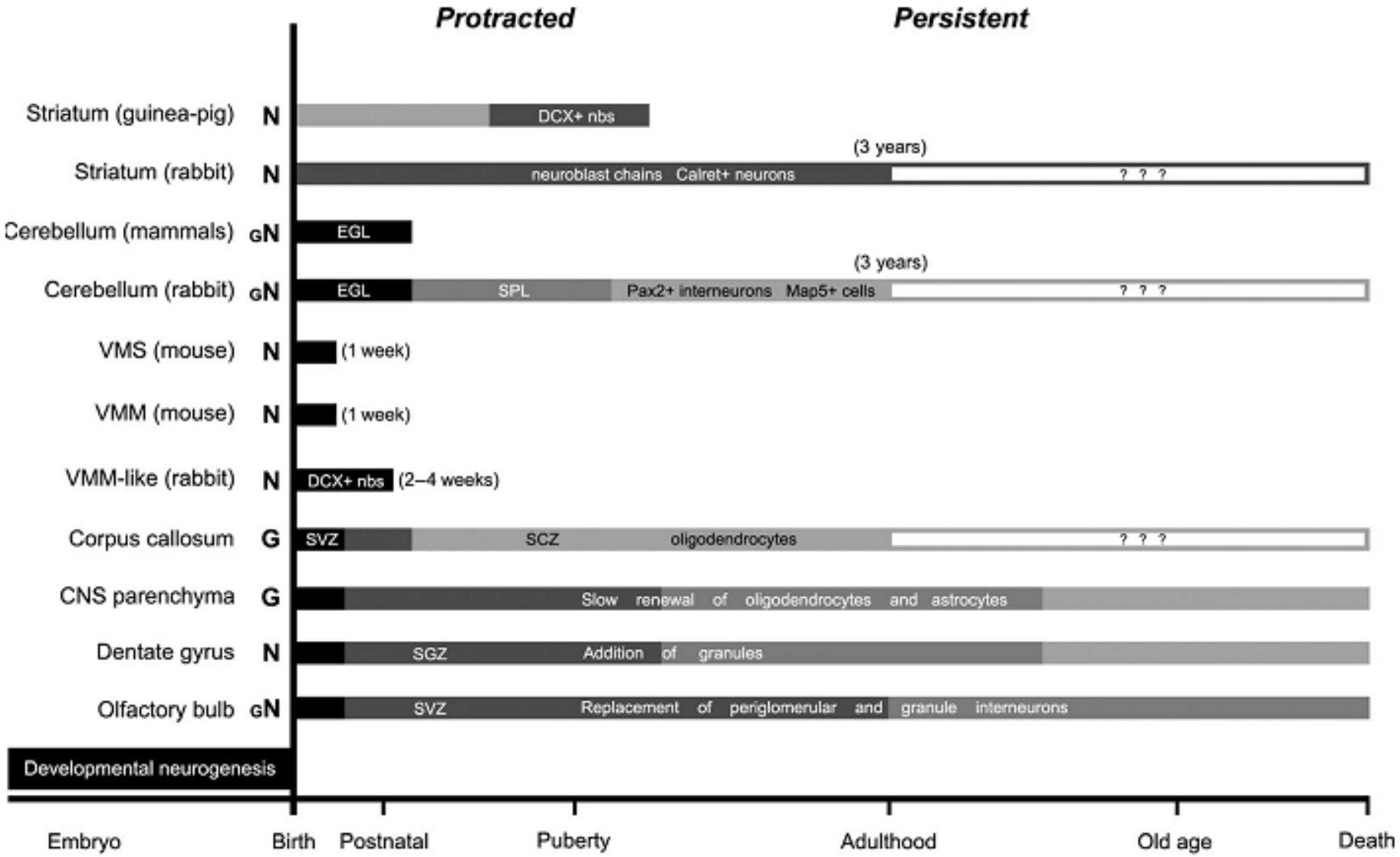




European Journal of Neuroscience, Vol. 34, pp. 930–950, 2011 doi:10.1111/j.1460-9568.2011.07832.x

Adult neurogenesis in mammals – a theme with many variations

Luca Bonfanti[1,2] and Paolo Peretto[1,3]

Comparative aspects of adult neural stem cell activity in vertebrates

Heiner Grandel · Michael Brand

Latent progenitors	Reparative neurogenesis
+	–
?	–
?	–

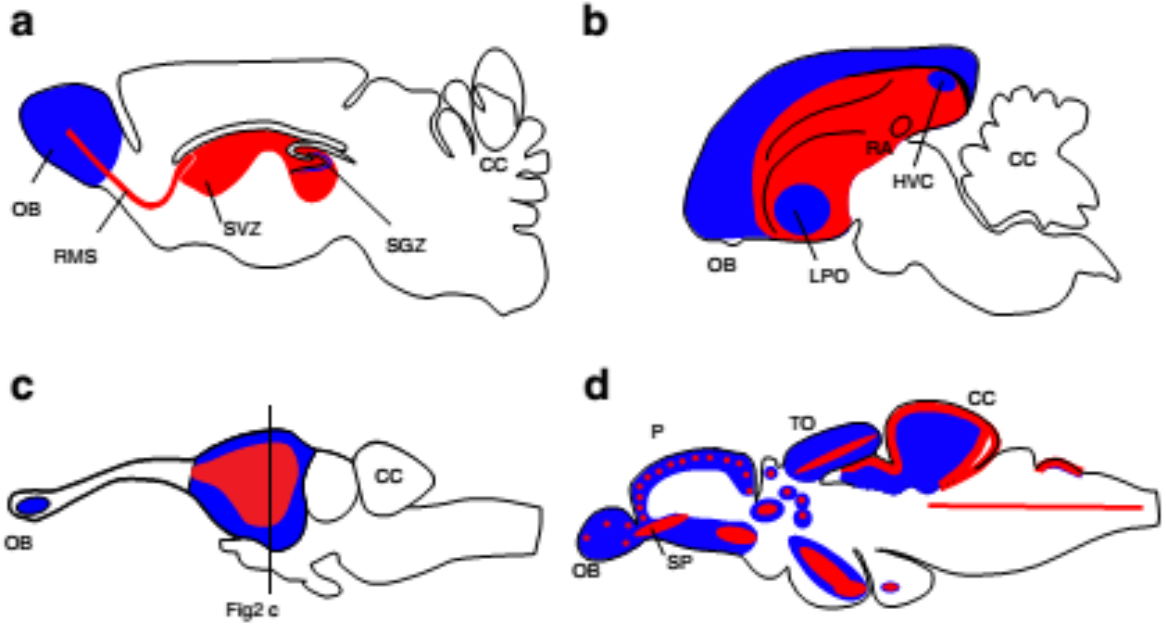


Fig. 1 Parasagittal sections through the brains of an adult **a** rodent (mouse), **b** bird (canary), **c** reptile (lizard) and **d** fish (zebrafish) indicating regions of constitutive proliferation (*red*) and neurogenesis (*blue*). Encricled area in **a** is magnified in Fig. 3a. The EGL (*red*) generates most granule neurons (*blue*). EGL is defined as the external granule layer with superficial and deep layers of dividing cells and neuroblasts, respectively. *CC* corpus cerebelli, *HVC* nucleus engaged in song learning and production, *LPO* lobus parolfactorius, *OB* olfactory bulb, *P* pallium (dorsal telencephalon), *RA* robust nucleus of the archistriatum, *RMS* rostral migratory stream, *SGZ* subgranular zone, *SP* subpallium (ventral telencephalon), *SVZ* subventricular zone, *TO* optic tectum. **c** Indicates the plain of the section shown in Fig. 2c. Images modified from Doetsch and Scharff (2001), Hatten and Heintz (1995), Kaslin et al. (2008), Northcutt (2011), Peñafiel et al. (1996), Pérez-Cañellas and García-Verdugo (1996) and Stamatakis et al. (2004)

A com

Alessandro

Constitutive neuronal progenitors
Latent neural progenitors

Fish

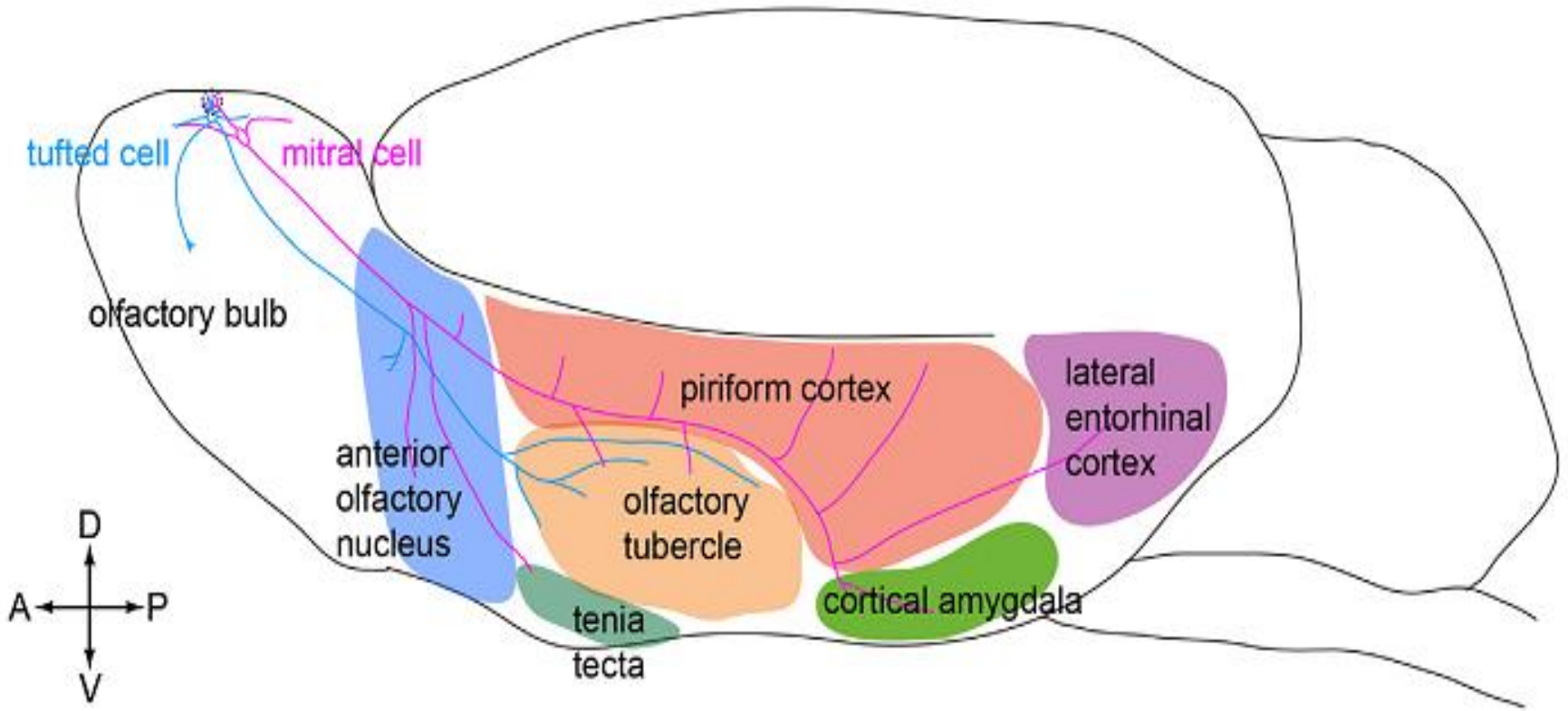
Sc	–	+	+
R	+	+	+

зони **нейроно**генезу **зрілого** мозку ссавців

- нюхова цибулина
- гіпокамп
- гіпоталамус
- стріатум

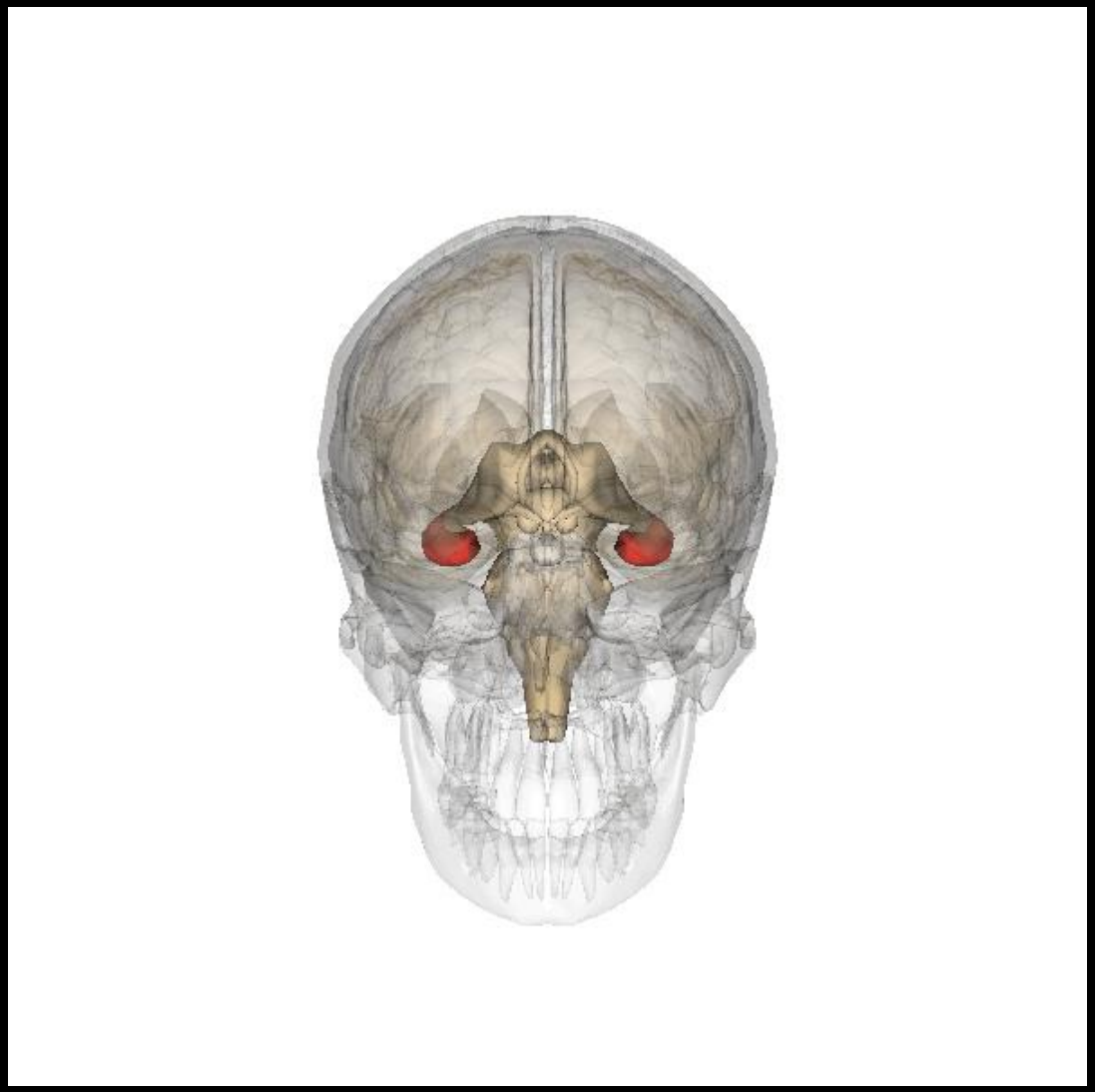
нюхова цибулина

T. Imai / Seminars in Cell & Developmental Biology 35 (2014) 180–188

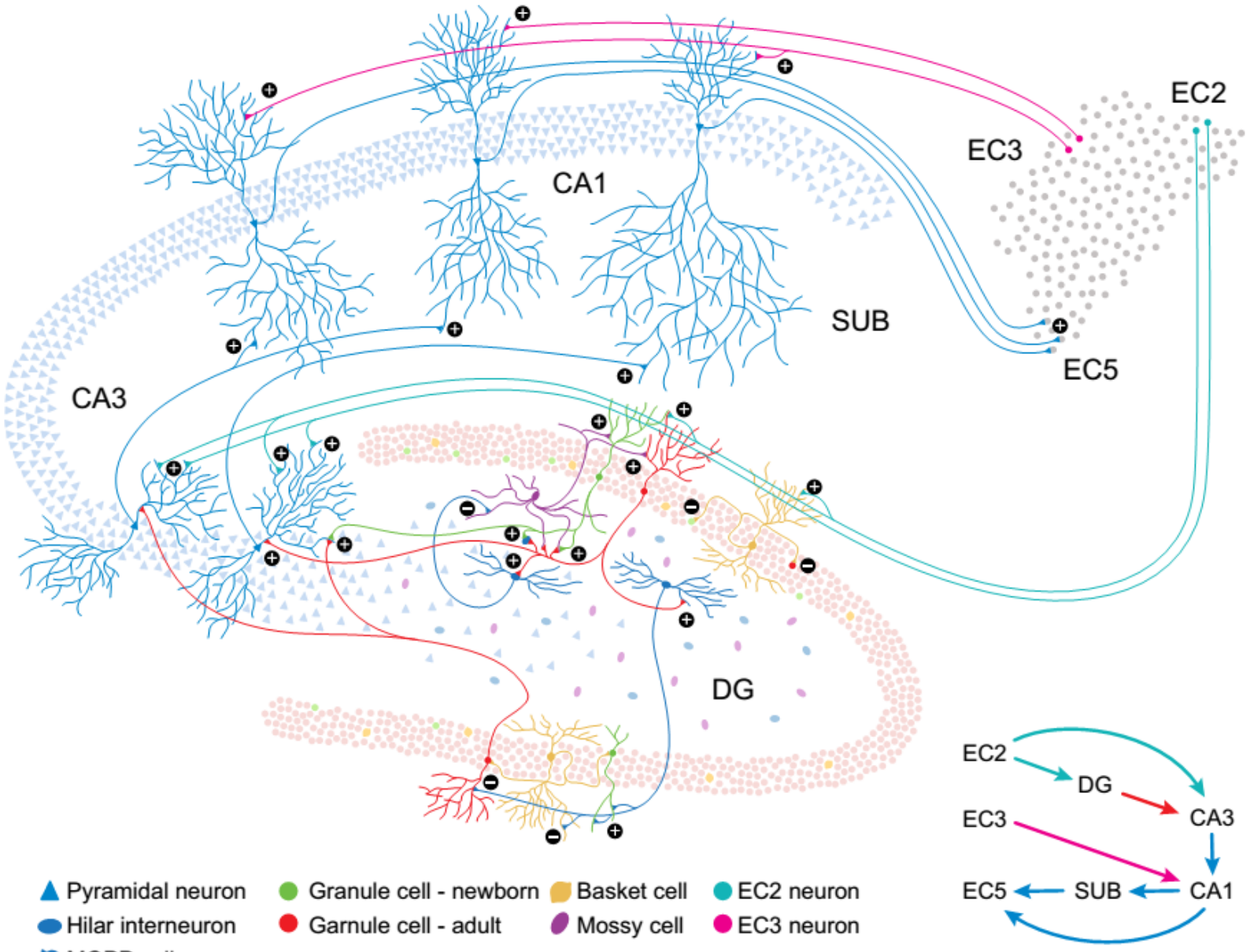


Інтенсивність нейроногенезу у гризунів: ~1 % нейронів щодня, протягом місяця половина новоутворених нейронів гине.

