CAMPI ELETTROMAGNETICI:

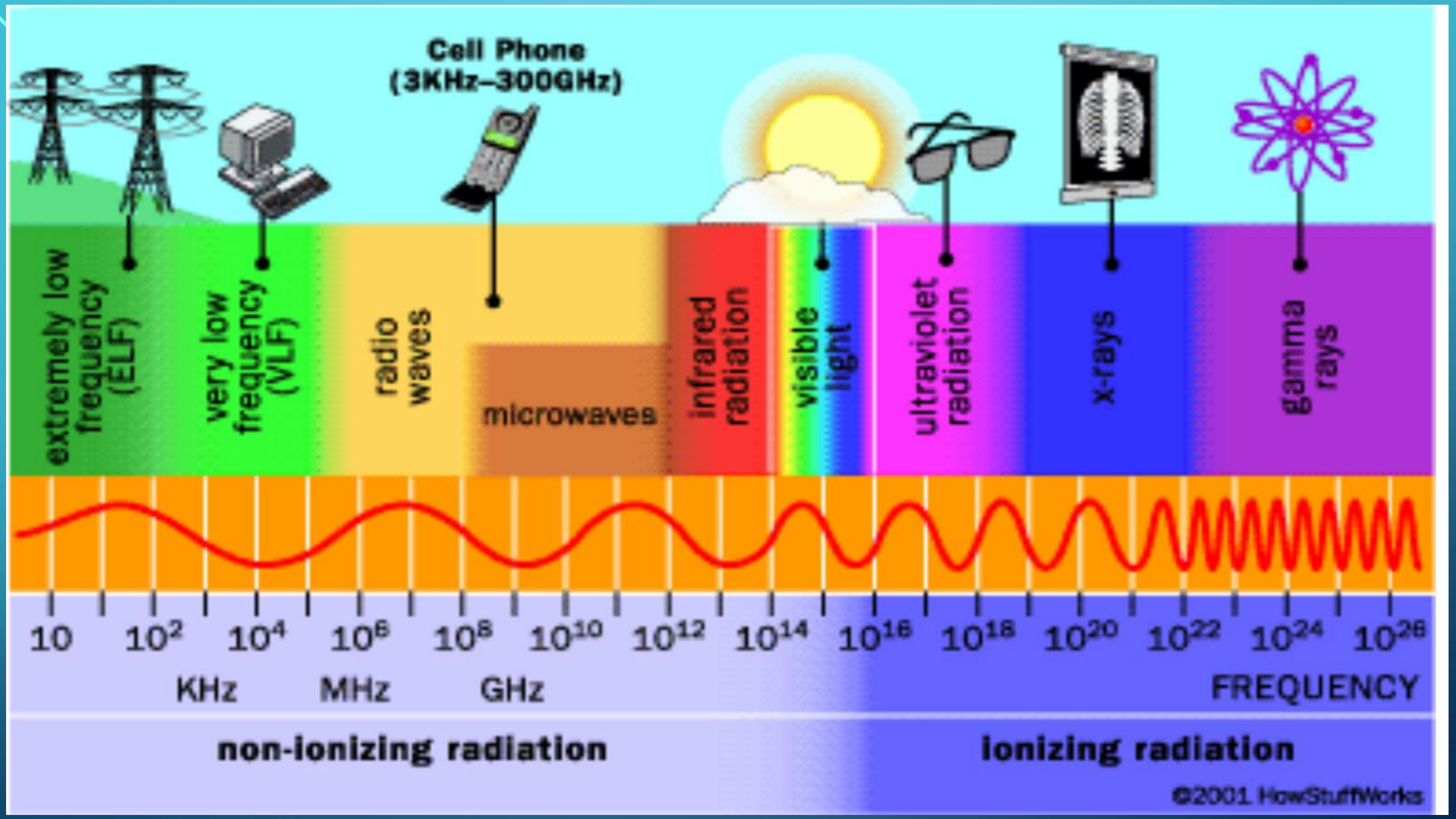
DALLE SINAPSI AI NETWORK NEURONALI

DOTT.SSA GIOVANNA SORRENTINO

NEUROLOGO DIRIGENTE MEDICO IRCCS FONDAZIONE STELLA MARIS (PI)

MASTER IN PSICONEUROENDOCRINOIMMUNOLOGIA

SOCIO SIPNEI


Cell Phone
(3KHz–300GHz)
extremely low frequency (ELF)
very low frequency (VLF)
radio waves
microwaves
infrared radiation
visible light
ultraviolet radiation
x-rays
gamma rays
10
10²
10⁴
10⁶
10⁸
10¹⁰
10¹²
10¹⁴
10¹⁶
10¹⁸
10²⁰
10²²
10²⁴
10²⁶
KHz
MHz
GHz
FREQUENCY
non-ionizing radiation
ionizing radiation
©2001 HowStuffWorks

Valentini et al, *Occup Enviro Med*, 2010 - Kwon and Hamalainem, *Bioelectromagnetics*, 2011 – Barth et al, *Bioelectromagnetics*, 2012

STORICAMENTE GLI EFFETTI ACUTI DELL'ESPOSIZIONE A CAMPI ELETTROMAGNETICI (EMF) SULLE FUNZIONI CEREBRALI, HANNO SEMPRE DESTATO PREOCCUPAZIONE, MA I DATI SPESSO SI SONO RIVELATI INCONSISTENTI O IN CONTRADDIZIONE.

ISTITUTO SUPERIORE DI SANITA'
«EFFETTI SULLA SALUTE»
30.05.2008

«…teoricamente **qualunque esposizione** *(…a campi elettromagnetici-nda)* provoca delle risposte che possono tradursi in **effetti biologici**…gli effetti biologici chiaramente documentati non sono necessariamente **nocivi**: alcuni possono essere **benefici**, altri non avere conseguenze ed altri infine provocare danni alla salute…»

«…gli effetti accertati sono **acuti** (cioè si presentano come risposta immediata all'esposizione) e si osservano solo al di sopra di determinate **soglie**…E' stata avanzata l'**ipotesi** che l'esposizione a campi di intensità inferiore alle soglie per l'insorgenza di effetti acuti, **soprattutto se cronica**, possa causare **malattie degenerative** ed in particolare il **cancro** (effetti a lungo termine).»

World Health Organization

International Agency for Research on Cancer

I CAMPI ELETTROMAGNETICI SONO CLASSIFICATI COME «POSSIBILI CANCEROGENI PER L'UOMO» E QUINDI ALLOCATI NEL GRUPPO 2B DEL SISTEMA DI CLASSIFICAZIONE DEGLI AGENTI CANCEROGENI.