Утворюються ГАМК-ергічні інтернейрони НЦ , а також глутаматергічні мітральні та пучкові (*'tufted'*) клітини

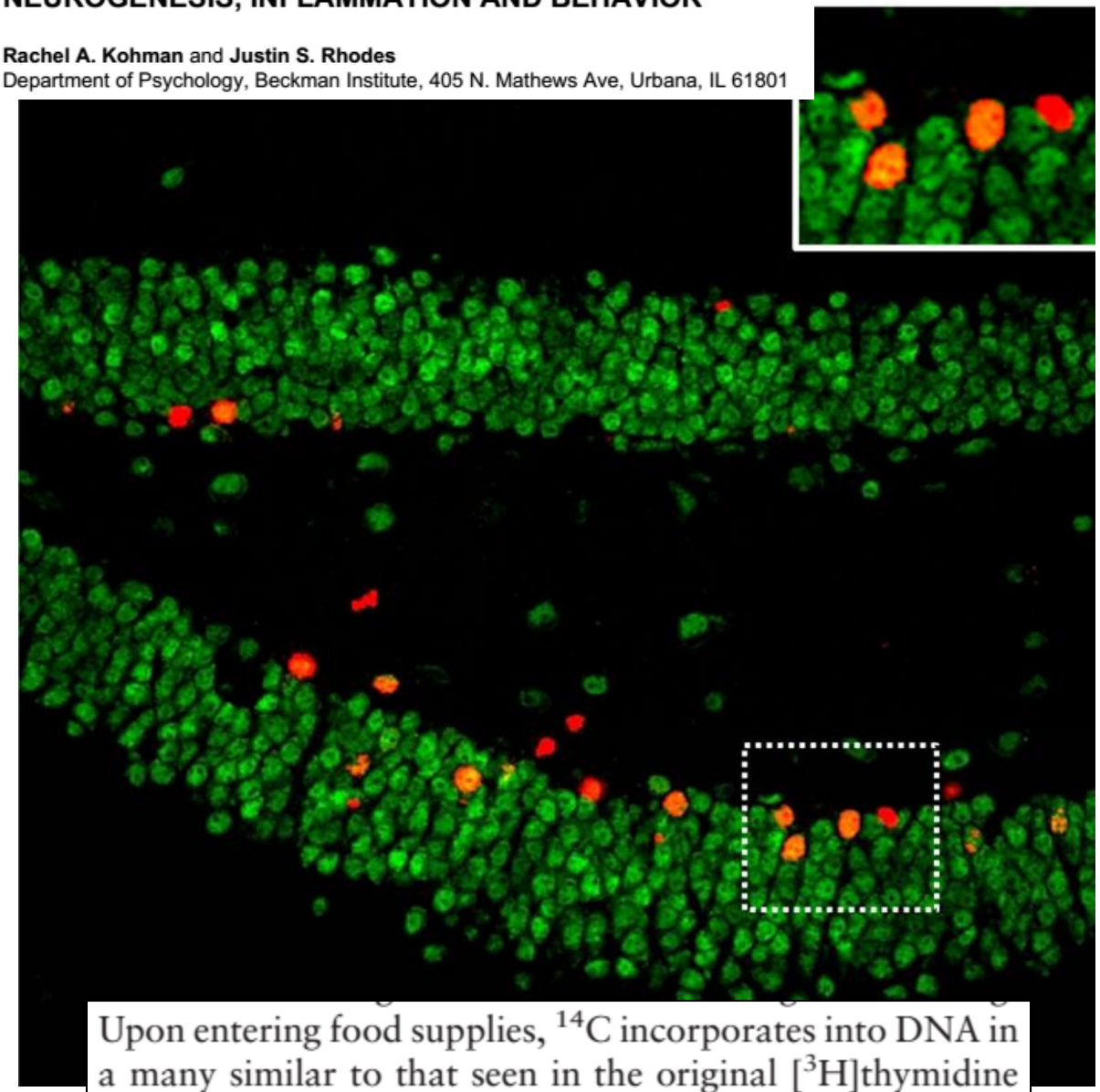


гіппокамп

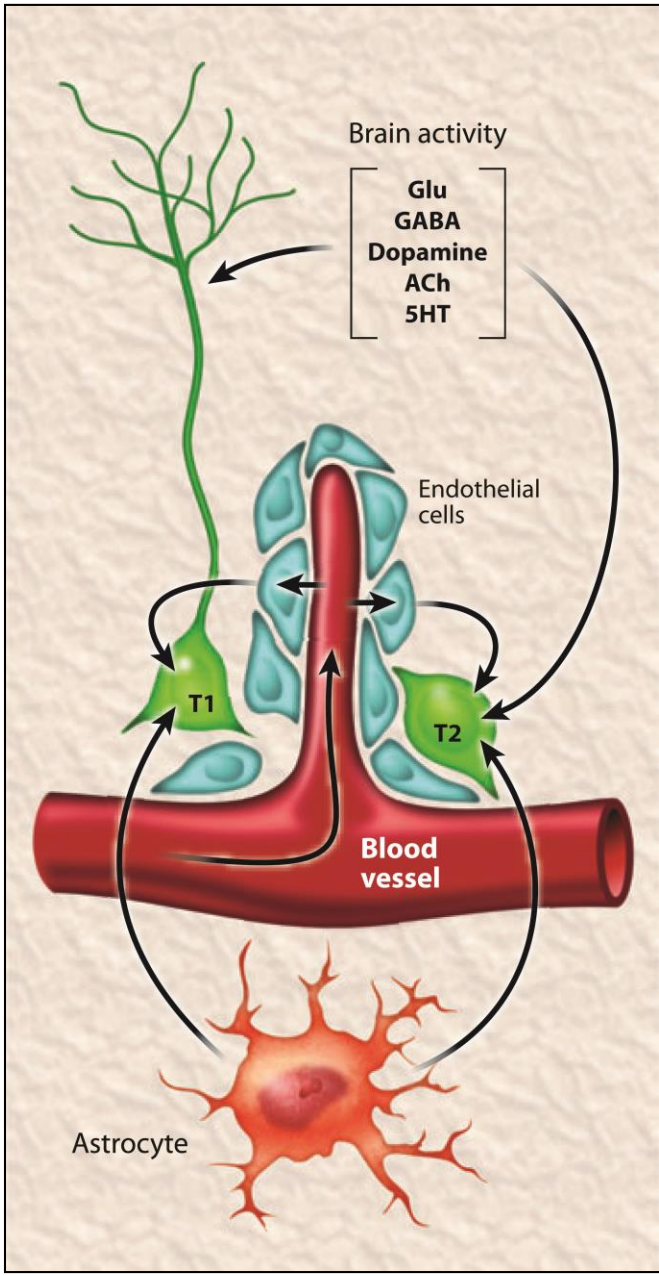

EC2
EC3
CA1
SUB
EC5
CA3
DG
EC2
DG
CA3
EC3
EC5
SUB
CA1
Pyramidal neuron
Granule cell - newborn
Basket cell
EC2 neuron
Hilar interneuron
Garnule cell - adult
Mossy cell
EC3 neuron
MOPP cell

NEUROGENESIS, INFLAMMATION AND BEHAVIOR

Rachel A. Kohman and **Justin S. Rhodes**
Department of Psychology, Beckman Institute, 405 N. Mathews Ave, Urbana, IL 61801



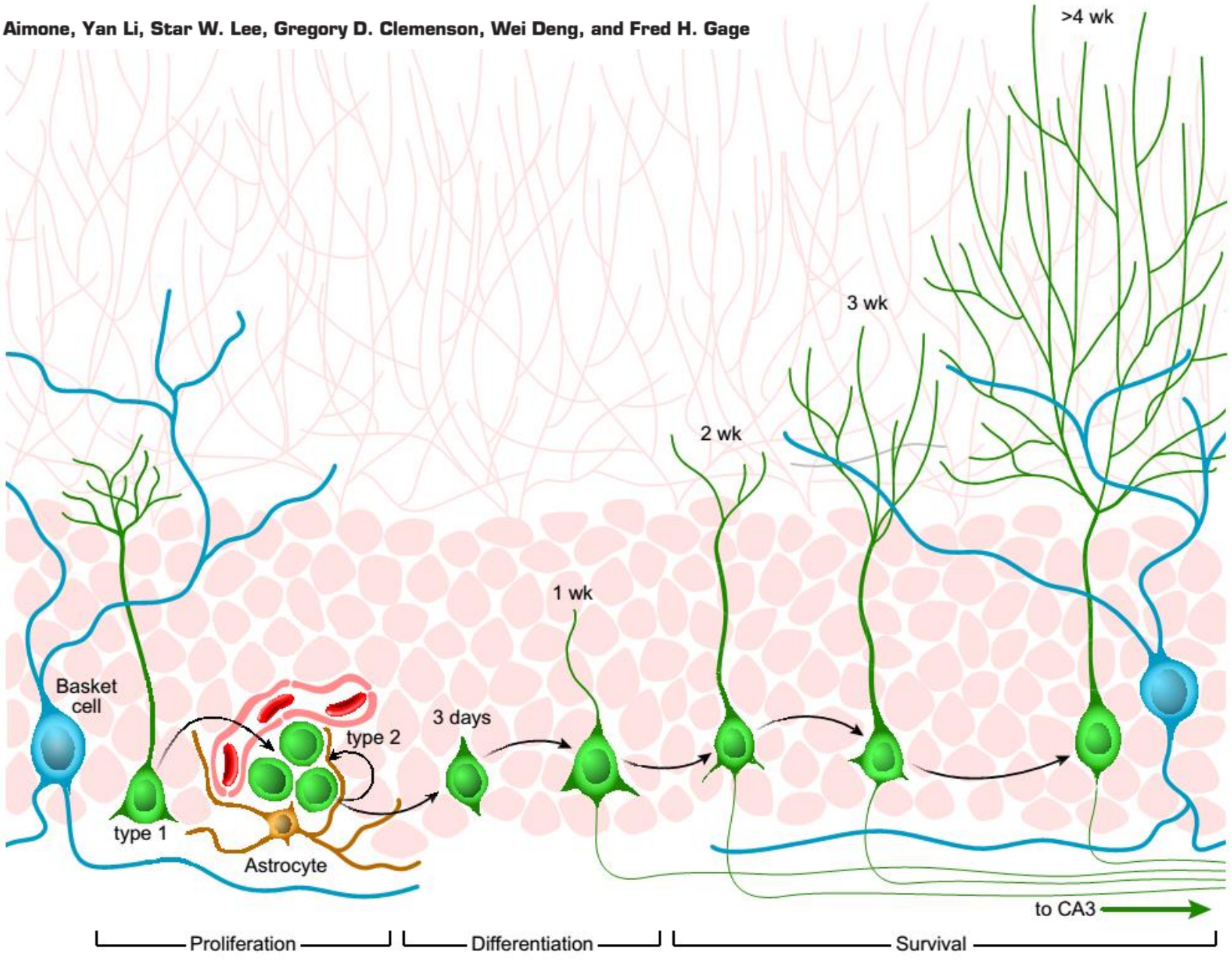
Upon entering food supplies, ^{14}C incorporates into DNA in a many similar to that seen in the original [^{3}H]thymidine studies. This technique enables estimates of overall levels of neurogenesis, showing a turnover rate of GCs at ~1.75% a year. Notably, subsequent studies with this technique did not observe new neuron turnover in the OB; however, it



H. Suh и соавт., 2009

REGULATION AND FUNCTION OF ADULT NEUROGENESIS: FROM GENES TO COGNITION

James B. Aimone, Yan Li, Star W. Lee, Gregory D. Clemenson, Wei Deng, and Fred H. Gage



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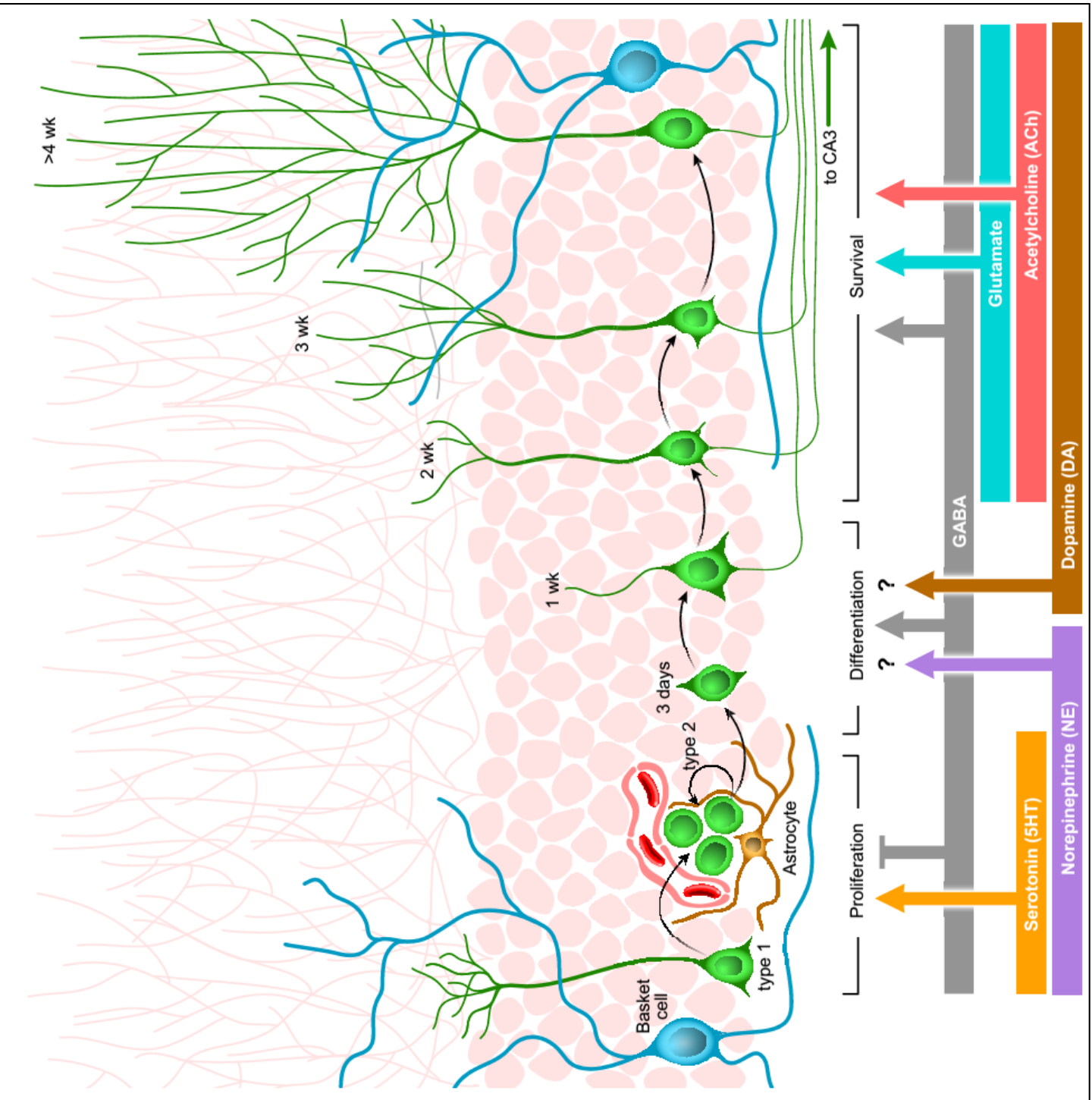
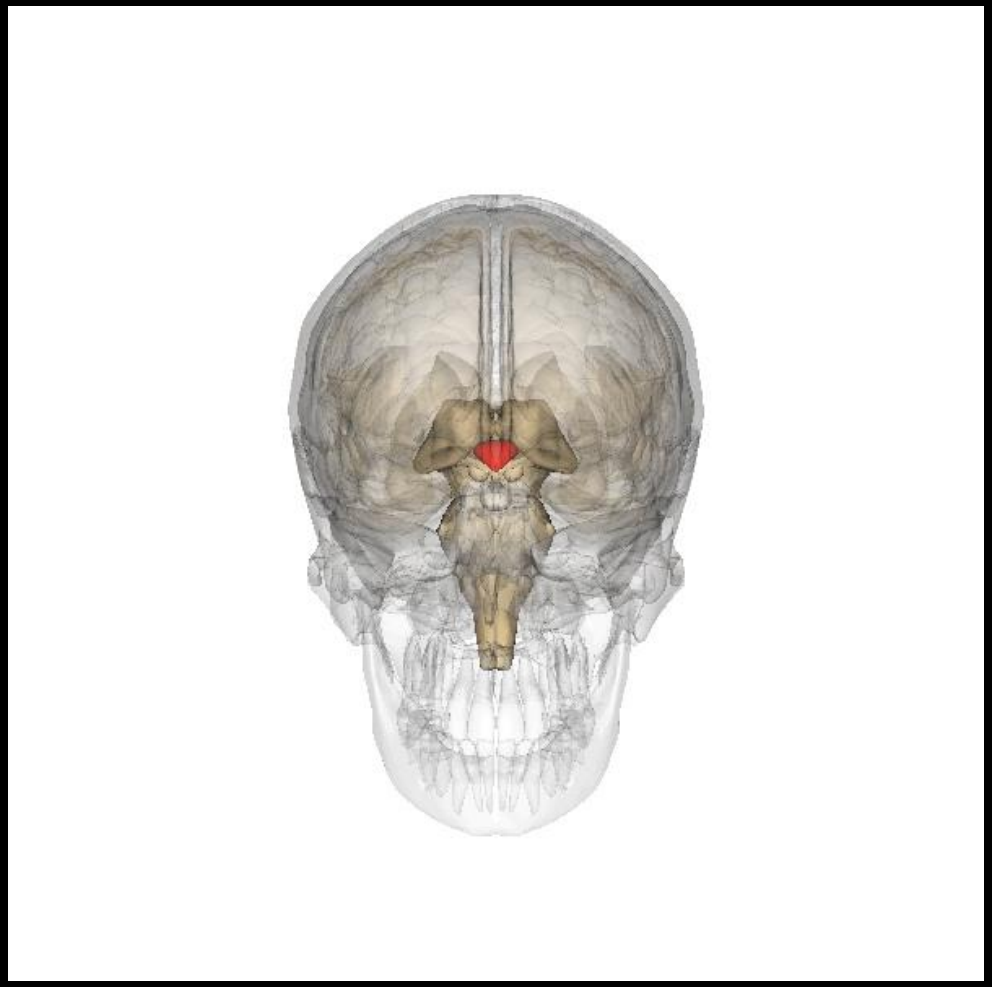




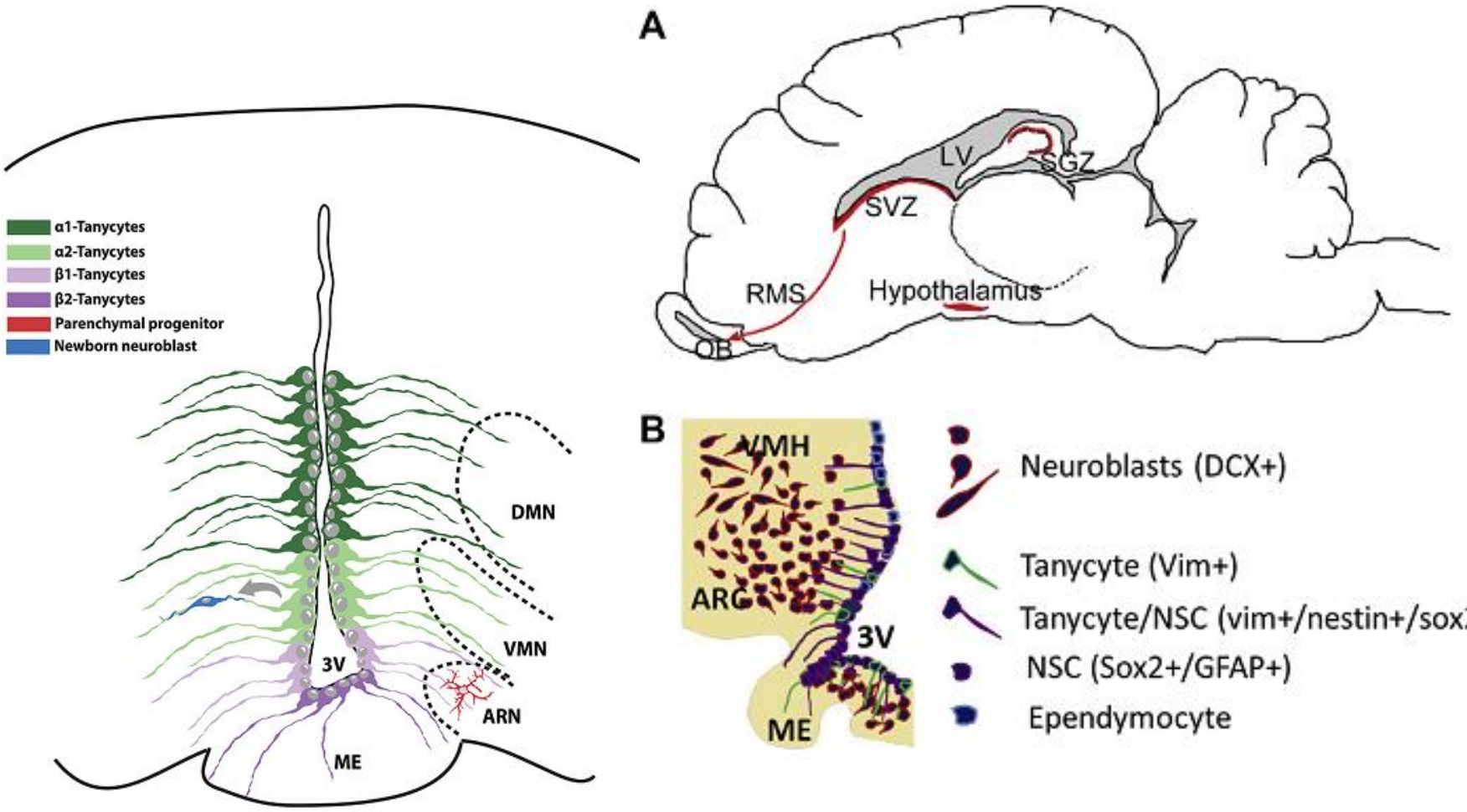
гіпоталамус

frontiers in
CELLULAR NEUROSCIENCE

MINI REVIEW ARTICLE
published: 06 January 2015
doi: 10.3389/fncel.2014.00440

Neurodevelopmental origin and adult neurogenesis of the neuroendocrine hypothalamus

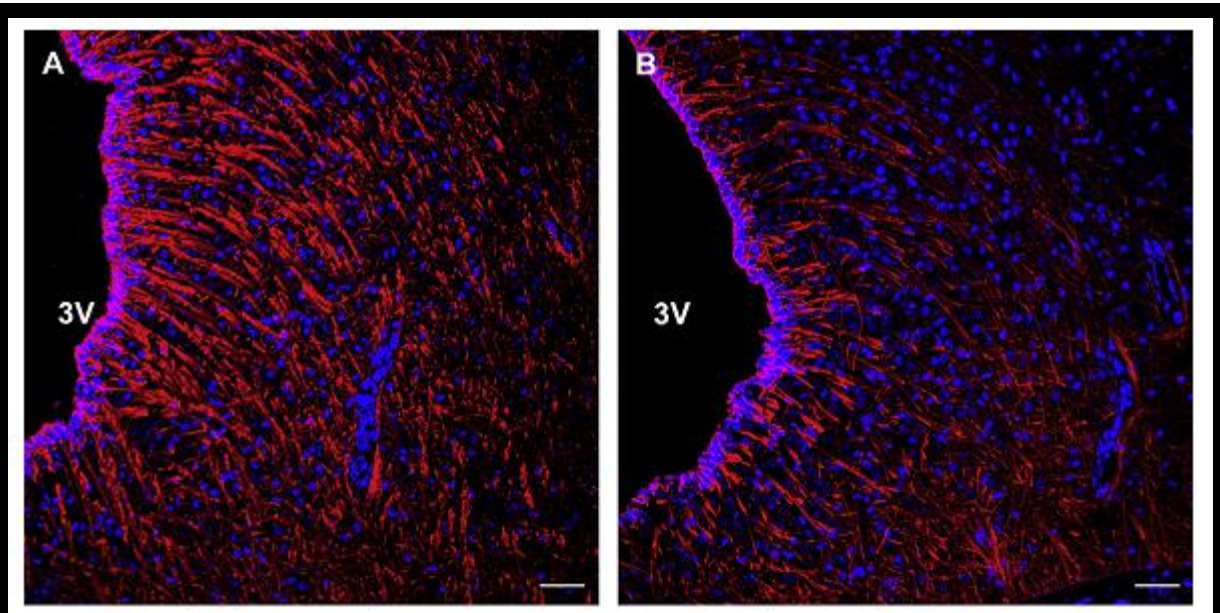
Roberto Maggi[1,2], Jacopo Zasso[3] and Luciano Conti[3]**

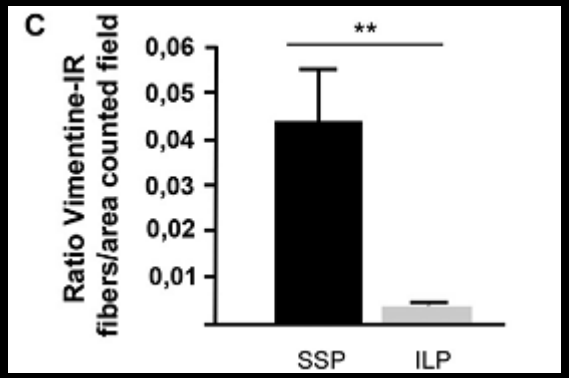


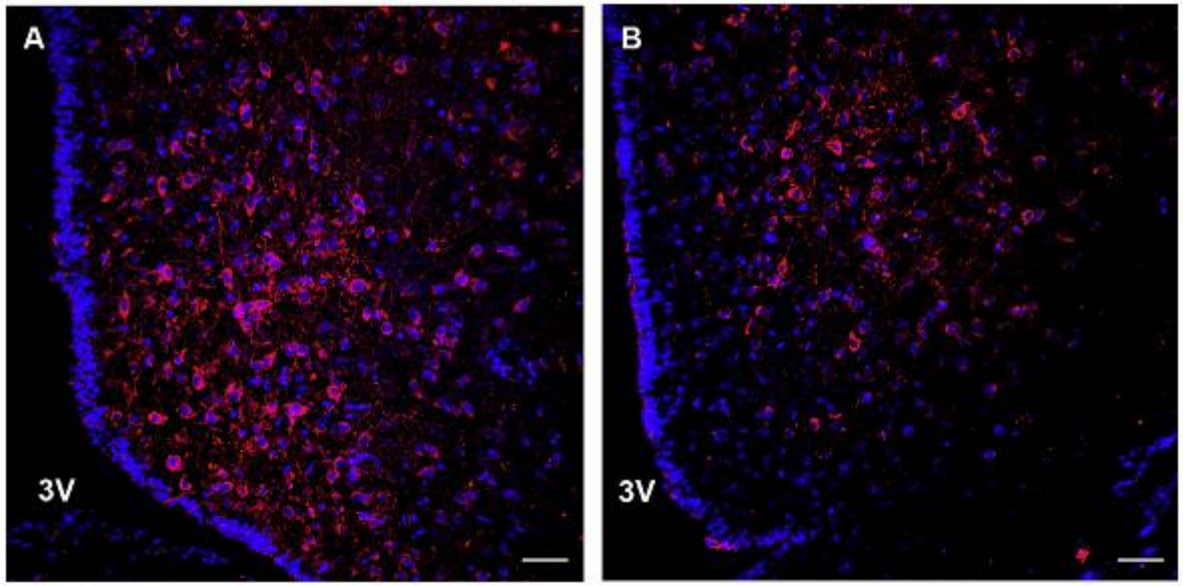
Review

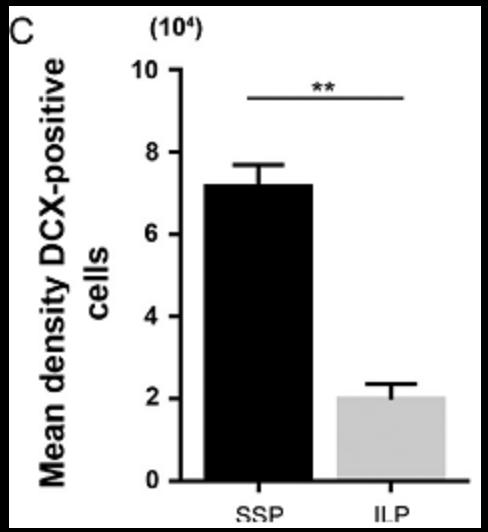
Seasonal regulation of structural plasticity and neurogenesis in the adult mammalian brain: Focus on the sheep hypothalamus

Martine Migaud *, Lucile Butrille, Martine Batailler









hermore, we determined that young neurons that
rtin (DCX) were observed in sheep hypothalamic
the ARC and ME (Batailler et al., 2014). We con-

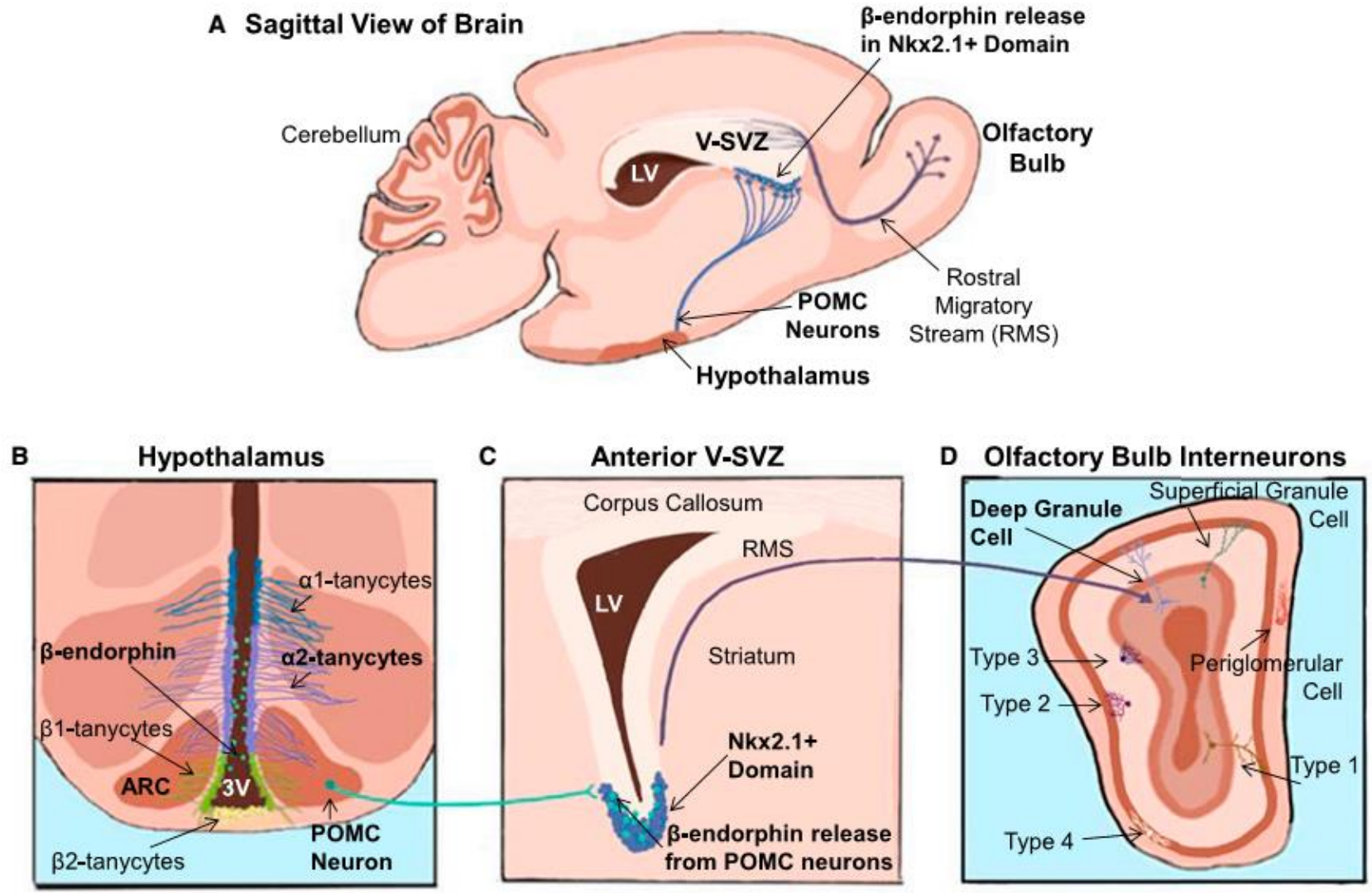
Appetite for Neurogenesis

Developmental Cell *42*, August 7, 2017

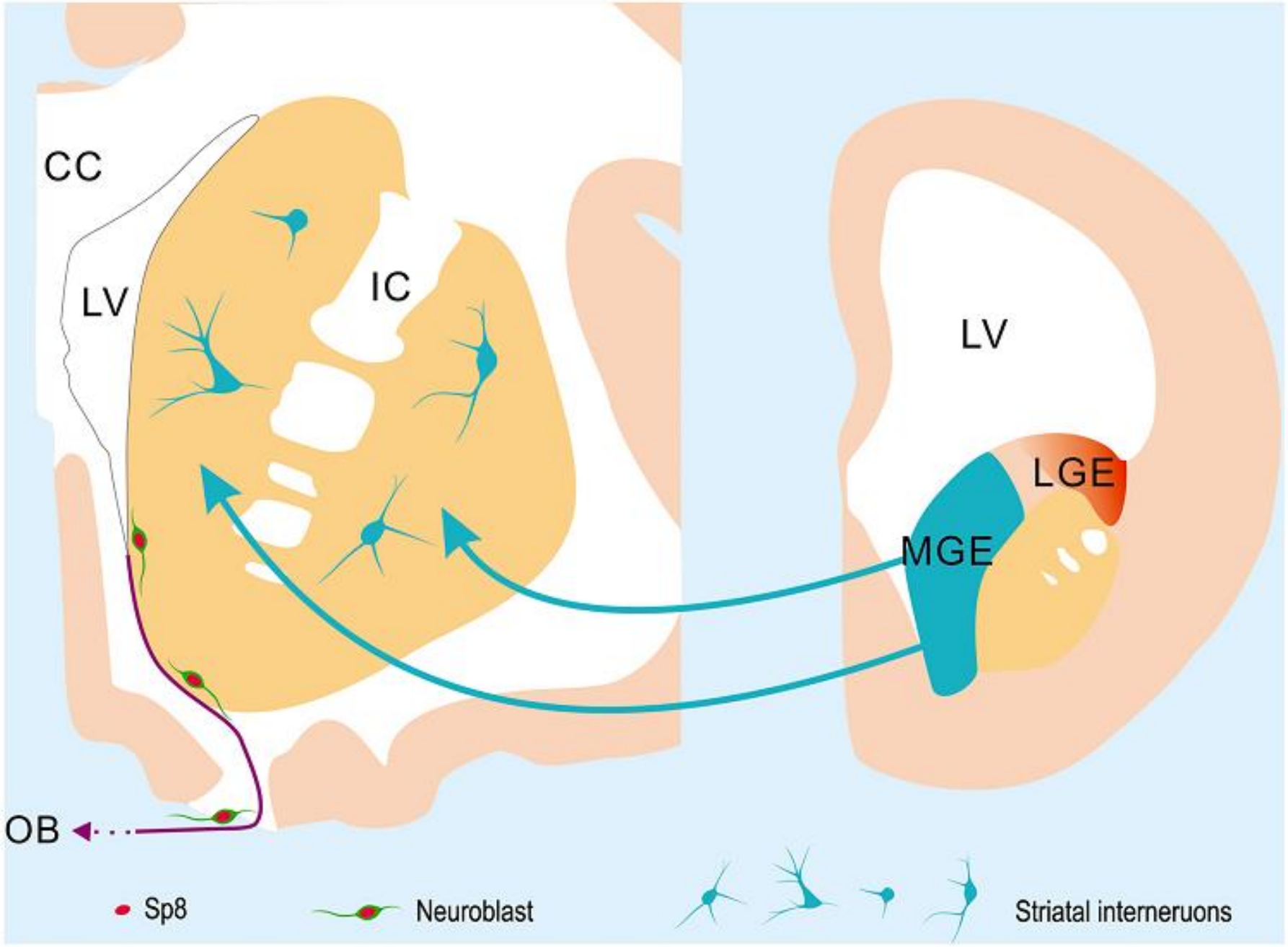
Kristen L. Zuloaga[1,*] and Sally Temple[2,*]

Hypothalamic regulation of regionally distinct adult neural stem cells and neurogenesis

Alex Paul,[1,2] Zayna Chaker,[3] Fiona Doetsch[2,3,*]



стріатум


CC
LV
IC
LV
LGE
MGE
OB
Sp8
Neuroblast
Striatal interneruons

соціогенні впливи на нейрогенез

Cell and Tissue Research
https://doi.org/10.1007/s00441-017-2735-4

REVIEW

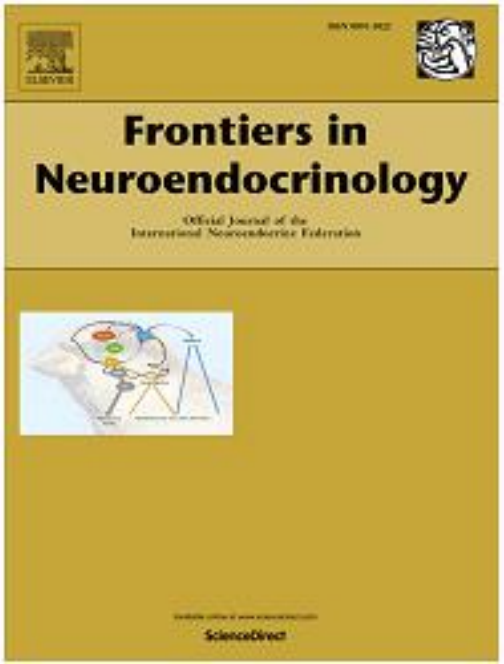
Social Behavior, Hormones and Adult Neurogenesis

Maya Opendak, Brandy A. Briones, Elizabeth Gould

Review: adult neurogenesis contributes to hippocampal plasticity

Social regulation of adult neurogenesis: A comparative approach

Melissa M. Holmes



Social manipulation	Cell proliferation	Cell survival/differentiation
Isolation	↓ male and female rats, male guinea pigs, female prairie voles, male and female marmosets	↓ male mice, male and female rats, female prairie voles, male and female marmosets
	↑ male and female naked mole-rats	
Social defeat	↓ male rats, male mice, male tree shrews, male marmosets	↓ male rats, male mice
Social status (subordination)	↓ male baboons	↓ male rats, male baboons
		↑ male and female naked mole-rats

ENESIS

al plasticity, or life stage.

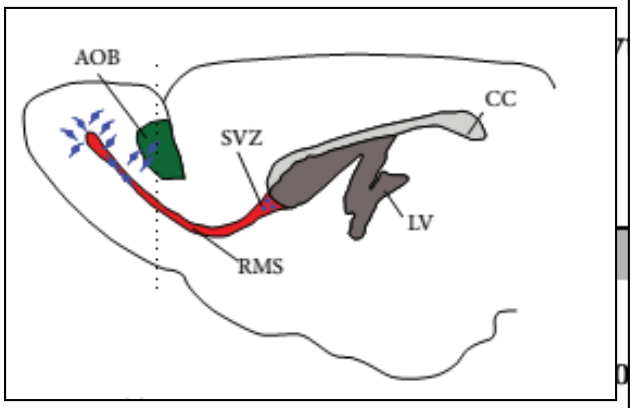
ds: basolateral amygdala, doublecortin, eusocial,
pus, neurogenesis, piriform cortex.