STUDI EPIDEMIOLOGICI MOSTRANO L'ASSOCIAZIONE TRA USO DI TELEFONI CELLULARI E RISCHIO DI TUMORI INTRACRANICI (GLIOMI E NEURINOMI DEL NERVO ACUSTICO)

Byun et al, *PLoS ONE, 2013* - Sage, *Rev Environm Health, 2015* - Lenhart, http://pewinternet.org, *2015* - Sage, Hardell & Carpenter, *Bioelectromagnetics, 2015* – Guxens et al, *Environ Res, 2016* – *Zhang et al, Bioelectromagnetics 2017*

PIU' RECENTEMENTE, TUTTAVIA, STUDI COMPIUTI SU PIU' AMPIE POPOLAZIONI, HANNO DIMOSTRATO COME I CAMPI ELETTROMAGNETICI (EMF), INCLUSI QUELLI A FREQUENZA ESTREMAMENTE BASSA (ELF), E I CAMPI A RADIOFREQUENZA (RFR), ANCHE A BASSE DOSI DI ESPOSIZIONE POSSONO DETERMINARE EFFETTI SULLE FUNZIONI CEREBRALI. TALI CAMPI SONO GENERATI DA TELEFONI CELLULARI, CORDLESS, ANTENNE E TORRI DI TRASMISSIONE.

Bioelectromagnetics 38:329–338 (2017)

Review

Acute Effects of Radiofrequency Electromagnetic Field Emitted by Mobile Phone on Brain Function

Jun Zhang,[1] Alexander Sumich,[2] and Grace Y. Wang[3]*

[1]*School of Electrical Engineering and Automation, Tianjin University, Tianjin City, China*
[2]*Division of Psychology, School of Social Sciences, Nottingham Trent University, Nottingham, United Kingdom*
[3]*Department of Psychology, AUT University, Auckland, New Zealand*

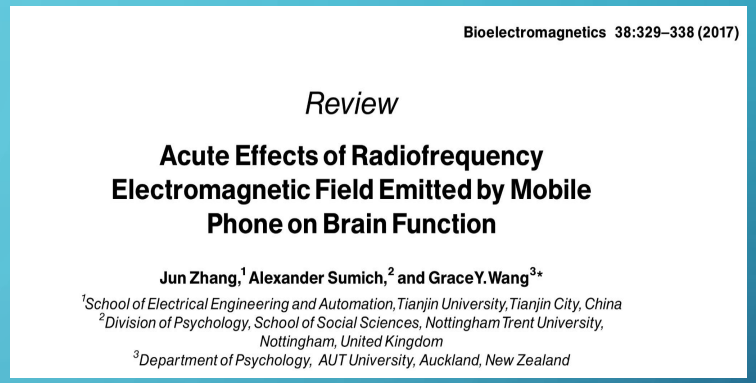
16 STUDI SELEZIONATI

TERMINI DI RICERCA CORRELATI A: «PHONE» AND «COGNITION»

TECNICHE DI RILEVAZIONE : *fMRI, PET, EEG*

CIRCA 300 ADULTI SANI (eccetto 1 adolescente, 1 pz epilettico)

EFFETTI ACUTI DELL'ESPOSIZIONE A CAMPI ELETTROMAGNETICI: AUMENTO DELL'ECCITABILITÀ CORTICALE


Bioelectromagnetics 38:329–338 (2017)

Review

Acute Effects of Radiofrequency Electromagnetic Field Emitted by Mobile Phone on Brain Function

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[3]Department of Psychology, AUT University, Auckland, New Zealand

* PET:

↑ METABOLISMO CEREBRALE

↑ * EEG:

↓ ONDE DI ALTA FREQUENZA

ONDE DI BASSA FREQUENZA

MODIFICHE DELL'ARCHITETTURA DEL RITMO DEL SONNO CON ONDE ALFA E DELTA IN FASE 2 (ALFA-DELTA SLEEP EEG PATTERN)

LE REGIONI FRONTALE E TEMPORALE APPAIONO ESSERE LE PIU' SUSCETTIBILI

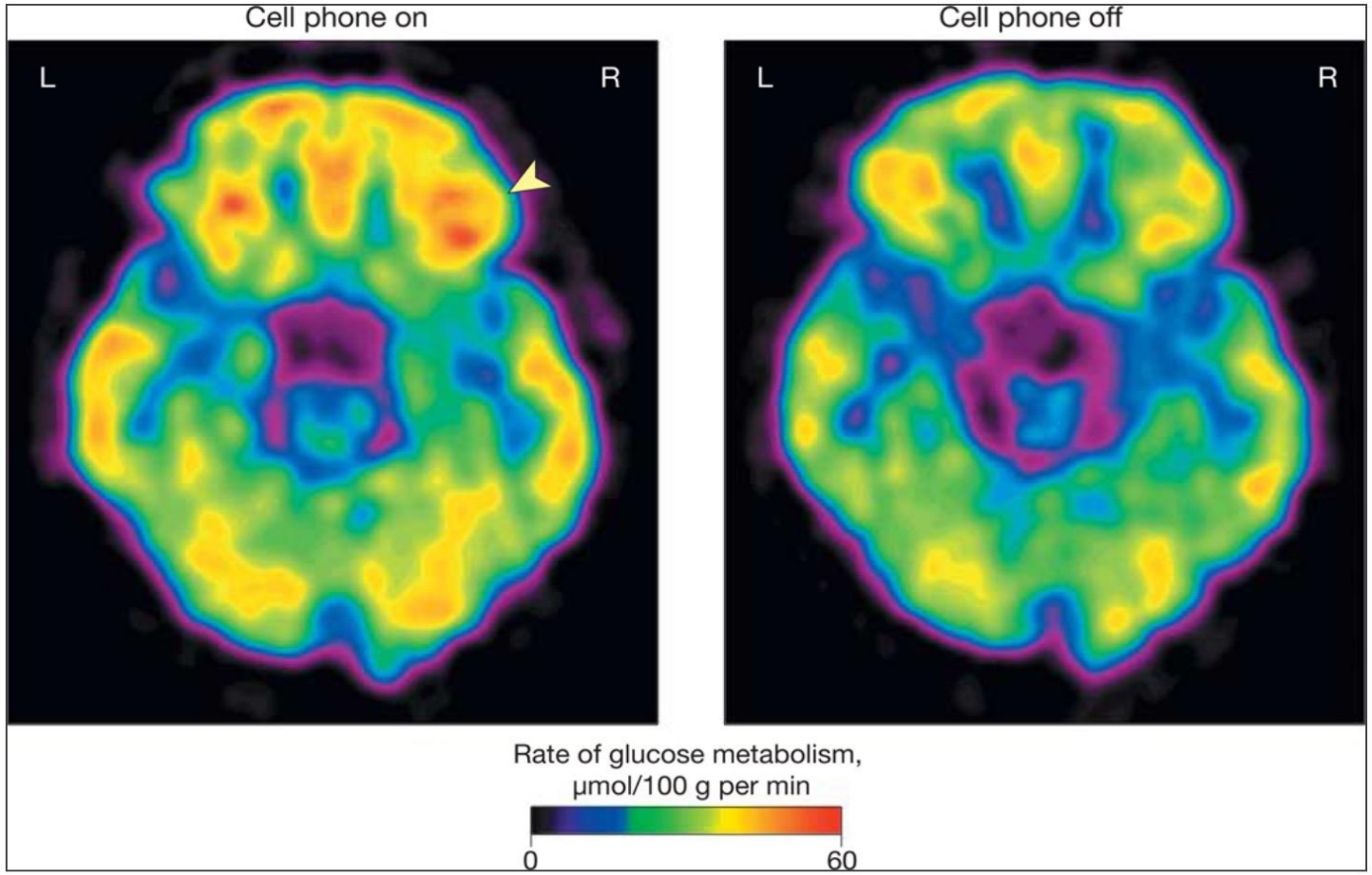

Figure 2

Brain Glucose Metabolic Images Showing Axial Planes at the Level of the Orbitofrontal Cortex