Review

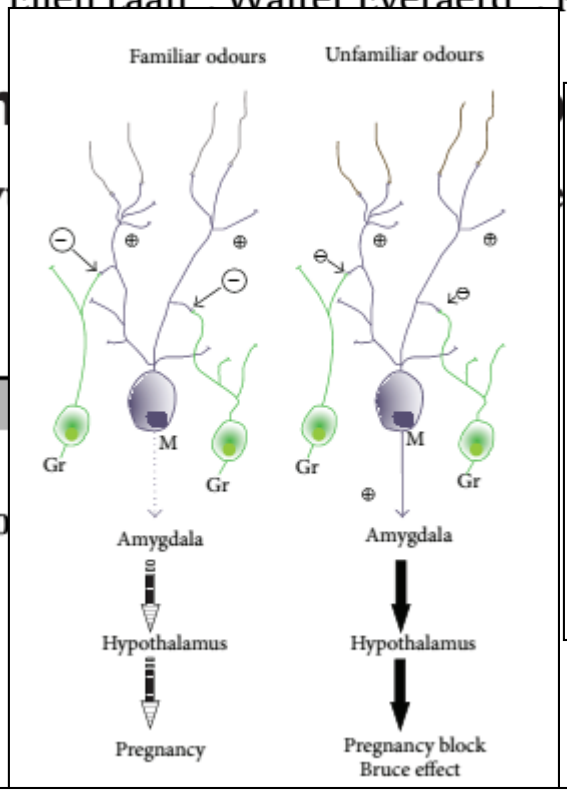
The role of conditioning, learning and dopamine in sexual behavior: A narrative review of animal and human studies

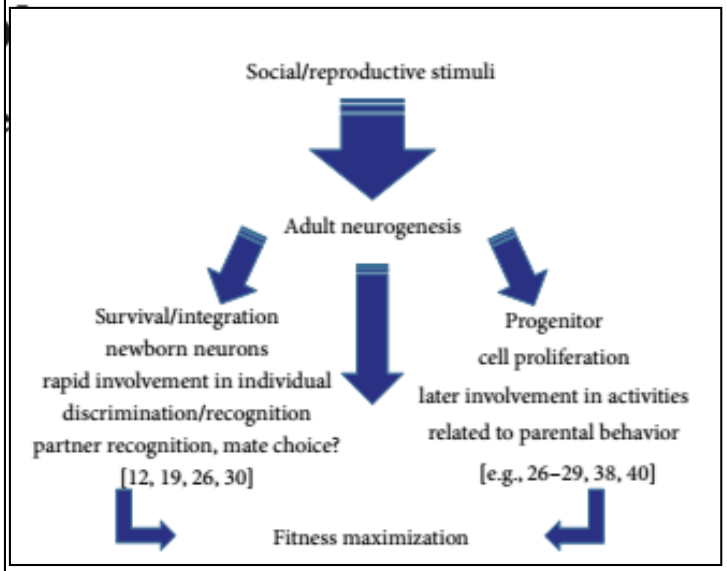
Mirte Brom[a,b,*], Stephanie Both[b], Ellen Laan[c], Walter Everaerd[d], Philip Spinhoven[a,e]

Sexual imprinting in



Heather Hoffmann

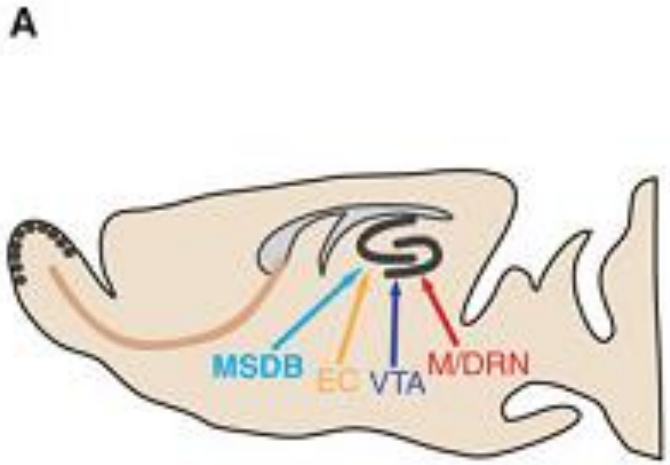


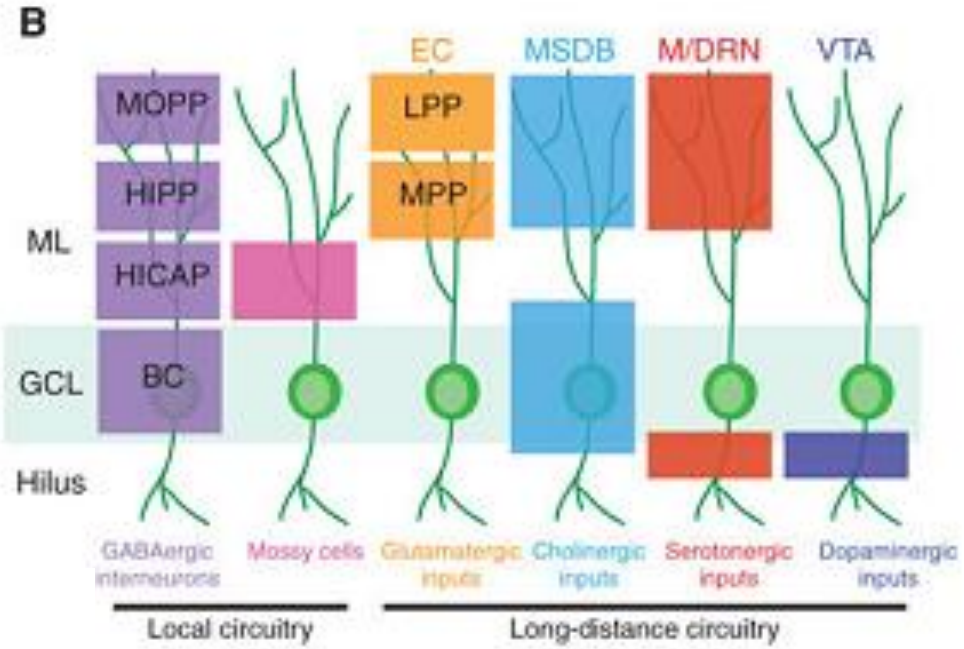


Review Article

The Interplay between Reproductive Social Stimuli and Adult Olfactory Bulb Neurogenesis

Paolo Peretto,[1,2] Roberta Schellino,[1,2] Silvia De Marchis,[1,2] and Aldo Fasolo[1,2]

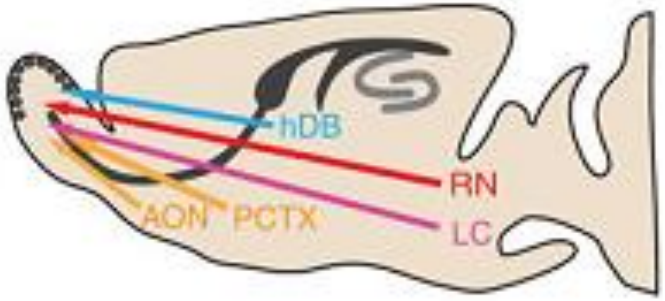


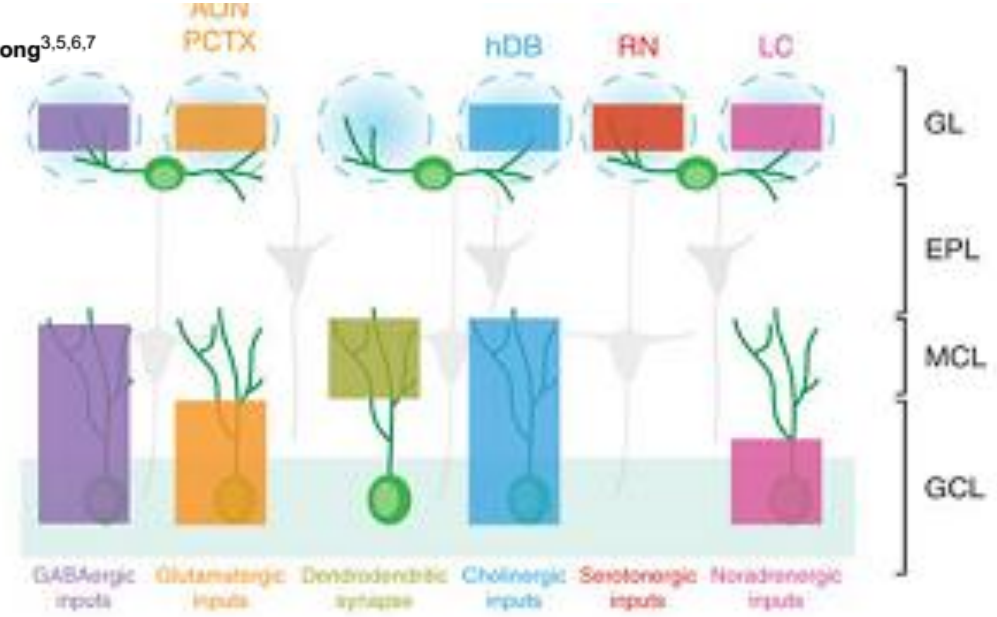


Cold Spring Harb Perspect Biol. ; 8(8): . doi:10.1101/cshperspect.a018937.

Neuronal Circuitry Mechanisms Regulating Adult Mammalian Neurogenesis

Juan Song[1,2], **Reid H.J. Olsen**[1], **Jiaqi Sun**[3,4], **Guo-li Ming**[3,5,6,7], and **Hongjun Song**[3,5,6,7]





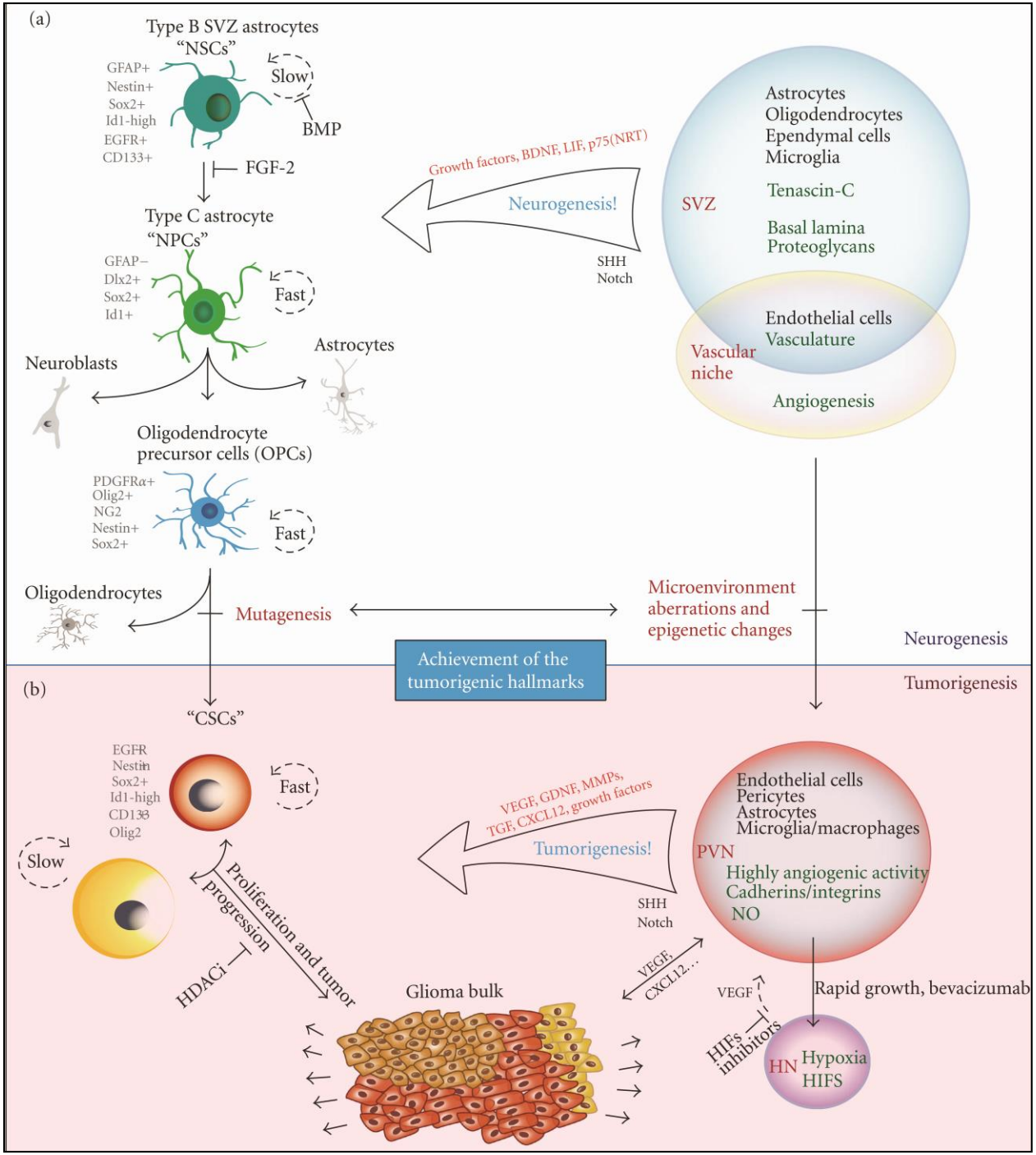
Нейрогенні стовбурові клітини – учасники патологічних процесів у мозку

1. **Епілепсія** (*патологія нейрогенезу у гіпокампі*)
2. **Пухлини головного мозку (***патологія нейрогенних стовбурових клітин мозку загалом***)**
3. **Великий депресивний розлад (***патологія нейрогенезу у гіпокампі***)**
4. **Шизофренія (***патологія нейрогенезу у нюховій цибулині і, можливо, у гіпокампі***)**

Механізми:

Накопичення мутацій, ендореплікація, копіювання та розповсюдження мутованих ділянок ДНК серед власного клітинного потомства або серед інших клітин тканини.

пухлини головного мозку


(a)
Type B SVZ astrocytes
"NSCs"
GFAP+
Nestin+
Sox2+
Id1-high
EGFR+
CD133+
Slow
BMP
FGF-2
Type C astrocyte
"NPCs"
GFAP−
Dlx2+
Sox2+
Id1+
Fast
Neuroblasts
Astrocytes
Oligodendrocyte
precursor cells (OPCs)
PDGFRα+
Olig2+
NG2
Nestin+
Sox2+
Fast
Oligodendrocytes
Mutagenesis
Growth factors, BDNF, LIF, p75(NRT)
Neurogenesis!
SHH
Notch
Astrocytes
Oligodendrocytes
Ependymal cells
Microglia
SVZ
Tenascin-C
Basal lamina
Proteoglycans
Endothelial cells
Vasculature
Vascular
niche
Angiogenesis
Microenvironment
aberrations and
epigenetic changes
Neurogenesis
Achievement of the
tumorigenic hallmarks
Tumorigenesis
(b)
"CSCs"
EGFR
Nestin
Sox2+
Id1-high
CD133
Olig2
Fast
Slow
Proliferation and tumor
progression
HDACi
VEGF, GDNF, MMPs,
TGF, CXCL12, growth factors
Tumorigenesis!
SHH
Notch
Endothelial cells
Pericytes
Astrocytes
Microglia/macrophages
PVN
Highly angiogenic activity
Cadherins/integrins
NO
VEGF,
CXCL12…
Glioma bulk
VEGF
HIFs
inhibitors
Rapid growth, bevacizumab
HN
Hypoxia
HIFS

Генів, що мутують під час гліомагенезу не менше 200 (*P.A. Northcott et al., 2012; G. Robinson et al., 2012*).

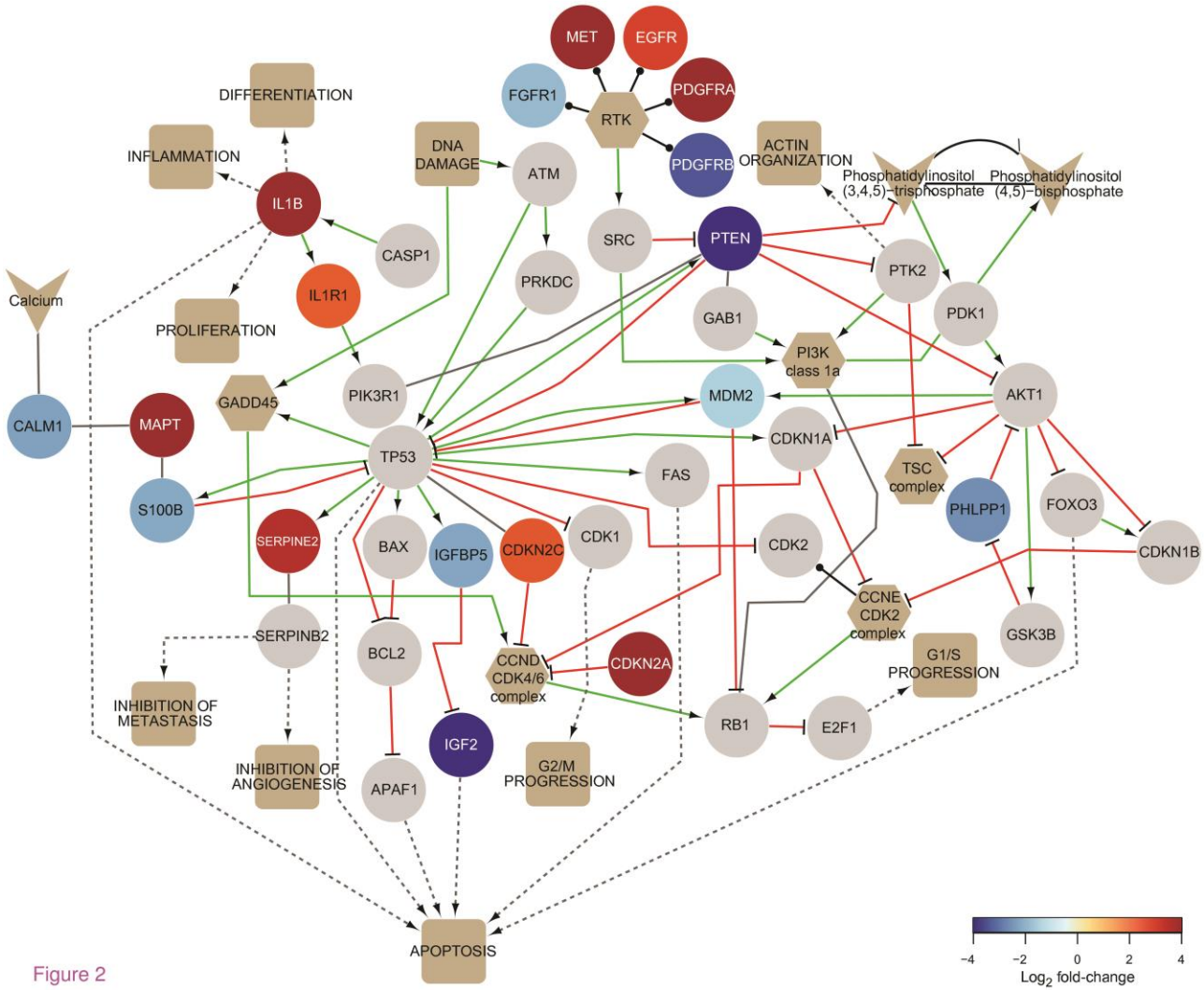
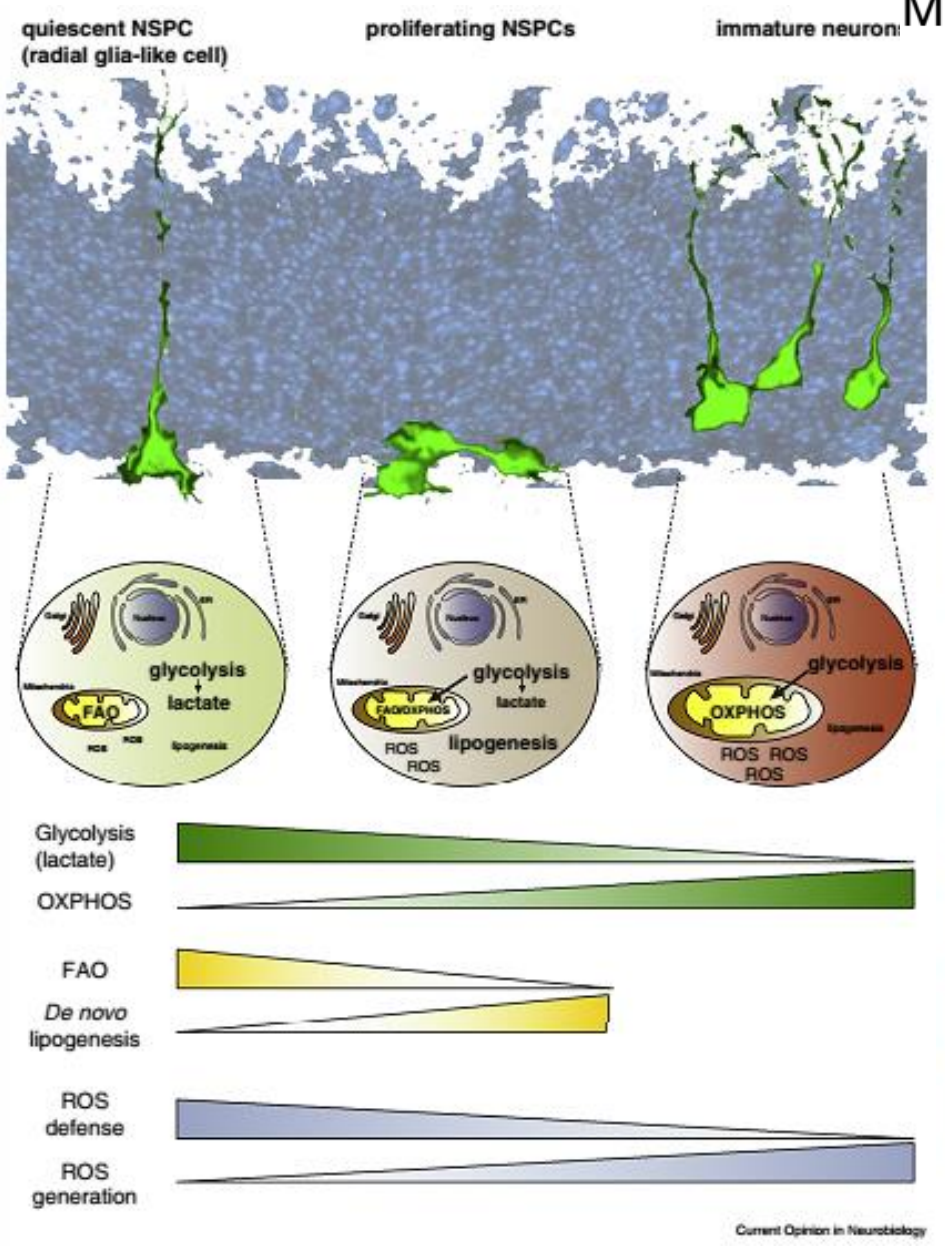
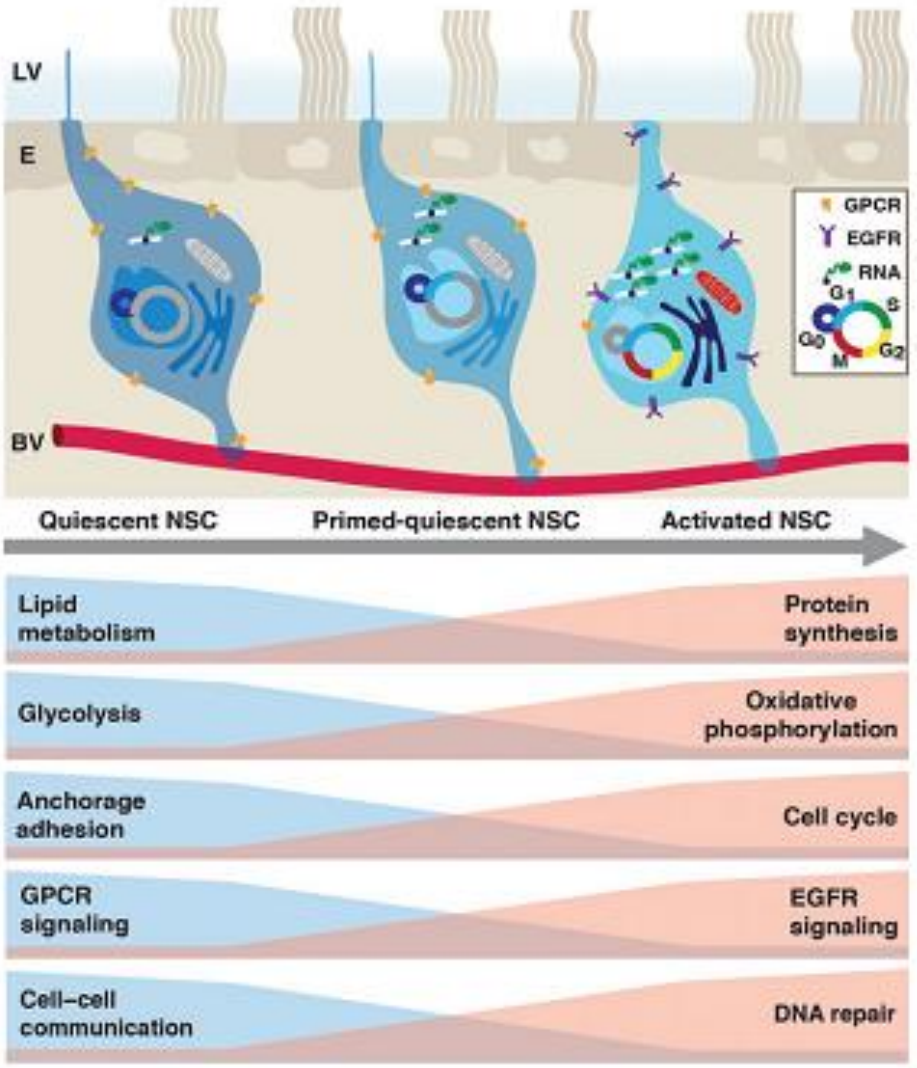


Figure 2

Metabolism and neurogenesis

Marlen Knobloch and Sebastian Jessberger





IMMUNOCYTOCHEMICAL LOCALIZATION OF DNA DOUBLE-STRAND BREAKS IN HUMAN AND RAT BRAINS

G. TORRES,* J. R. LEHESTE AND R. L. RAMOS

Department of Biomedical Sciences, New York Institute of Technology, College of Osteopathic Medicine, Old Westbury, NY 11568, USA

Abstract—Post-mitotic neurons are particularly susceptible to DNA double-strand breaks during their relatively long lifespan. Here, we report the anatomical distribution and subcellular localization of a molecule first identified as a DNA damage checkpoint protein. Immunocytochemical analysis of 53BP1 showed that this nuclear molecule is widely expressed in adult human and rat brains. Further, we showed that 53BP1 routinely co-clusters with γ-amino-butyric acid neurons throughout the rat neuraxis. Notably, 53BP1 is only expressed in neuronal cells as the DNA damage checkpoint protein was virtually absent from glial cells. Finally, we found that human neural progenitors showed a differential index of DNA fragmentation at different stages of cellular differentiation. These data provide additional and important anatomical findings for the distribution and phenotype of DNA double-strand breaks in the mammalian brain, and suggest that DNA fragmentation is a spontaneous event routinely occurring in neural progenitors and adult neurons.

lesions and maintain genome stability (Santivasi and Xia, 2013). One of these repairing pathways is the so-called non-homologous end-joining (NHEJ) pathway which repairs helix-distorting lesions such as those induced by ultraviolet radiation (McKinnon, 2013). If NHEJ is disrupted during neurogenesis, neurodevelopmental defects arise in the form of microcephaly and/or high-grade gliomas (Gilmore and Walsh, 2013). Another repairing pathway operating in the nervous system is ATM (ataxia telangiectasia, mutated) which responds to DNA damage-responsive kinases. Of interest, disruption of ATM may lead to neurodegenerative disorders causing widespread synaptic signal dysfunction throughout the brain (Suberbielle et al., 2013). Indeed, the importance of the aforementioned repair pathways is underscored by the consequences of their failure to respond to DNA fragmentation in a number of disorders with neurodegenerative phenotypes, including Xeroderma Pigmentosum and the Cockayne syndrome (Jeppesen et al., 2011; McKinnon, 2013).

To fully appreciate the biological contributions of DNA double-strand breaks in the mammalian brain, we must first expand our knowledge of the anatomical distribution of such a DNA damage signaling focus within the adult human nervous system and to compare whether its expression and distribution pattern is phenotypically

психічна патологія

Five criteria for accepting the neurogenic theory of depression and anxiety

Criteria	Evidence
1. AHN is altered in depression or anxiety.	Rodent • Diverse rodent models of depression and anxiety impair AHN. Non-human primate • Social stress impairs AHN. Human • No direct measurements of AHN in depression or anxiety. • Postmortem studies show inconsistent effects on progenitor cells. • Postmortem studies show decreased dentate gyrus size and granule cell number. • MRI studies show decreased dentate gyrus size.
2. Impaired AHN is sufficient to induce depression or anxiety.	Rodent • Ablation of AHN does not increase baseline depression or anxiety-like behaviors in most studies, but it may potentiate these behaviors after acute stress. • Ablation of AHN impairs pattern separation and cognitive flexibility: cognitive functions relevant to depression and anxiety. Non-human primate • Not studied. Human • No direct studies. • Cancer treatments that ablate AHN increase depression and anxiety.

THE CURRENT STATE OF THE NEUROGENIC THEORY OF DEPRESSION AND ANXIETY

Bradley R. Miller, MD, PhD[1] and **René Hen, PhD**[2,*]

nesis and psychiatric

ult human hippocampal
hought that neurogenesis
ortant in memory forma-
nctional neurogenic pat-
nd psychiatric disorders.
mination of hippocampal
it was found that there is
f proliferating cells in the
depression contribute to
epressant treatment has
ippocampal neurogenesis
ll clarify the connections
atric disorders, and may
d neurogenesis therapies

an V–SVZ is increasingly
in the development of
Recent attention has been
in schizophrenia, where
o contribute to the pathol-
al., 2010). Several reports
t neurogenesis in the V–
chizophrenia by targeting
et al., 2012). Given the
s from the V–SVZ migrate
lausible to speculate that
rations in V–SVZ neurogenic activity may contribute to the
hology of schizophrenia in humans. A more thorough
derstanding of V–SVZ neurogenesis and the migratory
acity of progenitors to the striatum in humans may be
pful in harnessing the regenerative capacity of NSCs to
at neurodegenerative and psychiatric diseases in humans.

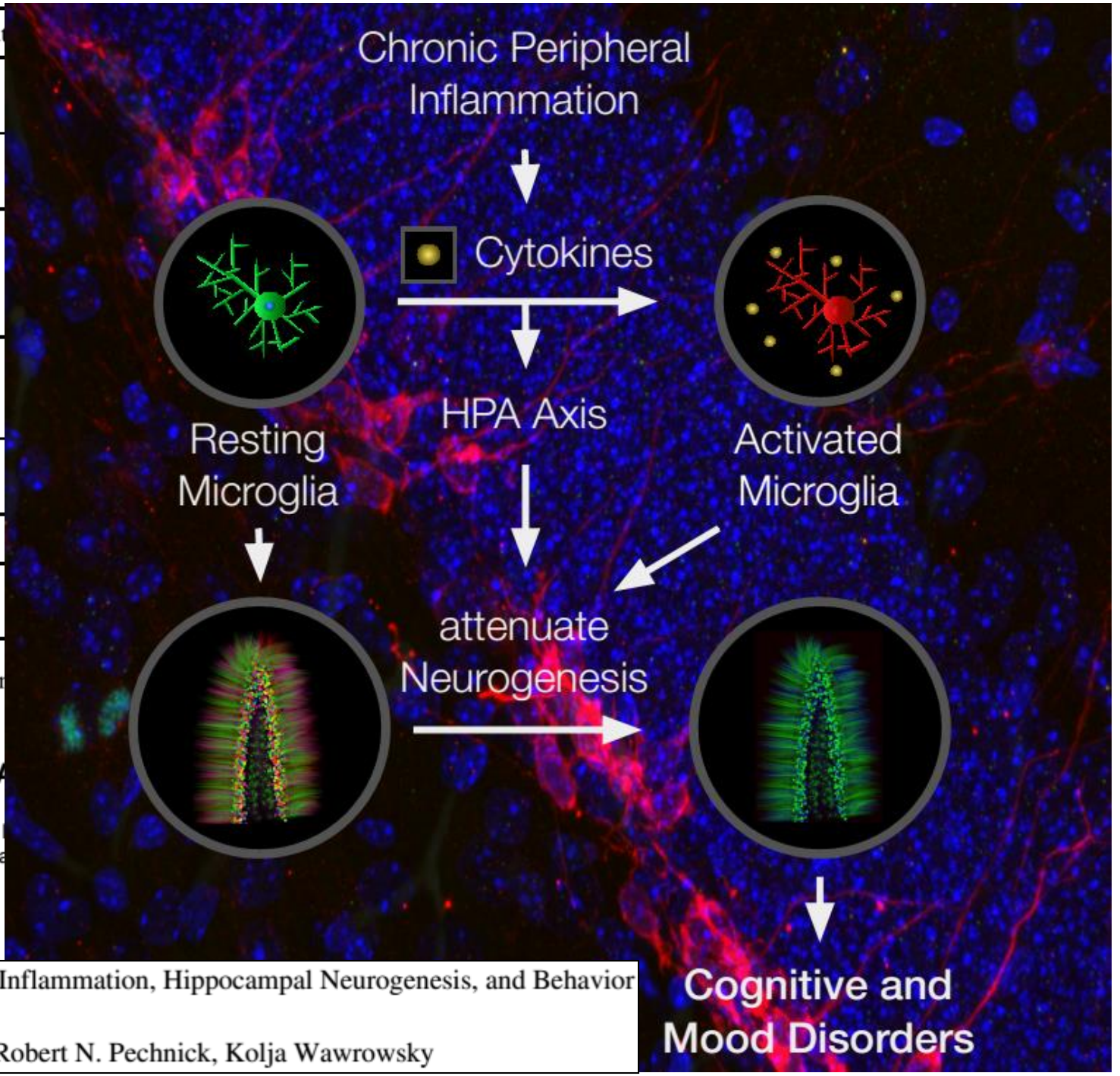
периферичні впливи

	Proliferat
IL-6	↓
IL-1beta	↓
TNF-alpha	↓
Prostaglandins	
TGF-beta	
IL-4	
IL-10	↑

Note: ↓ indicates a reduction ar

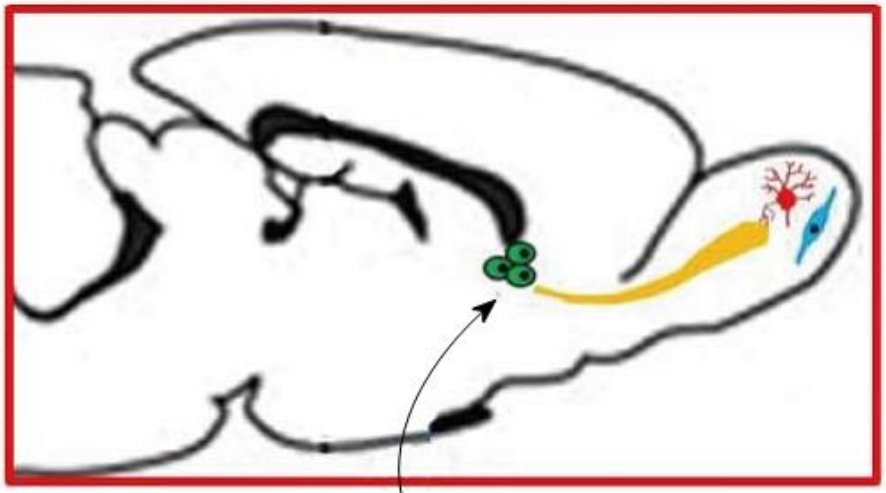
NEUROGENESIS, INFLA

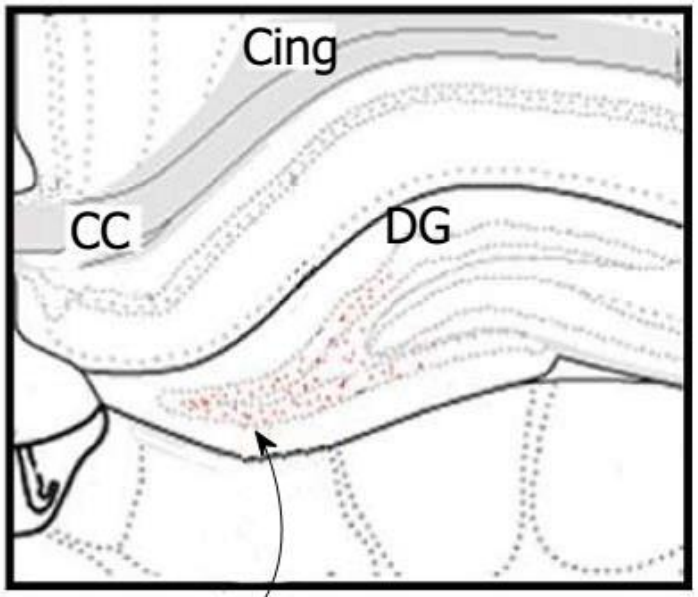
Rachel A. Kohman and **Justin S.**
Department of Psychology, Beckma



Chronic Peripheral Inflammation, Hippocampal Neurogenesis, and Behavior

Vera Chesnokova, Robert N. Pechnick, Kolja Wawrowsky

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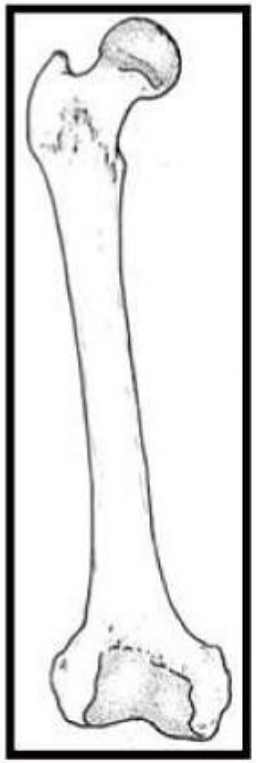
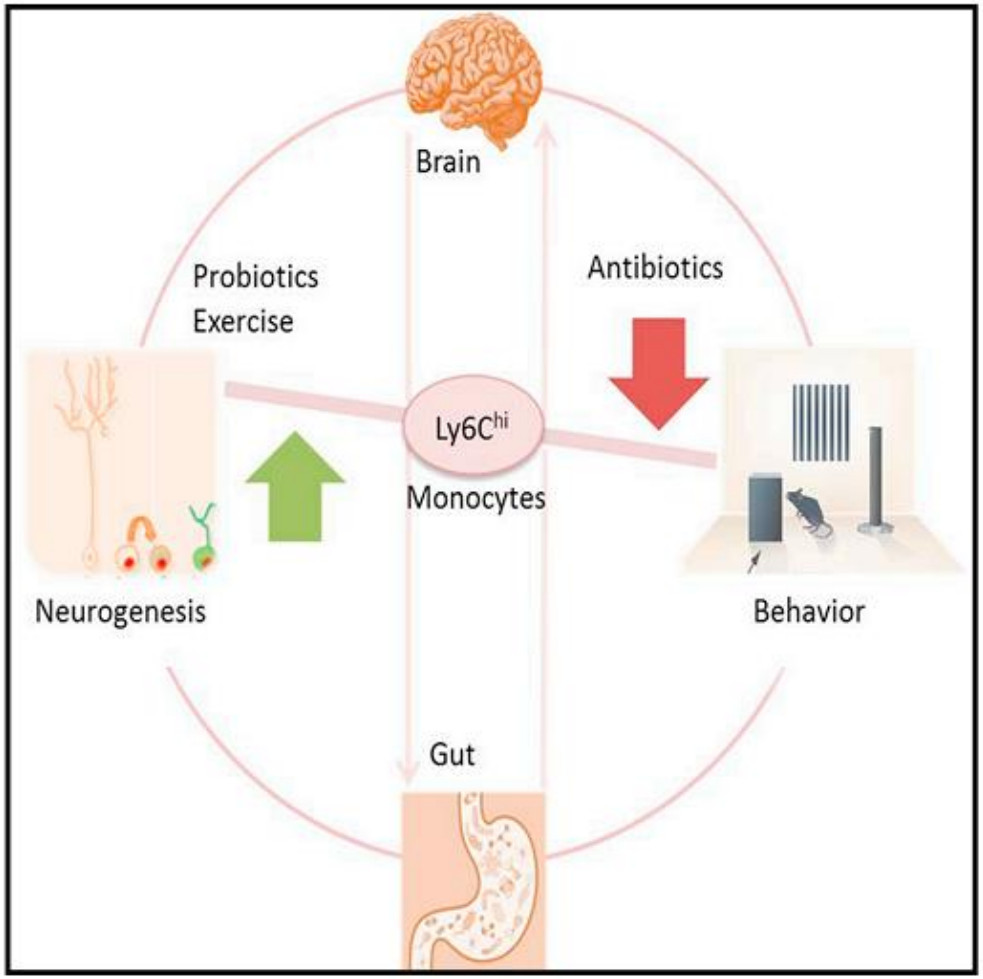


Cell Reports

Ly6C^{hi} Monocytes Provide a Link between Antibiotic-Induced Changes in Gut Microbiota and Adult Hippocampal Neurogenesis

Graphical Abstract



Authors

Luisa Möhle, Daniele Mattei, Markus M. Heimesaat, ..., Polly Matzinger, Ildiko R. Dunay, Susanne A. Wolf

Correspondence

susanne.wolf@mdc-berlin.de

In Brief

Möhle et al. show the impact of prolonged antibiotic treatment on brain cell plasticity and cognitive function. They were able to rescue the decrease in neurogenesis by probiotic treatment, physical exercise, or transfer of Ly6C^{pos} monocytes. They propose that the Ly6C^{hi} population is crucial for brain homeostasis and plasticity.