VOLKOW ET AL, JAMA 2011

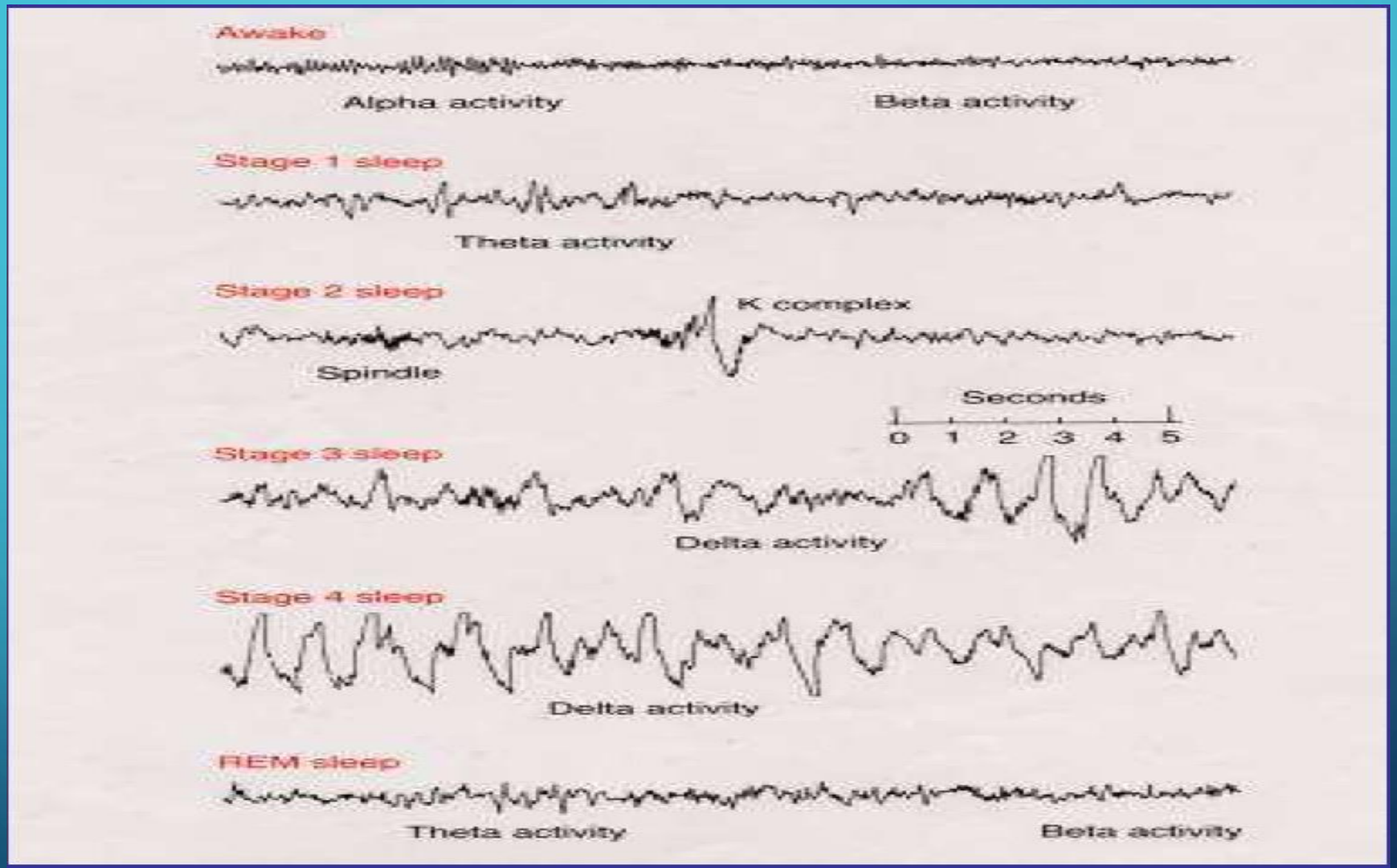
Awake
Alpha activity
Beta activity
Stage 1 sleep
Theta activity
Stage 2 sleep
K complex
Spindle
Seconds
0 1 2 3 4 5
Stage 3 sleep
Delta activity
Stage 4 sleep
Delta activity
REM sleep
Theta activity
Beta activity

Table 1
Radiofrequency (RF) Power Density and Specific Absorption Rate (SAR) Levels Reported to Cause Tissue Damage, Changes in Health Status, Neurological Function, Cognition, and Behavior Problems

Study	RF power density ($\mu W/cm^2$)	Reported health impacts
Zwamborn et al. (2003)	0.13	Anxiety, hostility, impaired cognition
Navarro et al. (2003)	0.01–0.11	Fatigue, headaches, sleeping problems
Oberfeld et al. (2004)	0.01	Sleep and concentration disruption, fatigue and cardiovascular problems
Hutter et al. (2006)	0.05–1.0	Headache, sleep, concentration problems, other neurological problems
Thomas et al. (2008)	0.005–0.04	Headaches and concentration difficulties with short-term cell phone radiation
Kundi and Hutter (2009)	0.05–0.1	Headaches, cardiac symptoms, fatigue, sleep and concentration disruption, and other impairments
Heinrich et al. (2010)	0.003–0.02	Headache, irritation, and concentration difficulties in schoolchildren and adolescents (8–17 years old) with short-term exposure to base-station level radiofrequency radiation
Thomas et al. (2010)	0.003–0.02	Conduct and behavioral problems in schoolchildren and adolescents (8–17 years old) exposed to short-term cell phone radiation
Mohler et al. (2010)	0.005	Sleep disturbances in adults with chronic cell phone tower exposure
Buchner and Eger (2011)	0.006–0.01	Significant impact on stress hormones especially in children and chronically ill adults
Avendano et al. (2012)	0.5–1.0	Decreased sperm viability and DNA breakage in human sperm with 4 hr exposure to Wi-Fi from laptop in wireless mode
Sage and Carpenter et al. (2012)	0.00034–0.07	DNA damage, impaired sperm quality, motility, and viability from cell phones on standby mode and wireless laptop use
	SAR	
Tas et al. (2014)	0.0369 W/kg[a] 2.023 W/kg[b]	Degeneration of testes tissues with 900 MHz cell phone radiation (3 hr per day exposure for 12 months)
Atasoy et al. (2013)	0.091 W/kg[a]	Damaged DNA and reduced DNA repair at levels that comply with 802.11 g Wi-Fi public safety limits
Dasdag et al. (2015a)	0.0369 W/kg[a] 2.023 W/kg[b]	Lowered microRNA activity in brain (3 hr per day exposure for 12 months)
Akdag et al. (2016)	141.4 μW/kg[a] 7127 μW/kg[b]	DNA damage in testes by comet assay (24/7 exposure for 12 months—900 MHz cell phone radiation)
Dasdag et al. (2015b)	141.4 μW/kg[a] 7127 μW/kg[b]	Lowered microRNA activity in brain (24/7 exposure for 12 months—2.45 GHz Wi-Fi radiation)

[a]Whole body. [b]Max SAR.

smartphone, and another 25%–30% have a basic cell populations is profiled by Kabali et al. (2015), who

Sage et Burgio, Child Develop, 2017

PATPHY-776; No. of Pages 19

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Pathophysiology xxx (2013) xxx–xxx

ISP PATHOPHYSIOLOGY

www.elsevier.com/locate/pathophys

Autism and EMF? Plausibility of a pathophysiological link – Part I

Martha R. Herbert [a,*], Cindy Sage [b]

[a] *TRANSCEND Research Program Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, MA 02129, USA*
[b] *Sage Associates, Santa Barbara, CA, USA*

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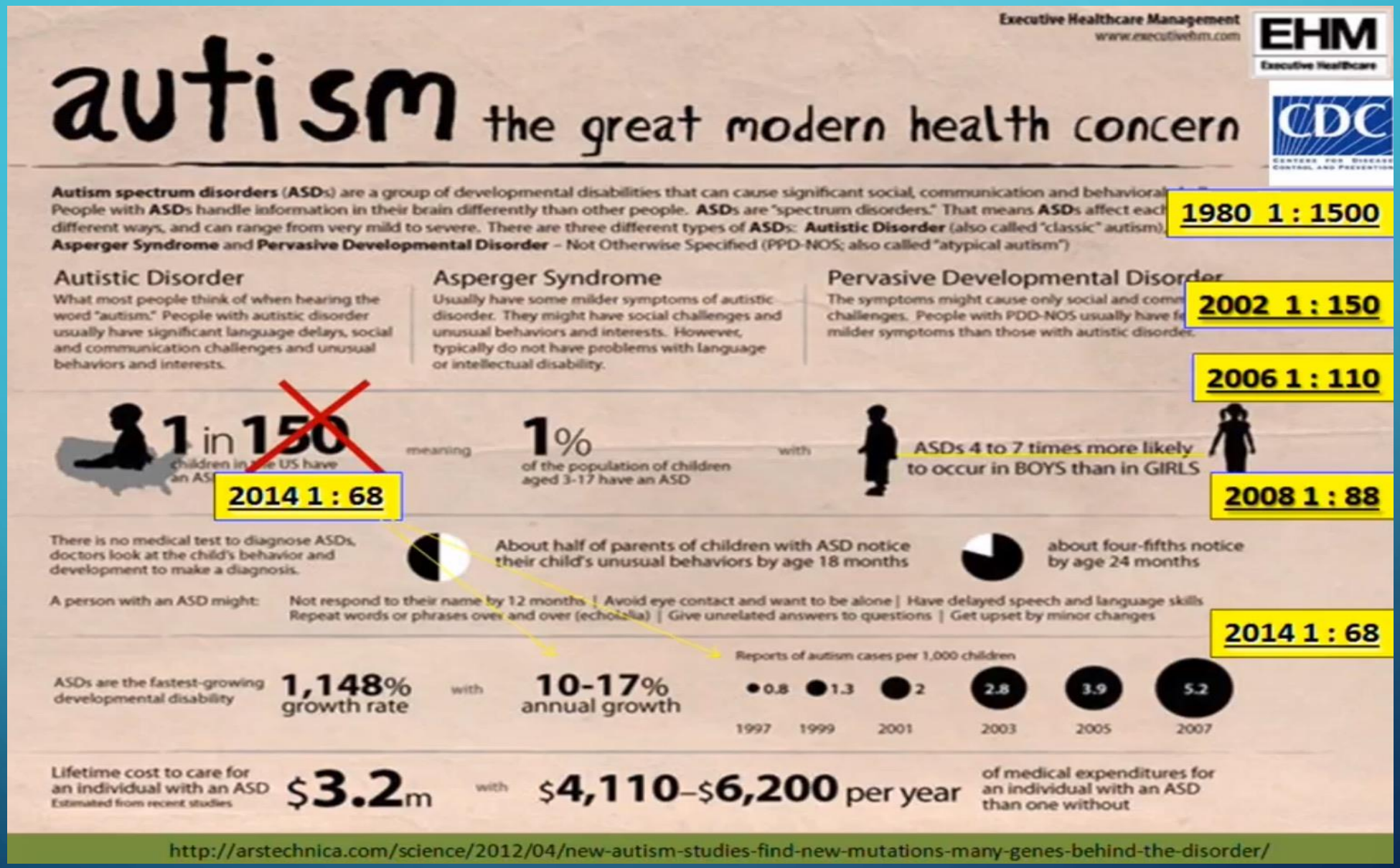
Abstract

Although autism spectrum conditions (ASCs) are defined behaviorally, they also involve multileveled disturbances of underlying biology that find striking parallels in the physiological impacts of electromagnetic frequency and radiofrequency exposures (EMF/RFR). Part I of this paper will review the critical contributions pathophysiology may make to the etiology, pathogenesis and ongoing generation of core features of ASCs. We will review pathophysiological damage to core cellular processes that are associated both with ASCs and with biological effects of EMF/RFR exposures that contribute to chronically disrupted homeostasis. Many studies of people with ASCs have identified oxidative stress and evidence of free radical damage, cellular stress proteins, and deficiencies of antioxidants such as glutathione. Elevated intracellular calcium in ASCs may be due to genetics or may be downstream of inflammation or environmental exposures. Cell membrane lipids may be peroxidized, mitochondria may be dysfunctional, and various kinds of immune system disturbances are common. Brain oxidative stress and inflammation as well as measures consistent with blood–brain barrier and brain perfusion compromise have been documented. Part II of this paper will review how behaviors in ASCs may emerge from alterations of electrophysiological oscillatory synchronization, how EMF/RFR could contribute to these by de-tuning the organism, and policy implications of these vulnerabilities. Changes in brain and autonomic nervous


Executive Healthcare Management
www.executivehm.com
EHM
Executive Healthcare
CDC
autism the great modern health concern
Autism spectrum disorders (ASDs) are a group of developmental disabilities that can cause significant social, communication and behavioral
People with ASDs handle information in their brain differently than other people. ASDs are "spectrum disorders." That means ASDs affect each
different ways, and can range from very mild to severe. There are three different types of ASDs: Autistic Disorder (also called "classic" autism),
Asperger Syndrome and Pervasive Developmental Disorder – Not Otherwise Specified (PPD-NOS; also called "atypical autism")
1980 1 : 1500
Autistic Disorder
What most people think of when hearing the word "autism." People with autistic disorder usually have significant language delays, social and communication challenges and unusual behaviors and interests.
Asperger Syndrome
Usually have some milder symptoms of autistic disorder. They might have social challenges and unusual behaviors and interests. However, typically do not have problems with language or intellectual disability.
Pervasive Developmental Disorder
The symptoms might cause only social and comm
challenges. People with PDD-NOS usually have f
milder symptoms than those with autistic disorder.
2002 1 : 150
2006 1 : 110
1 in 150
children in the US have
an AS
2014 1 : 68
meaning
1%
of the population of children aged 3-17 have an ASD
with
ASDs 4 to 7 times more likely to occur in BOYS than in GIRLS
2008 1 : 88
There is no medical test to diagnose ASDs, doctors look at the child's behavior and development to make a diagnosis.
About half of parents of children with ASD notice their child's unusual behaviors by age 18 months
about four-fifths notice by age 24 months
A person with an ASD might:
Not respond to their name by 12 months | Avoid eye contact and want to be alone | Have delayed speech and language skills
Repeat words or phrases over and over (echolalia) | Give unrelated answers to questions | Get upset by minor changes
2014 1 : 68
Reports of autism cases per 1,000 children
ASDs are the fastest-growing developmental disability
1,148% growth rate
with
10-17% annual growth
0.8
1.3
2
2.8
3.9
5.2
1997
1999
2001
2003
2005
2007
Lifetime cost to care for an individual with an ASD
Estimated from recent studies
$3.2m
with
$4,110–$6,200 per year
of medical expenditures for an individual with an ASD than one without
http://arstechnica.com/science/2012/04/new-autism-studies-find-new-mutations-many-genes-behind-the-disorder/