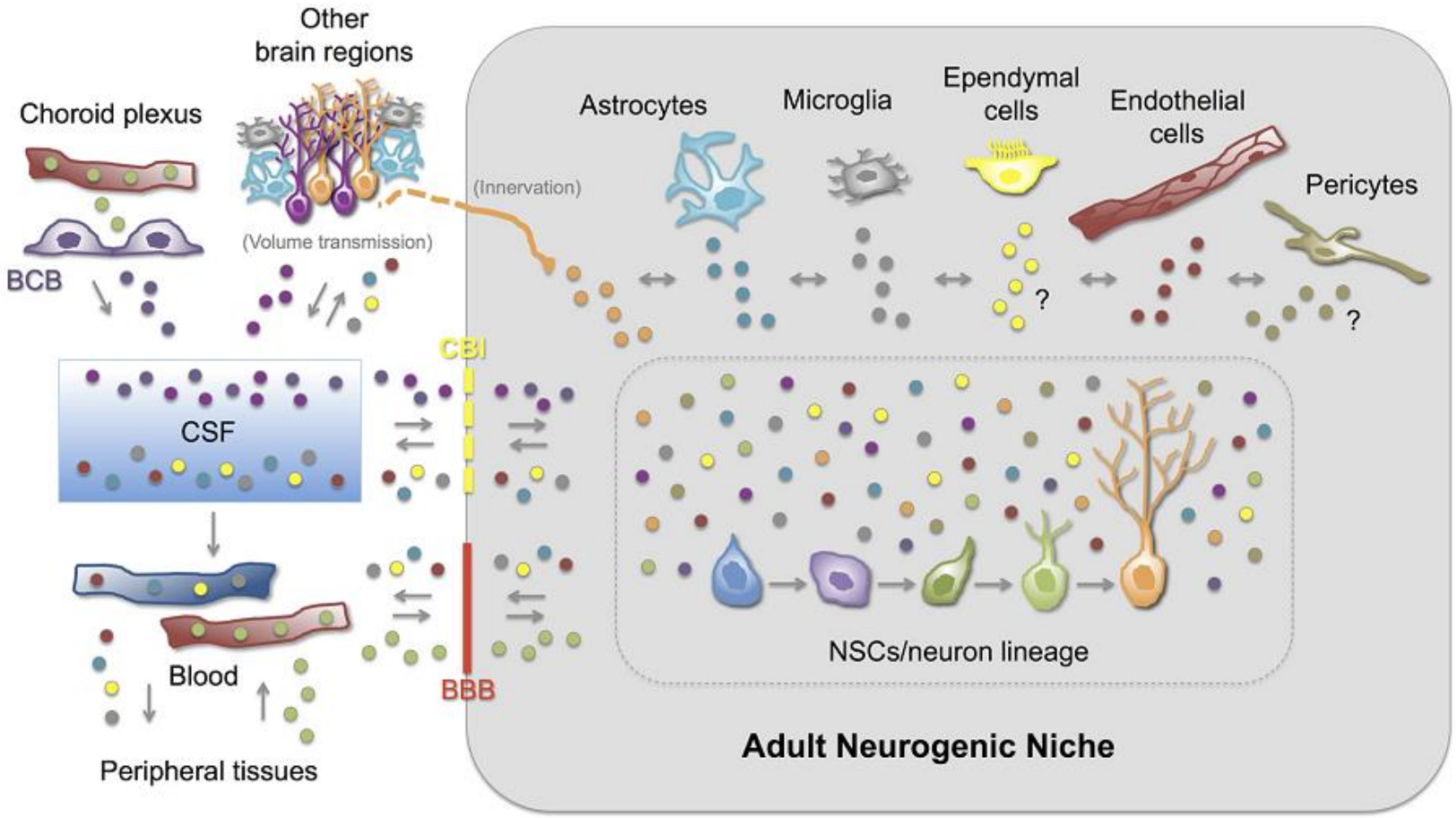


FIGURE 4 | **Exosomes as regulators of adult neurogenesis**. The NSC-neuron lineage is exposed to a complex mix of exosomes within the neurogenic niche.

Exosomes as Novel Regulators of Adult Neurogenic Niches

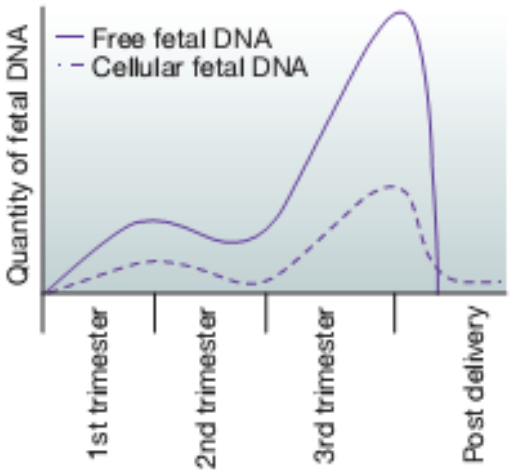
Luis Federico Bátiz[1,2,3], Maite A. Castro[1,2,4], Patricia V. Burgos[1,2,5], Zahady D. Velásquez[1,3], Rosa I. Muñoz[1,3], Carlos A. Lafourcade[6], Paulina Troncoso-Escudero[1,4] and Ursula Wyneken[6]**

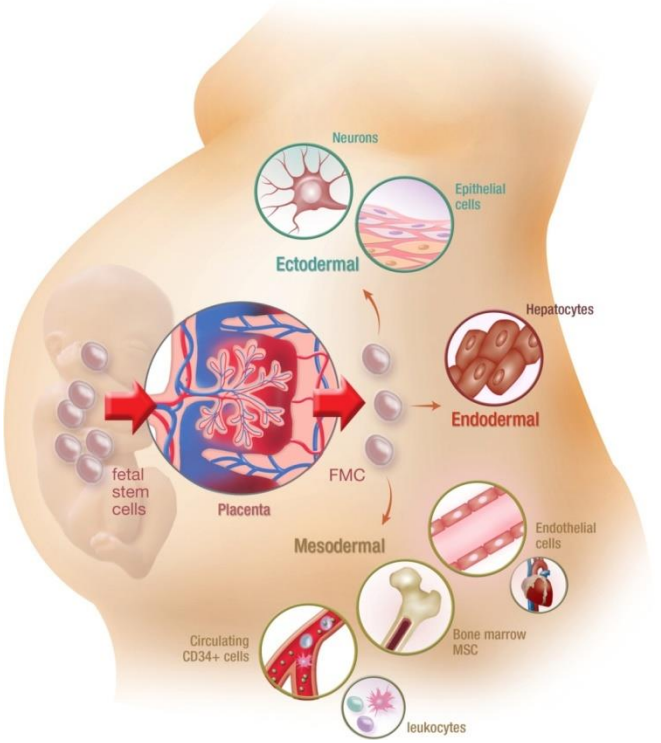
трансфер геномів

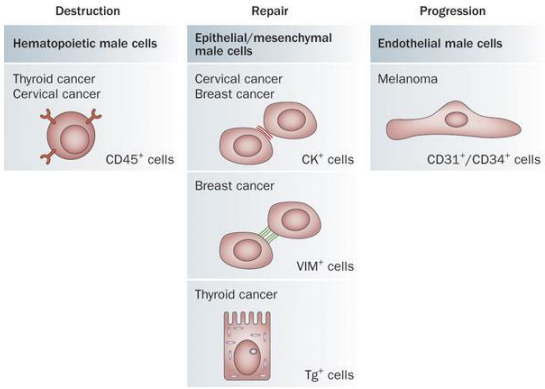
Fetal microchimerism: the cellular and immunological legacy of pregnancy

David M. Lissauer[1], Karen P. Piper[2], Paul A.H. Moss[2] and Mark D. Kilby[1,*]

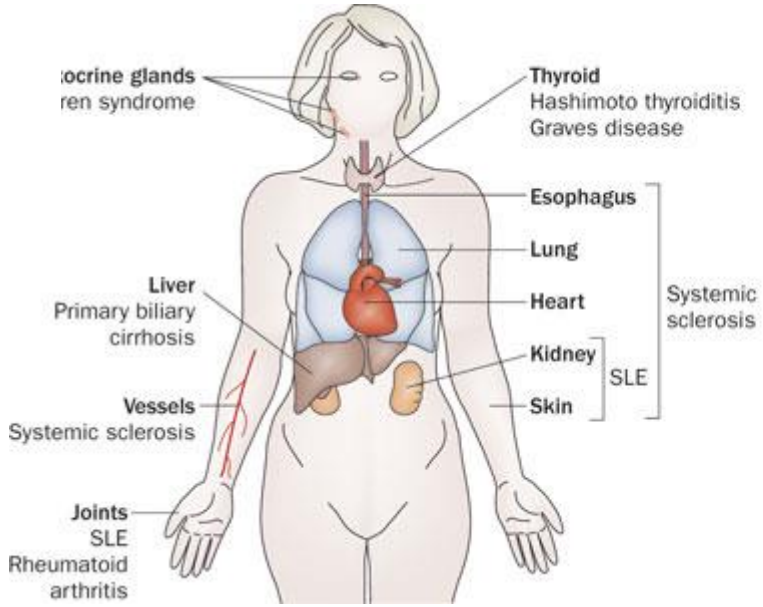
Vol. 11; e33; November 2009
expert reviews in molecular medicine

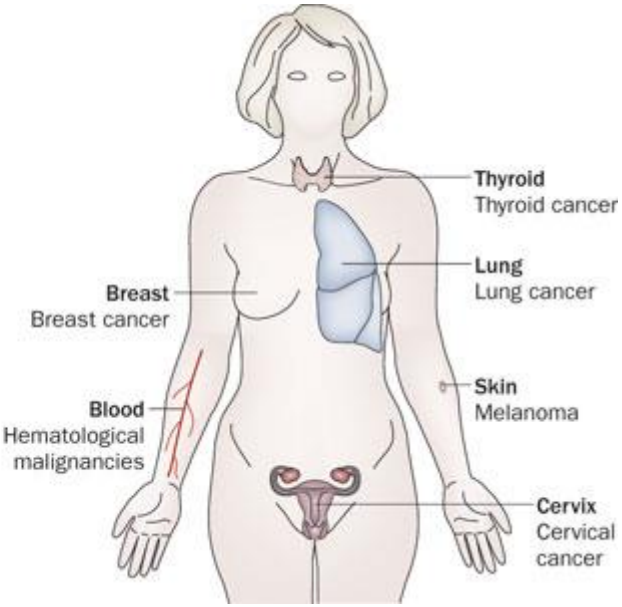






L. Fugazzola et al., 2011

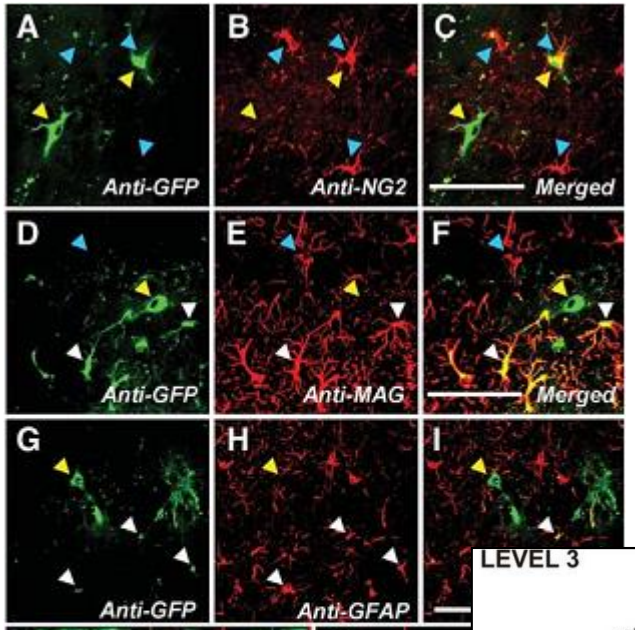


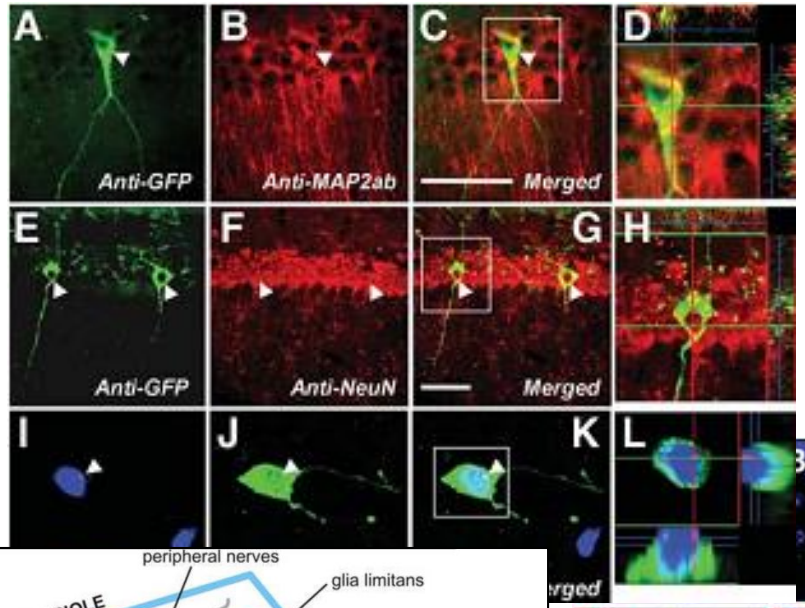


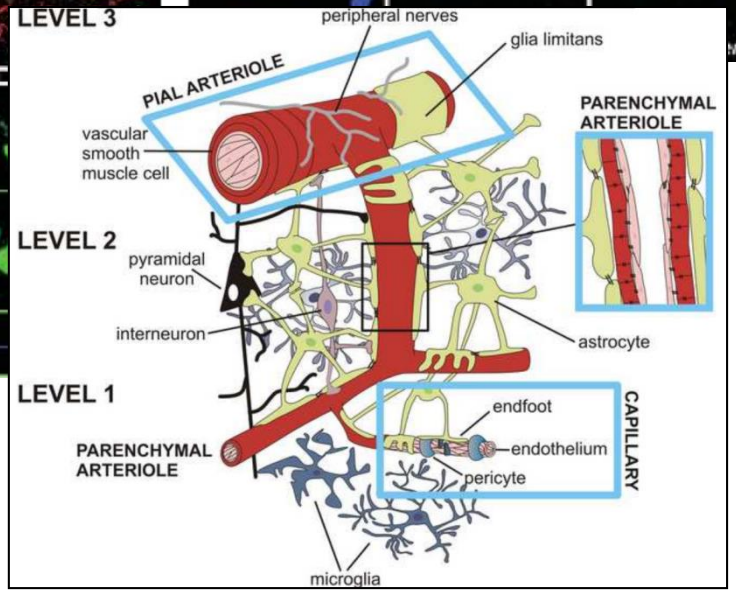
Fetal Microchimerism in the Maternal Mouse Brain: A Novel Population of Fetal Progenitor or Stem Cells Able to Cross the Blood–Brain Barrier?

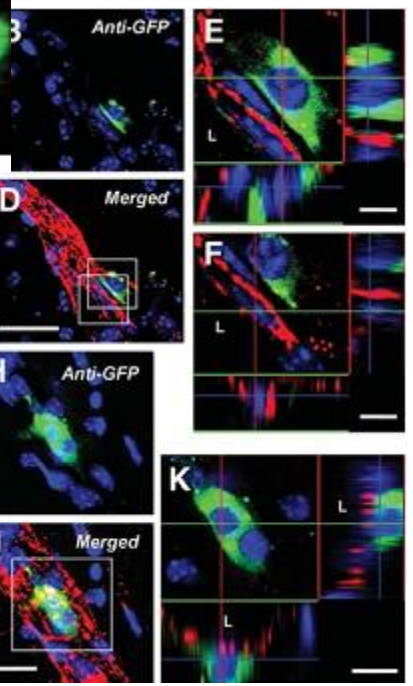
STEM CELLS 2005;23:1443–1452

XIAO-WEI TAN,[a,b] HONG LIAO,[b,c] LI SUN,[b] MASARU OKABE,[d] ZHI-CHENG XIAO,[b,c] GAVIN S. DAWE[a]









Male Microchimerism in the Human Female Brain

William F. N. Chan[1*¤], Cécile Gurnot[1], Thomas J. Montine[2], Joshua A. Sonnen[2], Katherine A. Guthrie[1], J. Lee Nelson[1,3]

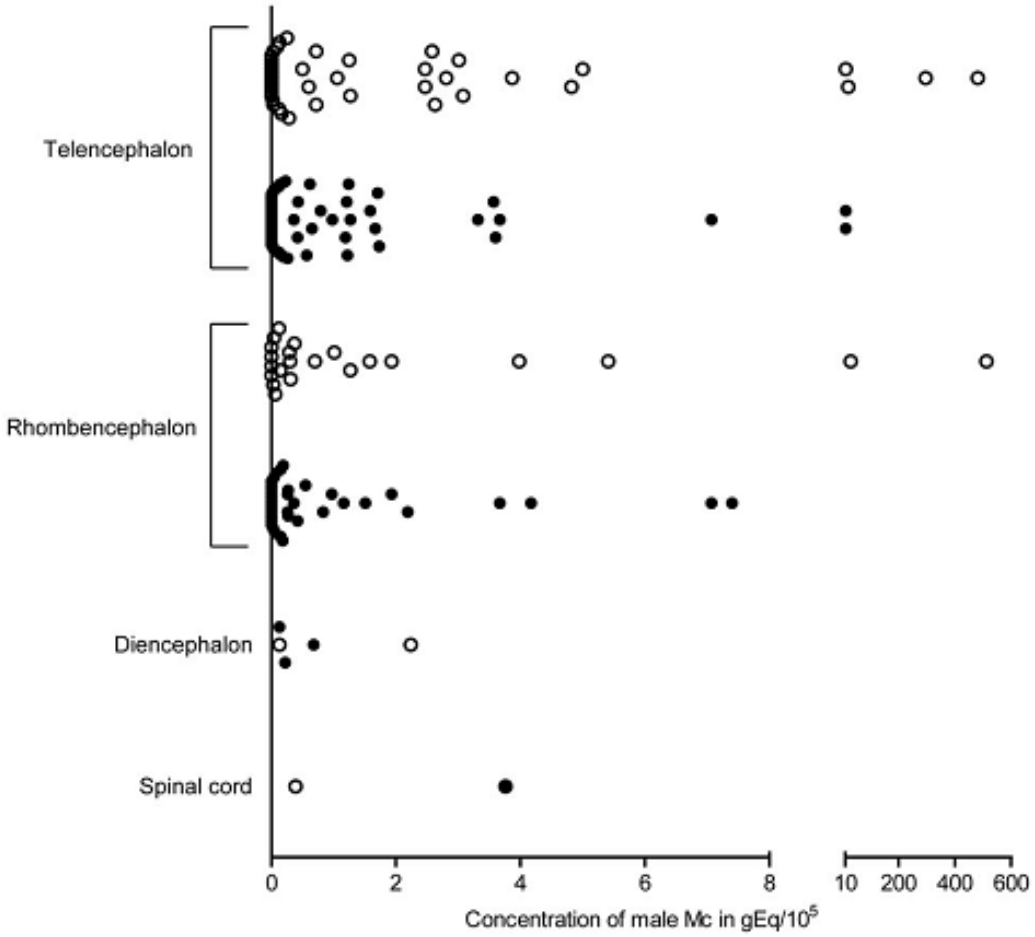


Figure 1. Concentration of male Mc in female human brain regions. Autopsied brain specimens of females without any neurologic disease (open circles) or with AD (filled circles) were tested by qPCR for male DNA. Each point represents one unique brain specimen. Telencephalon consists of neocortical regions (frontal, parietal, temporal, and occipital lobes), limbic regions (hippocampus, amygdala, and cingulate gyrus), and regions of the basal ganglia (putamen, caudate, and globus pallidus). Diencephalon consists of thalamus. Rhombencephalon consists of medulla, pons, and cerebellum. Due to the greater number of data points collected for telencephalon and rhombencephalon, data for each group have been plotted side by side to better present their distributions. Such separation was not done on the data for diencephalon and spinal cord.