Mobile Phone Use, Blood Lead Levels, and Attention Deficit Hyperactivity Symptoms in Children: A Longitudinal Study

Yoon-Hwan Byun[1], Mina Ha[2,3]*, Ho-Jang Kwon[2,3], Yun-Chul Hong[4], Jong-Han Leem[5], Joon Sakong[6], Su Young Kim[7], Chul Gab Lee[8], Dongmug Kang[9], Hyung-Do Choi[10], Nam Kim[11]

1 Department of Medicine, Dankook University College of Medicine, Cheonan, Korea, **2** Department of Preventive Medicine, Dankook University College of Medicine, Cheonan, Korea, **3** Environmental Health Center, Dankook University Medical Center, Cheonan, Korea, **4** Department of Preventive Medicine, Seoul National University College of Medicine, Seoul, Korea, **5** Department of Occupational and Environmental Medicine, Inha University College of Medicine, Incheon, Korea, **6** Department of Preventive Medicine, Yeungnam University College of Medicine, Daegu, Korea, **7** Department of Preventive Medicine, Cheju National University College of Medicine, Jeju, Korea, **8** Department of Occupational Medicine, Chosun University School of Medicine, Gwangju, Korea, **9** Department of Occupational Medicine, Busan National University School of Medicine, Busan, Korea, **10** Radio Technology Research Department, Electronics and Telecommunication Research Institute, Daejeon, Korea, **11** School of Information and Communication Engineering, Chungbuk National University College of Electrical and Computer Engineering, Cheongju, Korea

Abstract

Background: Concerns have developed for the possible negative health effects of radiofrequency electromagnetic field (RF-EMF) exposure to children's brains. The purpose of this longitudinal study was to investigate the association between mobile phone use and symptoms of Attention Deficit Hyperactivity Disorder (ADHD) considering the modifying effect of lead exposure.

Methods: A total of 2,422 children at 27 elementary schools in 10 Korean cities were examined and followed up 2 years later. Parents or guardians were administered a questionnaire including the Korean version of the ADHD rating scale and questions about mobile phone use, as well as socio-demographic factors. The ADHD symptom risk for mobile phone use was estimated at two time points using logistic regression and combined over 2 years using the generalized estimating equation model with repeatedly measured variables of mobile phone use, blood lead, and ADHD symptoms, adjusted for covariates.

Results: The ADHD symptom risk associated with mobile phone use for voice calls but the association was limited to children exposed to relatively high lead.

Conclusions: The results suggest that simultaneous exposure to lead and RF from mobile phone use was associated with increased ADHD symptom risk, although possible reverse causality could not be ruled out.

Citation: Byun Y-H, Ha M, Kwon H-J, Hong Y-C, Leem J-H, et al. (2013) Mobile Phone Use, Blood Lead Levels, and Attention Deficit Hyperactivity Symptoms in Children: A Longitudinal Study. PLoS ONE 8(3): e59742. doi:10.1371/journal.pone.0059742

Editor: James G. Scott, The University of Queensland, Australia

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International Journal of Medicine and Medical Sciences

Full Length Research Paper

Autism: An epigenomic side-effect of excessive exposure to electromagnetic fields

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[3]Department of Genetics, Shadan College, Khairatabad, Hyderabad 500004, India.

Accepted 27 February, 2013

Autism is a disorder which mainly involves the nervous system. It is characterized by lack of communication, incoherent language and meaningless repetitive movements. Its onset is in early childhood and its incidence has been reported to be increasing. Several genes and environmental factors have been implicated in the causation of autism, and electromagnetic fields may be one of those environmental factors. Industrialization has added a large number of electronic gadgets around us. Indiscriminate use of these gadgets, particularly mobile phones, has raised the question of electropollution and health hazard caused by their usage. Electromagnetic fields emitted during their operation do not have enough energy to cause DNA alterations directly; however, ample evidence is available from *in vitro* and *in vivo* studies to demonstrate their ability to cause DNA alterations indirectly as well as epigenetic modifications. In addition to genetic alterations, the epigenetic modifications may have an important role in causing disruption of the nervous system leading to neurodegenerative disorders, including autism.

CHILD DEVELOPMENT

Child Development, xxxx 2017, Volume 00, Number 0, Pages 1–8

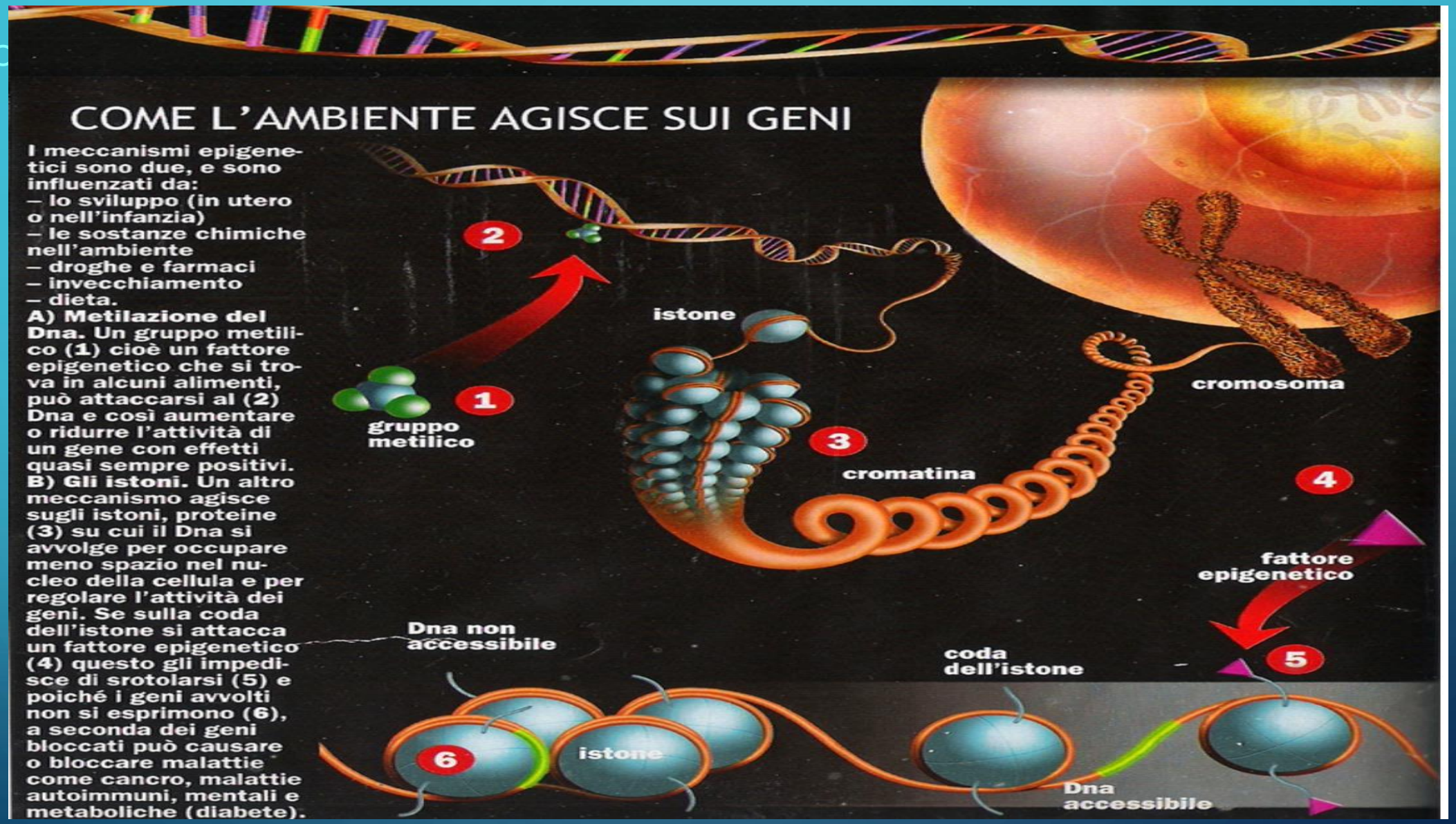
The title for this Special Section is **Contemporary Mobile Technology and Child and Adolescent Development**, edited by Zheng Yan and Lennart Hardell

Electromagnetic Fields, Pulsed Radiofrequency Radiation, and Epigenetics: How Wireless Technologies May Affect Childhood Development

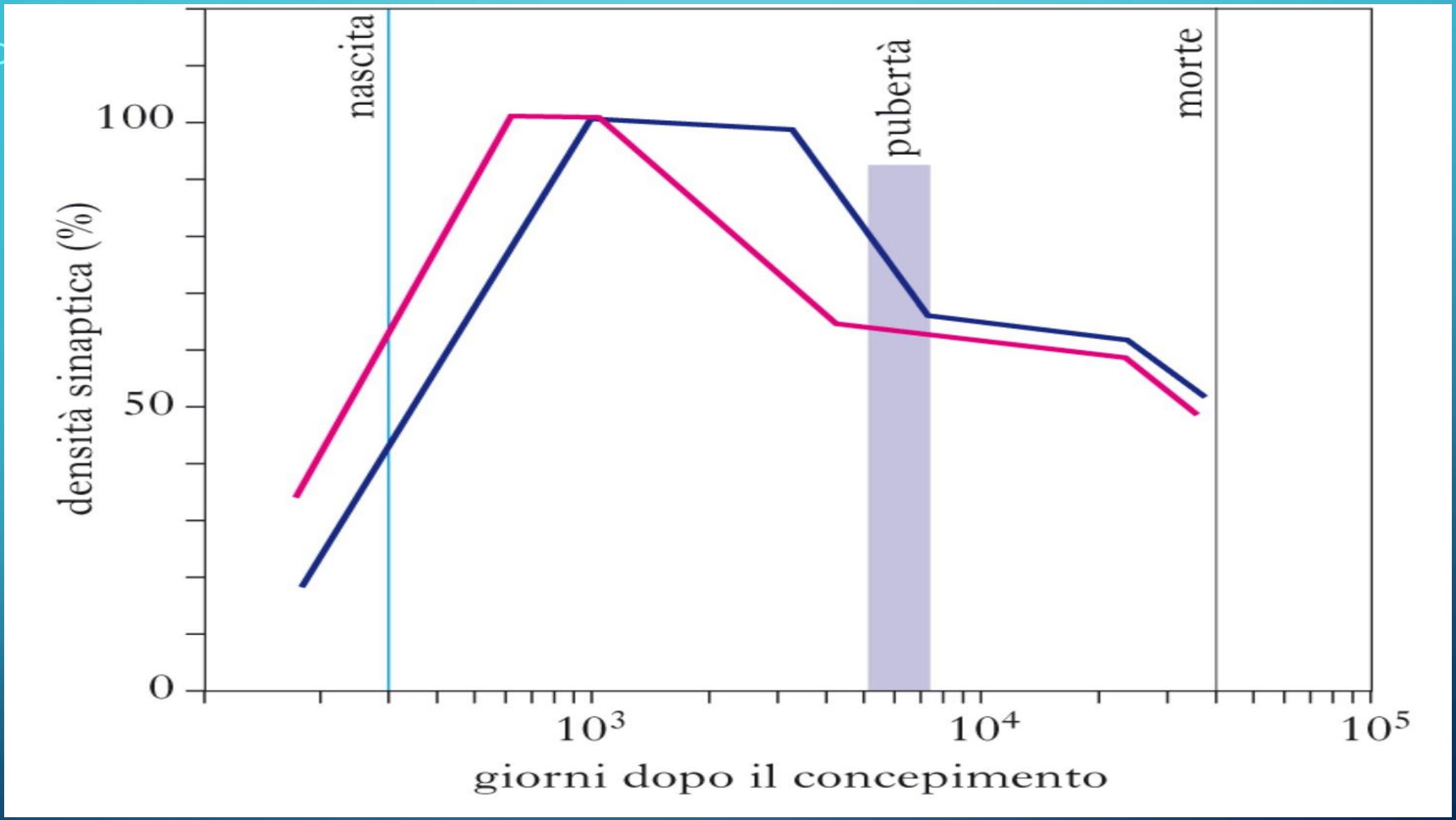
Cindy Sage
Sage Associates

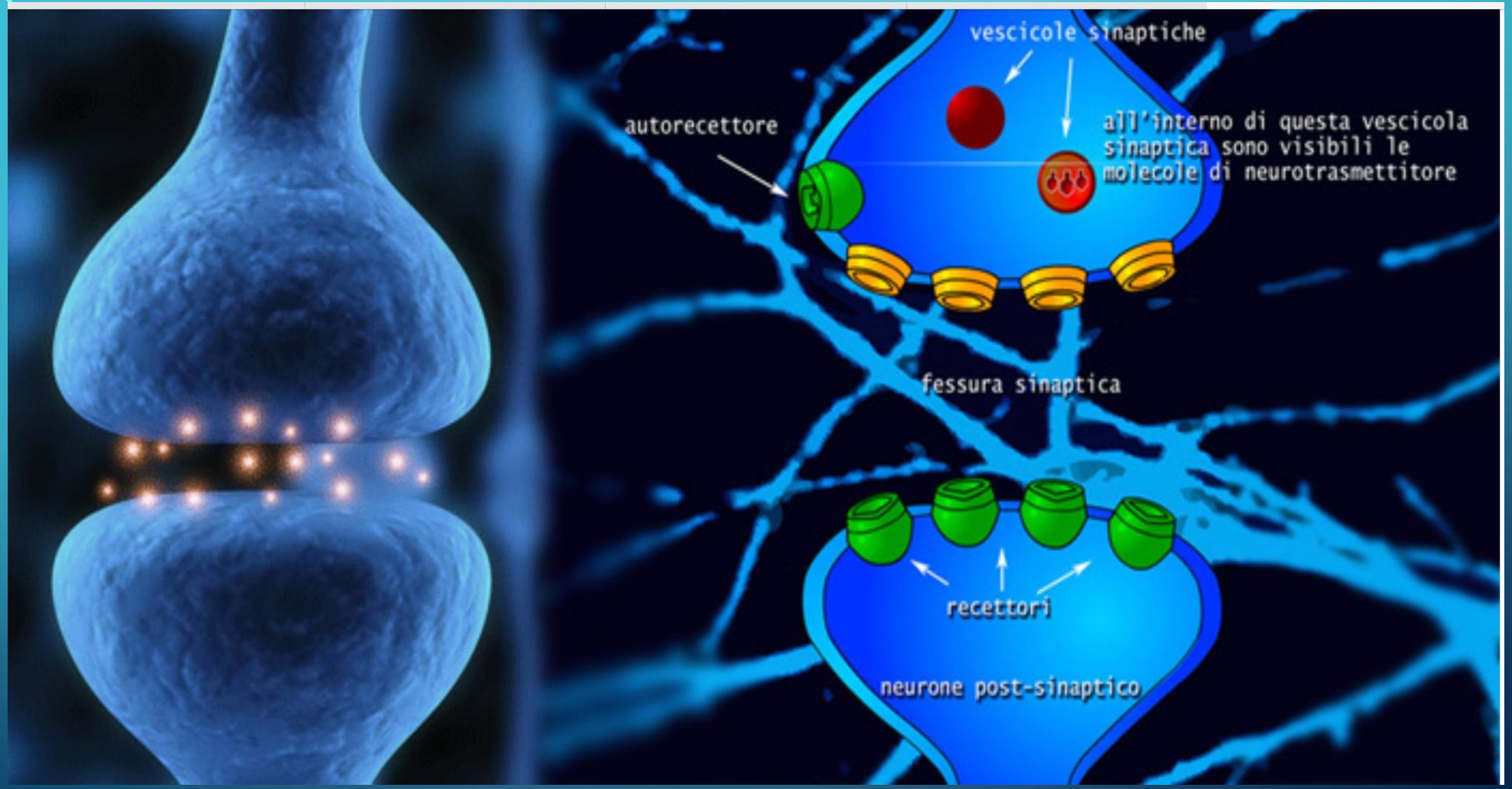
Ernesto Burgio
International Society of Doctors for Environment (ISDE) Scientific Office

Mobile phones and other wireless devices that produce electromagnetic fields (EMF) and pulsed radiofrequency radiation (RFR) are widely documented to cause potentially harmful health impacts that can be detrimental to young people. New epigenetic studies are profiled in this review to account for some neurodevelopmental and neurobehavioral changes due to exposure to wireless technologies. Symptoms of retarded memory, learning, cognition, attention, and behavioral problems have been reported in numerous studies and are similarly manifested in autism and attention deficit hyperactivity disorders, as a result of EMF and RFR exposures where both epigenetic drivers and genetic (DNA) damage are likely contributors. Technology benefits can be realized by adopting wired devices for education to avoid health risk and promote academic achievement.


COME L'AMBIENTE AGISCE SUI GENI
I meccanismi epigenetici sono due, e sono influenzati da:
– lo sviluppo (in utero o nell'infanzia)
– le sostanze chimiche nell'ambiente
– droghe e farmaci
– invecchiamento
– dieta.