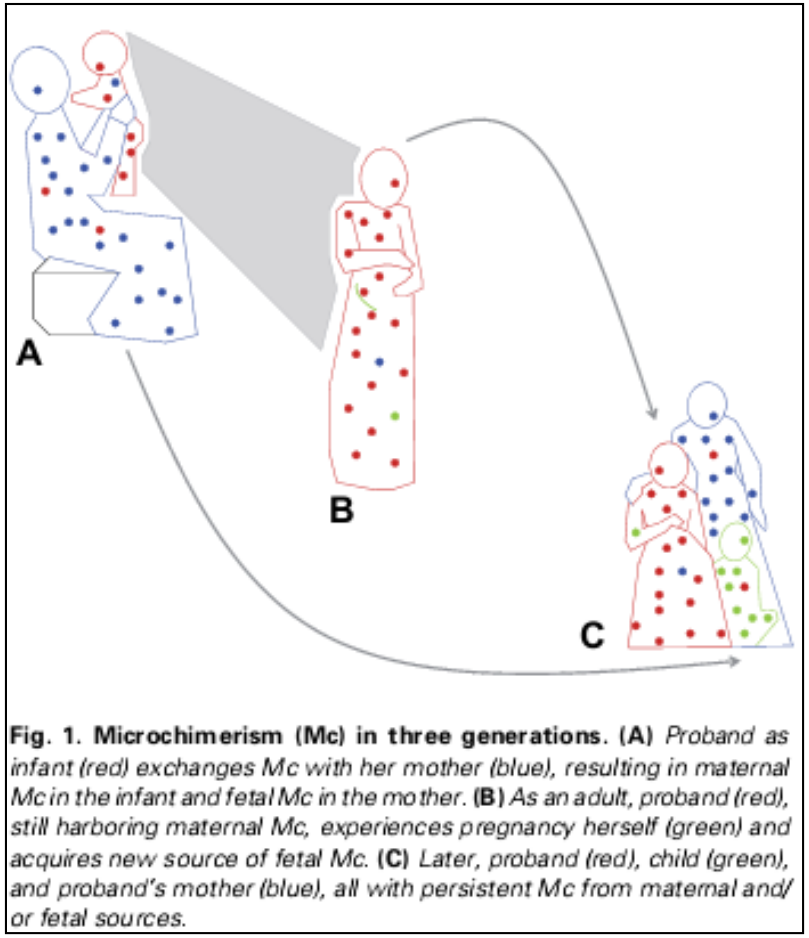
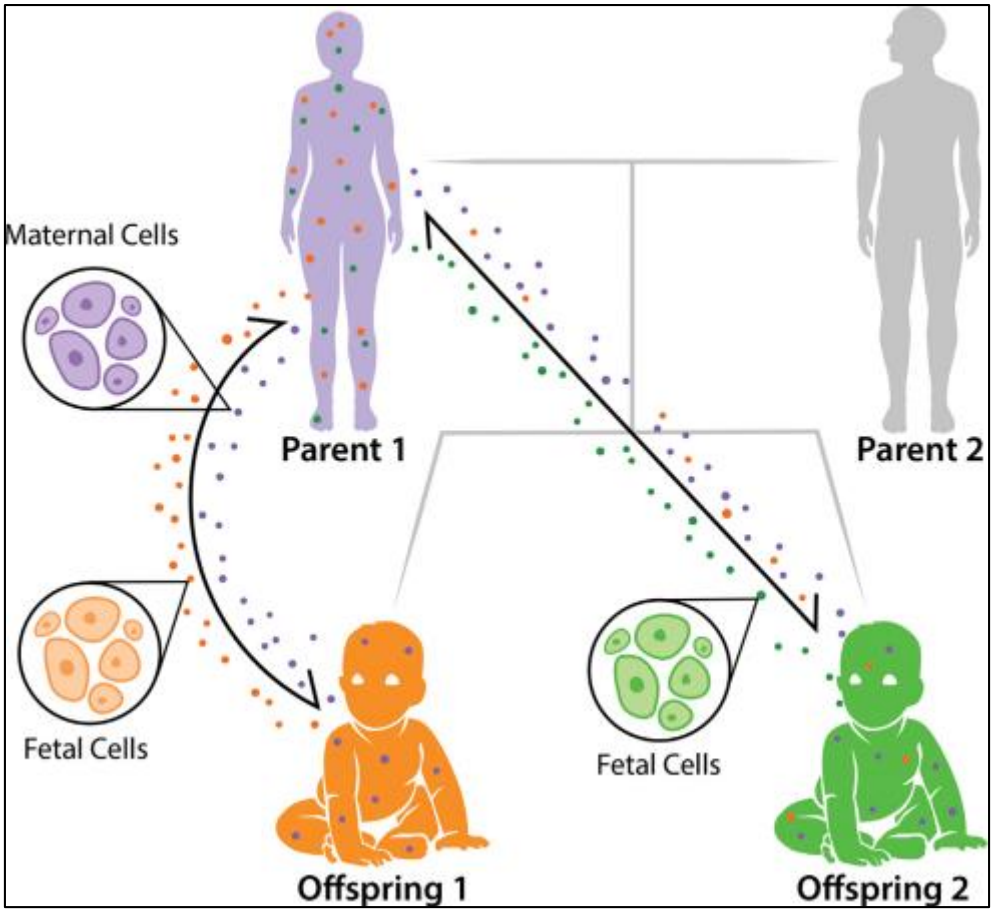


Fig. 1. Microchimerism (Mc) in three generations. **(A)** *Proband as infant (red) exchanges Mc with her mother (blue), resulting in maternal Mc in the infant and fetal Mc in the mother.* **(B)** *As an adult, proband (red), still harboring maternal Mc, experiences pregnancy herself (green) and acquires new source of fetal Mc.* **(C)** *Later, proband (red), child (green), and proband's mother (blue), all with persistent Mc from maternal and/or fetal sources.*

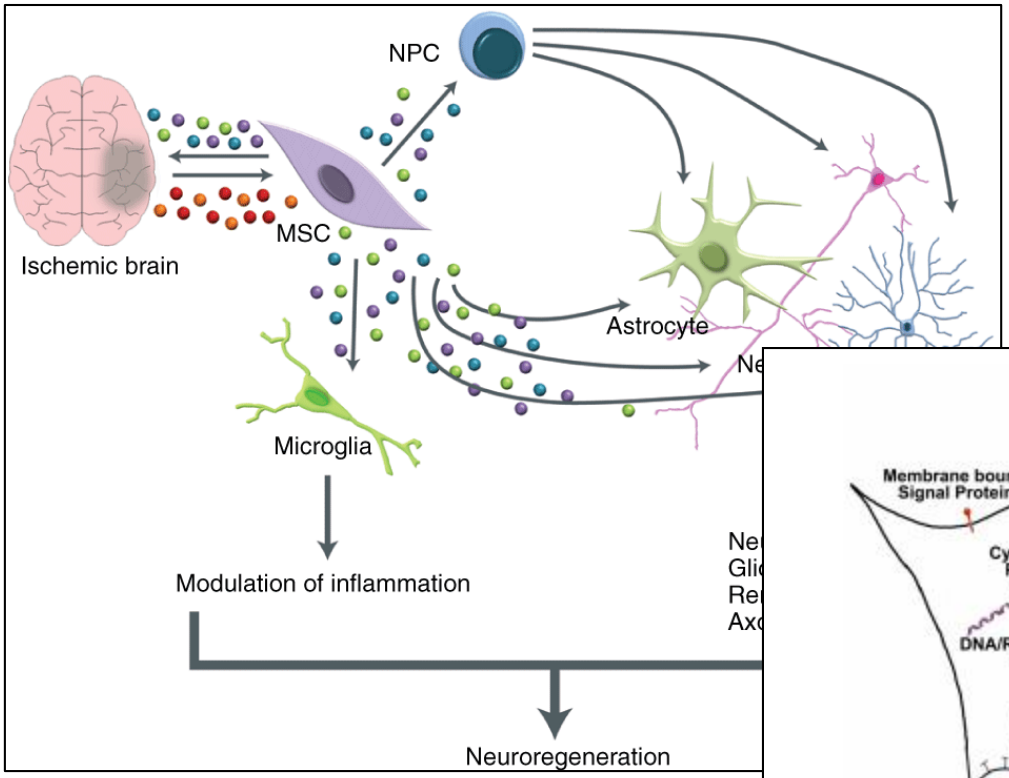


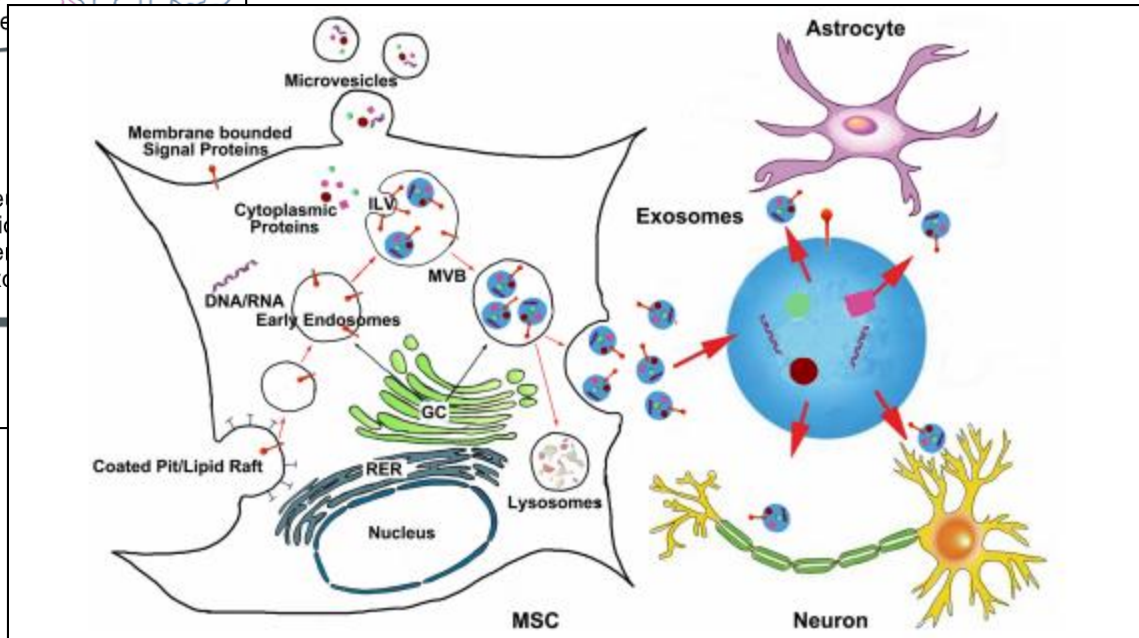
H.S. Gammill, J.L. Nelson, 2010

Chimerism 5:3-4, 63–67; July–December 2014; © 2014 Taylor & Francis Group, LLC

Exosomes: The missing link between microchimerism and acquired tolerance?

William J Burlingham*





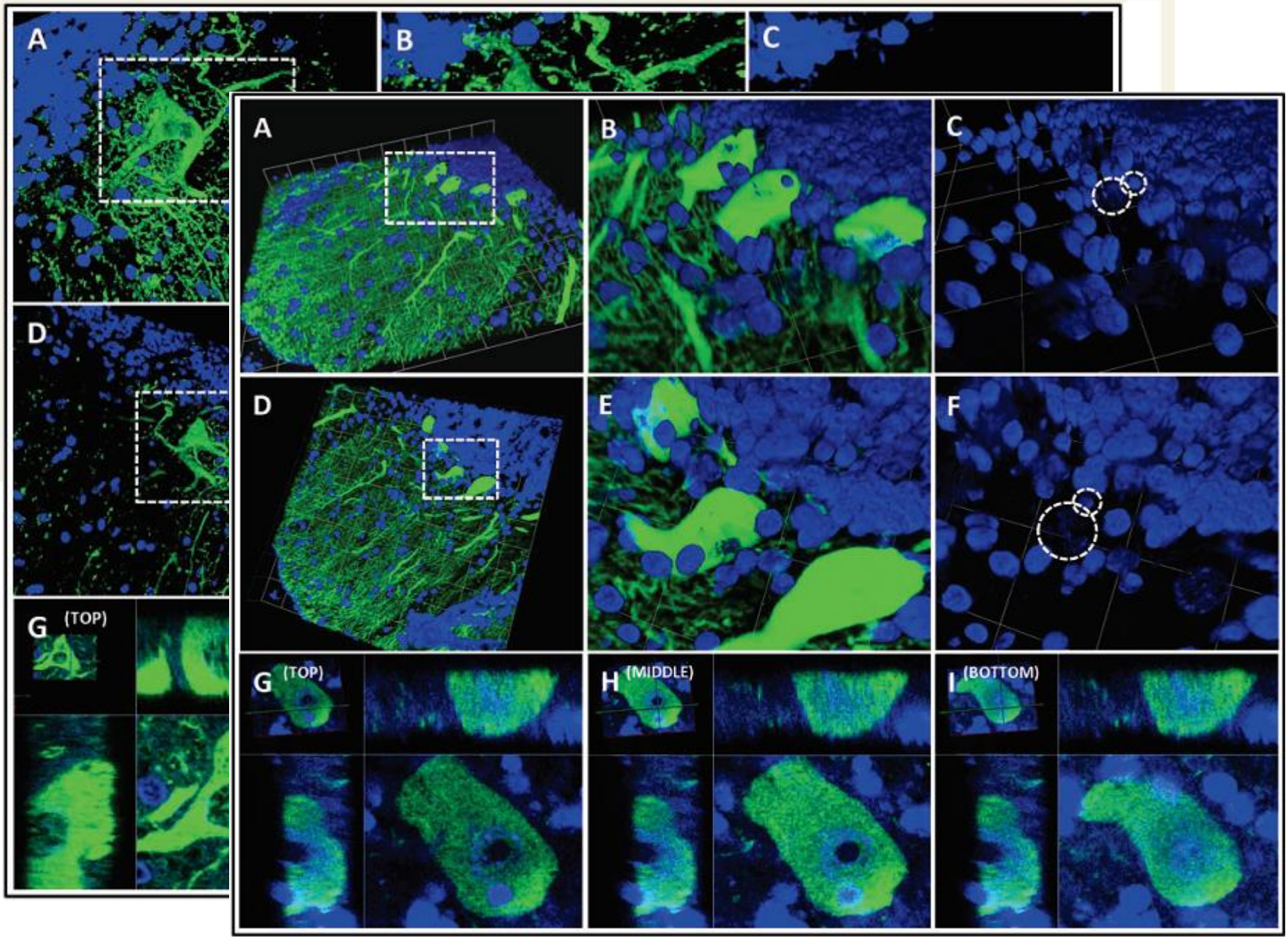
frontiers in CELLULAR NEUROSCIENCE

REVIEW ARTICLE
published: 10 November 2014
doi: 10.3389/fncel.2014.00377

Exosomes/miRNAs as mediating cell-based therapy of stroke

Hongqi Xin[1], Yi Li[1] and Michael Chopp[1,2]*

FIGURE 1 | The generation of MSC exosomes and bio-information shuttling between MSCs and brain parenchymal cells via exosomes. Exosomes are generated in the late endosomal compartment by inward budding of the limiting membrane of MVB. The exosome-filled MVBs are either fused with the plasma membrane to release exosomes or sent to lysosomes for degradation. Microvesicles are plasma membrane derived particles that are released into the extracellular environment by the direct outward budding and fission of the plasma membrane. The bio-information carried by MSC exosomes then transfer to brain parenchymal cells like astrocytes and neurons. ILV, intraluminal vesicles; MVB, multivesicular body; GC, Golgi complex; RER, rough endoplasmic reticulum.


A
B
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G (TOP)
G (TOP)
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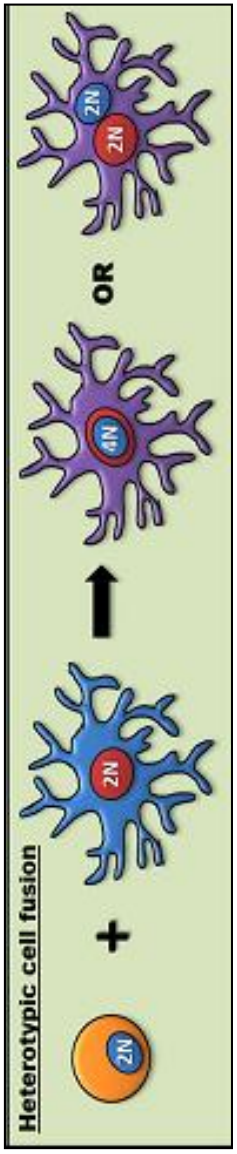
I (BOTTOM)

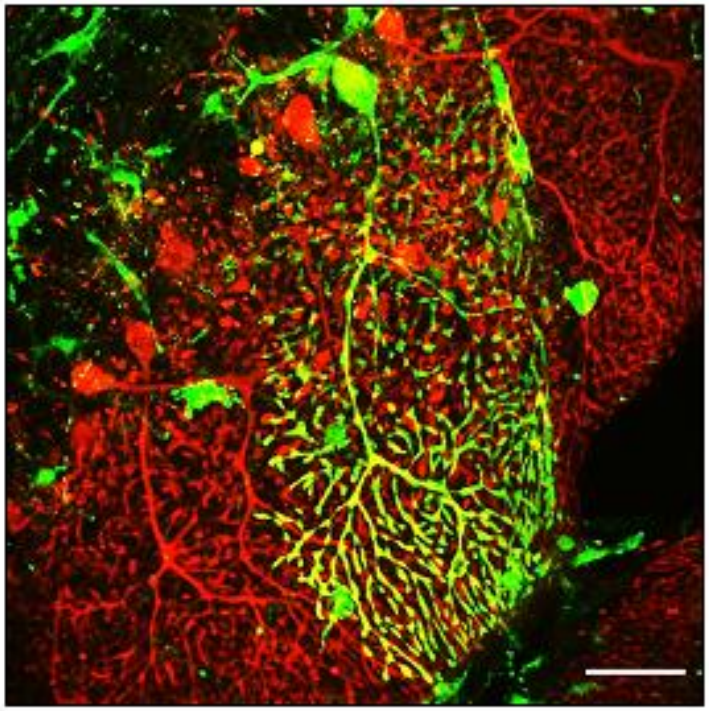
Acta Neuropathol (2014) 128:629–638
DOI 10.1007/s00401-014-1303-1

REVIEW

Cell fusion in the brain: two cells forward, one cell back

Kevin Kemp · Alastair Wilkins · Neil Scolding





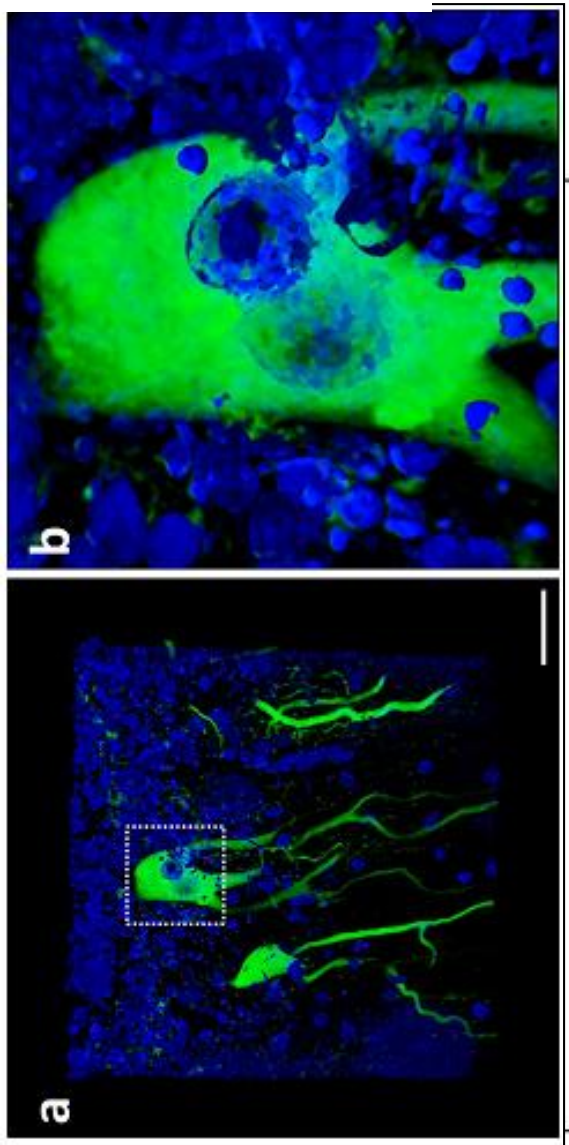
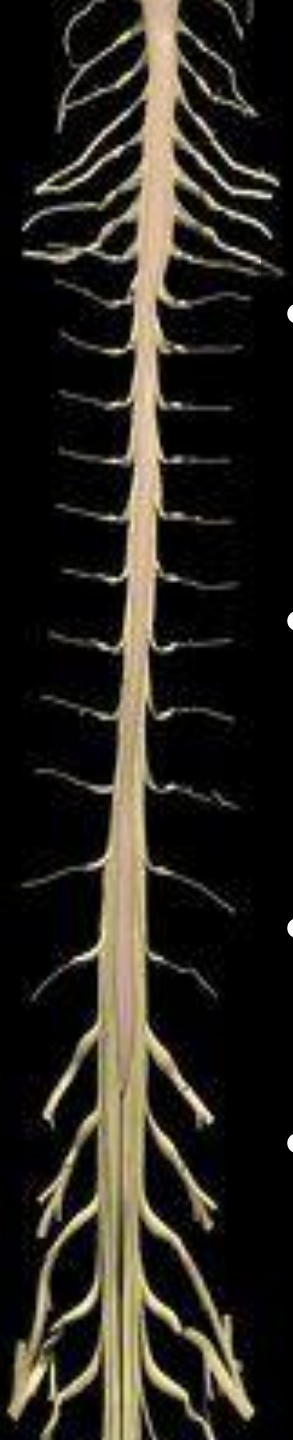


Fig. 3 A GFP-labelled bone marrow-derived Purkinje cell. A single GFP-positive Purkinje cell found within the cerebellum of a BM chimeric mouse (expressing GFP-tagged bone marrow) with experimental autoimmune encephalomyelitis (EAE). The image represents fusion between a GFP-labelled (*green*) bone marrow cell and a Calbindin-D28K positive Purkinje cell (*red*). Several GFP-positive glial cells can also be observed within the image. GFP-expressing bone marrow chimeras were produced through transplantation of GFP-tagged bone marrow stem cells into lethally irradiated mice (*scale bar* 50 μm)

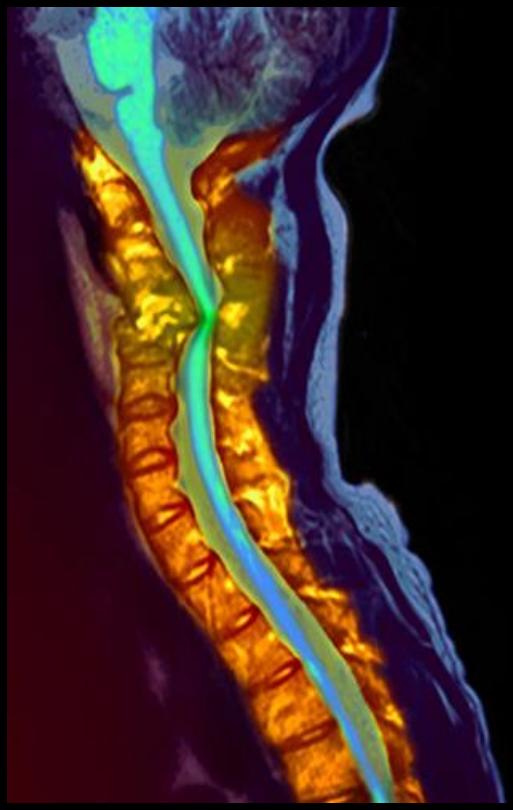
нейроінженерія

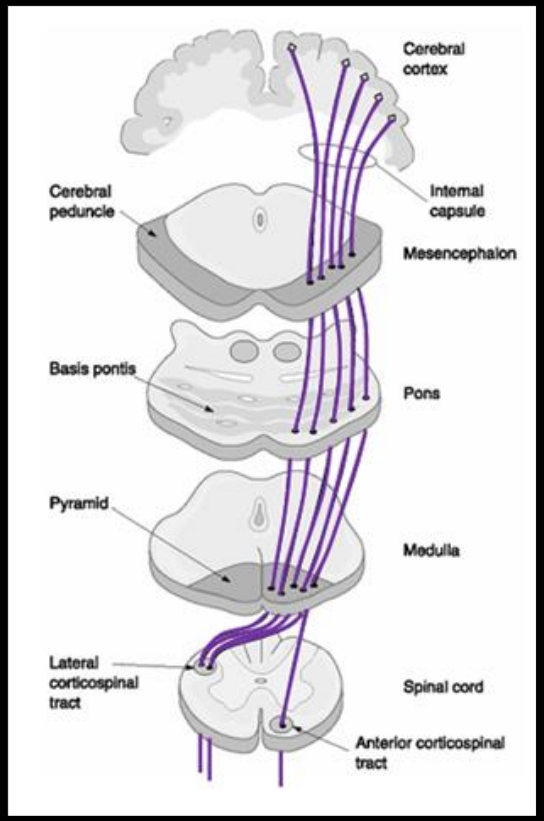
Spinal cord injury

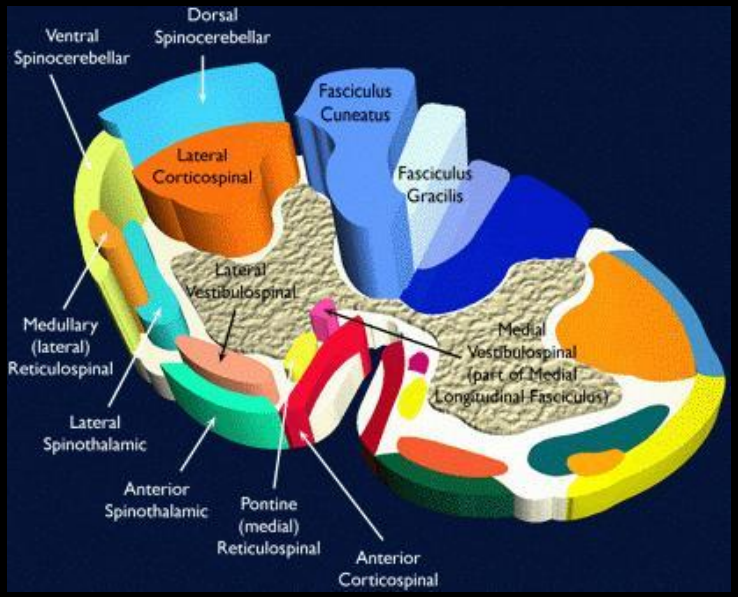


- 2,5 млн. травмованих
- чоловіки працездатного віку
- інвалідизація
- втрати і витрати

При травмі спинного мозку ушкоджуються нейрони і аксони



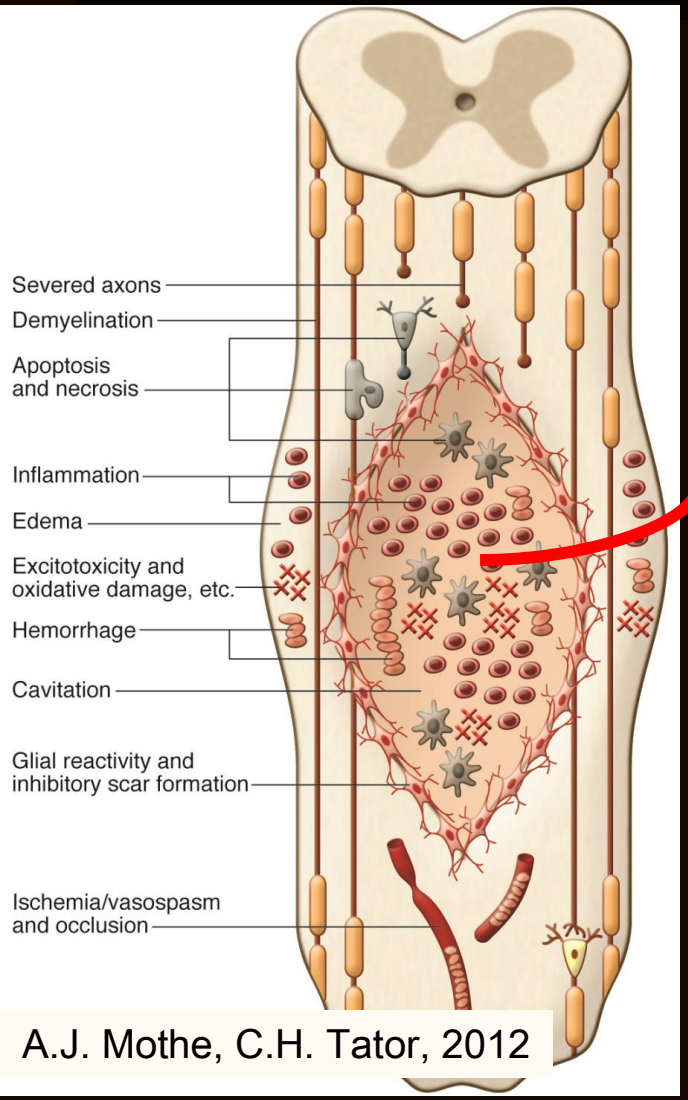


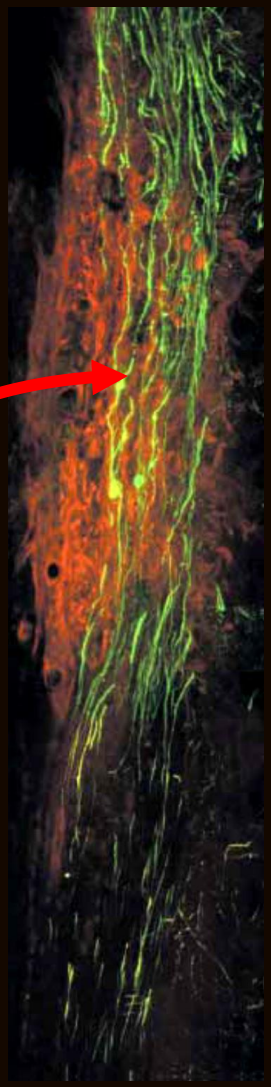


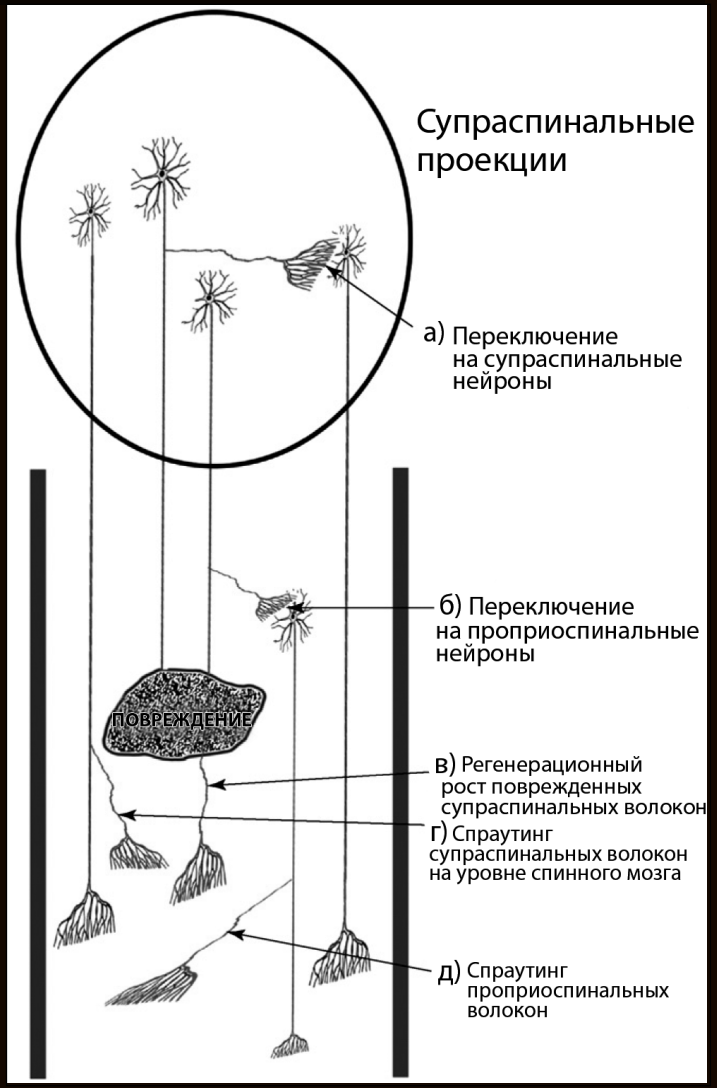
Більш важливе клінічне значення має ураження низхідних волокон спинного мозку, яке обумовлює втрату усвідомленого контролю над функцією м’язів

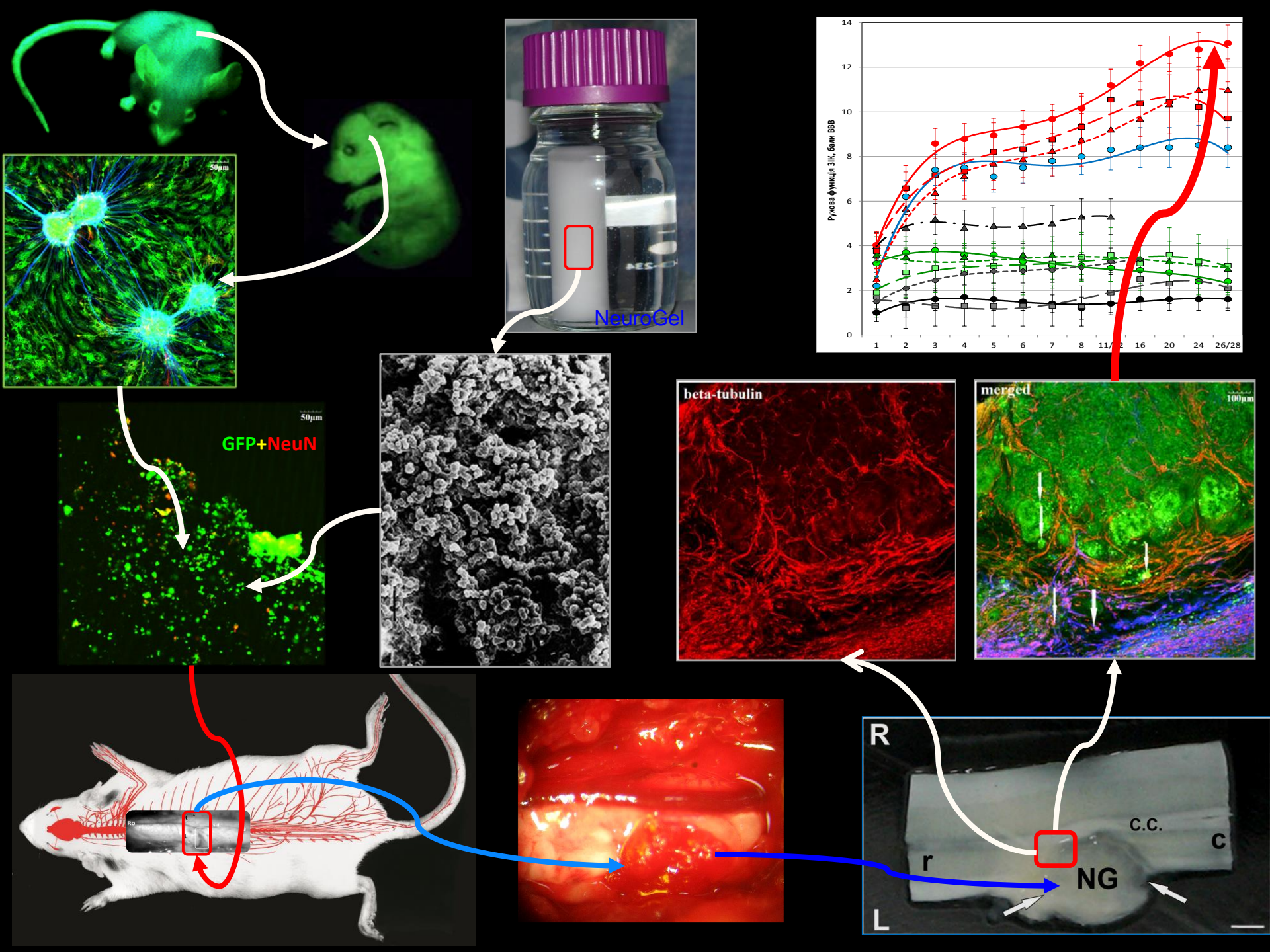
РІСТ АКСОНІВ ЧЕРЕЗ ЗОНУ ТРАВМИ БЛОКУЄТЬСЯ

Sema-3, ephrins, Nogo, OMGp, coll-IV, proteoglicans

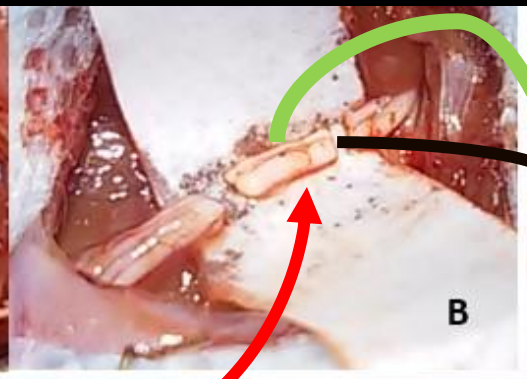


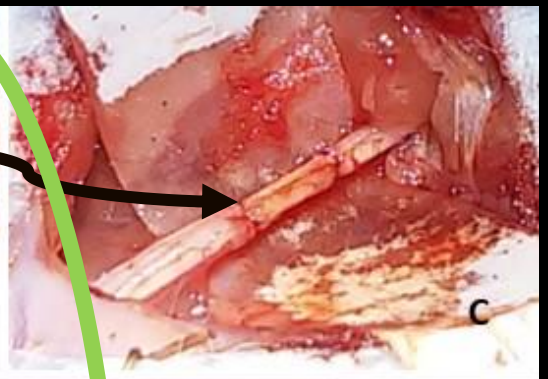




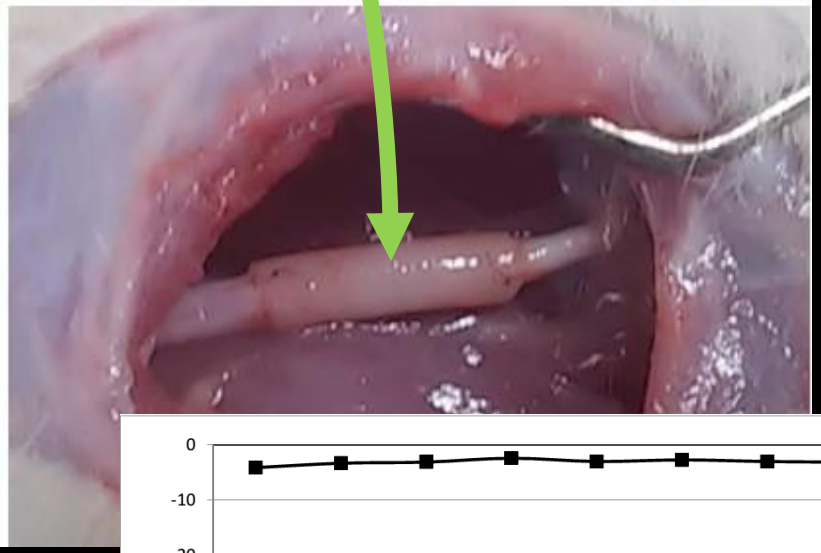

NeuroGel
Рухова функція ЗНК, бали ВВВ
GFP+NeuN
beta-tubulin
merged
R
L
r
NG
c.c.
c


A

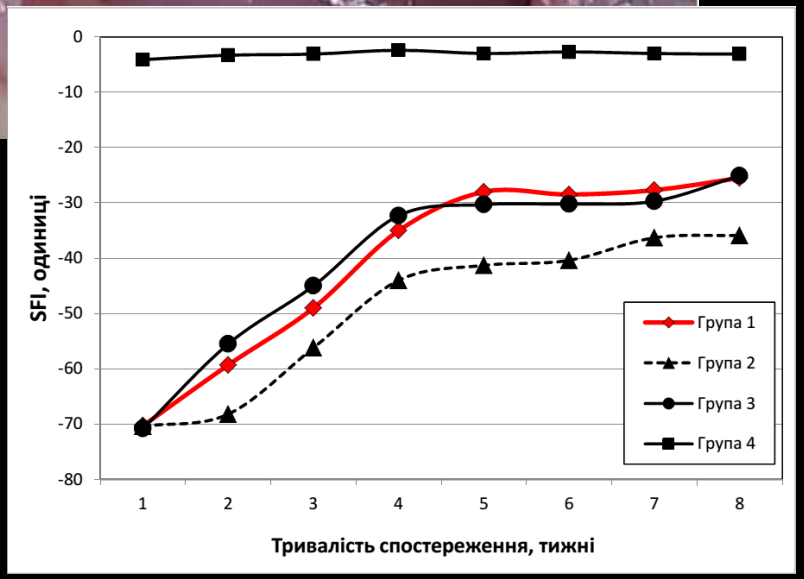

B


C


D




A
B


0
-10
-20
-30
-40
-50
-60
-70
-80
SFI, одиниці
1
2
3
4
5
6
7
8
Тривалість спостереження, тижні
Група 1
Група 2
Група 3
Група 4

Sapienti sat. Дякую.