A) Metilazione del Dna. Un gruppo metilico (1) cioè un fattore epigenetico che si trova in alcuni alimenti, può attaccarsi al (2) Dna e così aumentare o ridurre l'attività di un gene con effetti quasi sempre positivi.
B) Gli istoni. Un altro meccanismo agisce sugli istoni, proteine (3) su cui il Dna si avvolge per occupare meno spazio nel nucleo della cellula e per regolare l'attività dei geni. Se sulla coda dell'istone si attacca un fattore epigenetico (4) questo gli impedisce di srotolarsi (5) e poiché i geni avvolti non si esprimono (6), a seconda dei geni bloccati può causare o bloccare malattie come cancro, malattie autoimmuni, mentali e metaboliche (diabete).
2
1
gruppo metilico
istone
3
cromatina
cromosoma
4
fattore epigenetico
5
Dna non accessibile
coda dell'istone
6
istone
Dna accessibile

Hamada JL et al. ***Cell Phones and their Impact on Male Fertility: Fact or Fiction*** The Open Reproductive Science Journal, 2011, 5, 125-137


nascita
pubertà
morte
100
50
0
densità sinaptica (%)
10^3
10^4
10^5
giorni dopo il concepimento


vescicole sinaptiche
autorecettore
all'interno di questa vescicola sinaptica sono visibili le molecole di neurotrasmettitore
fessura sinaptica
recettori
neurone post-sinaptico

Neurochem Res. 2015 Apr;40(4):629-42. doi: 10.1007/s11064-014-1509-6. Epub 2015 Jan 13.

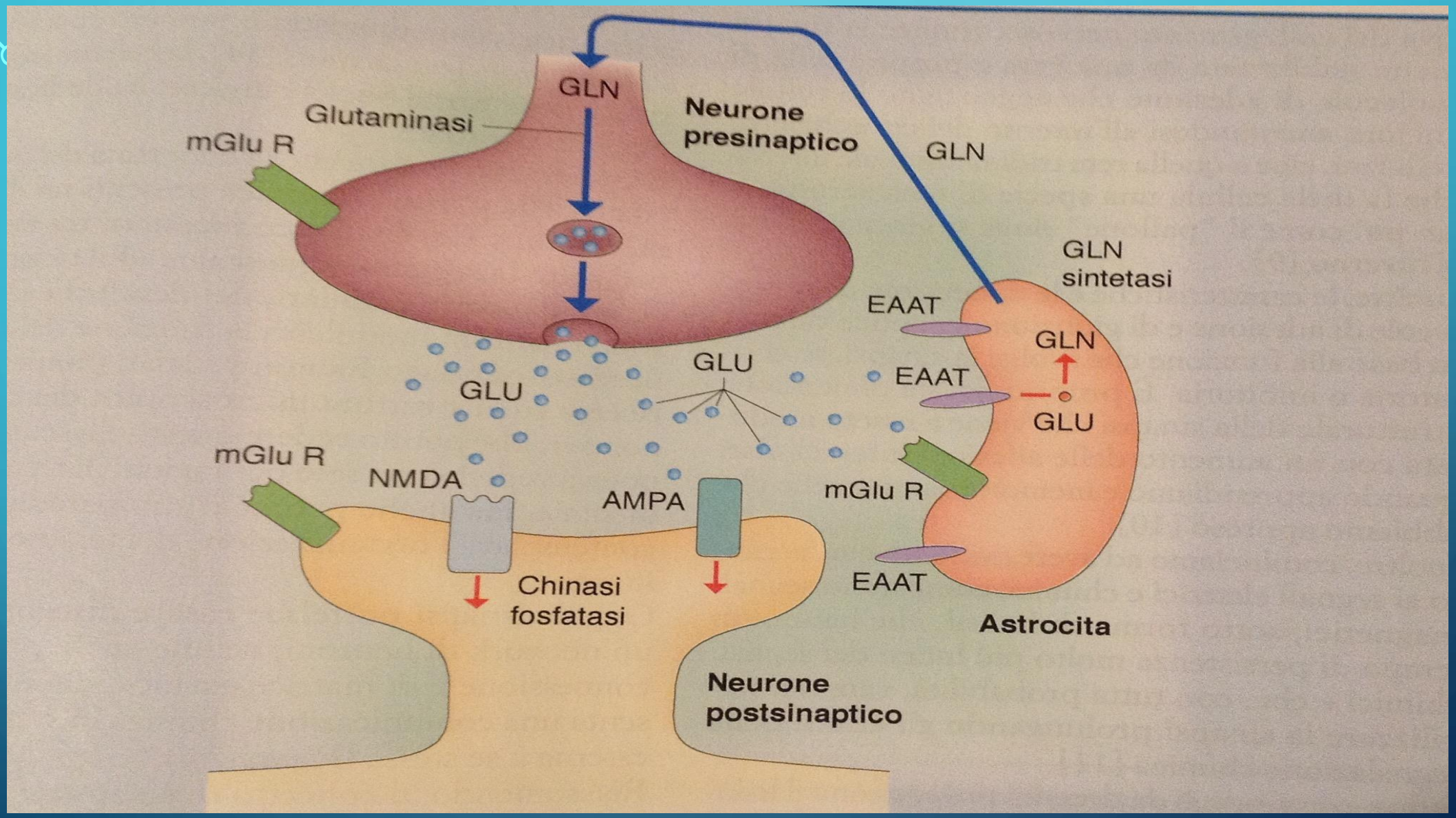
Variations of glutamate concentration within synaptic cleft in the presence of electromagnetic fields: an artificial neural networks study.

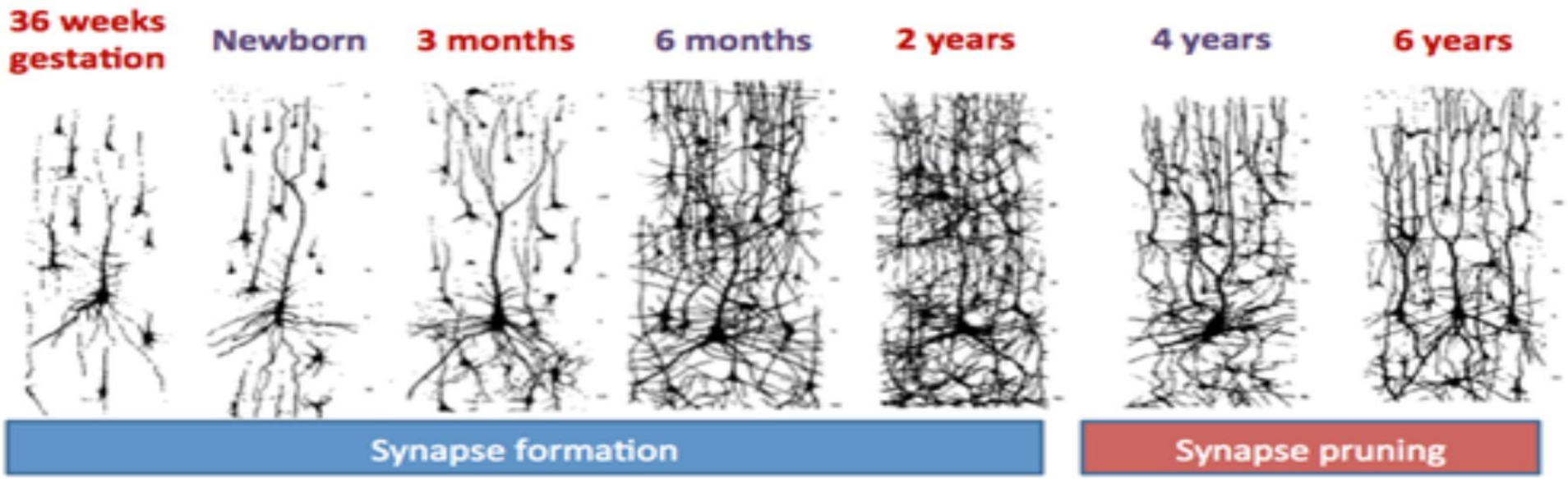
Masoudian N[1], Riazi GH, Afrasiabi A, Modaresi SM, Dadras A, Rafiei S, Yazdankhah M, Lyaghi A, Jarah M, Ahmadian S, Seidkhani H.

Author information

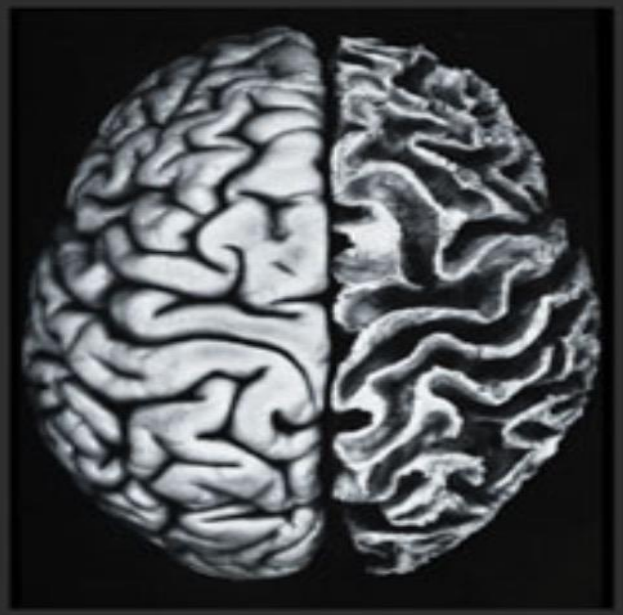
Abstract

Glutamate is an excitatory neurotransmitter that is released by the majority of central nervous system synapses and is involved in developmental processes, cognitive functions, learning and memory. Excessive elevated concentrations of Glu in synaptic cleft results in neural cell apoptosis which is called excitotoxicity causing neurodegenerative diseases. Hence, we investigated the possibility of extremely low frequency electromagnetic fields (ELF-EMF) as a risk factor which is able to change Glu concentration in synaptic clef. Synaptosomes as a model of nervous terminal were exposed to ELF-EMF for 15-55 min in flux intensity range from 0.1 to 2 mT and frequency range from 50 to 230 Hz. Finally, all raw data by INForm v4.02 software as an artificial neural network program was analyzed to predict the effect of whole mentioned range spectra. The results showed the tolerance of all effects between the ranges from -35 to +40 % compared to normal state when glutamatergic systems exposed to ELF-EMF. It indicates that glutamatergic system attempts to compensate environmental changes though release or reuptake in order to keep the system safe. Regarding to the wide range of ELF-EMF acquired in this study, the obtained outcomes have potential for developing treatments based on ELF-EMF for some neurological diseases; however, in vivo experiments on the cross linking responses between glutamatergic and cholinergic systems in the presence of ELF-EMF would be needed.

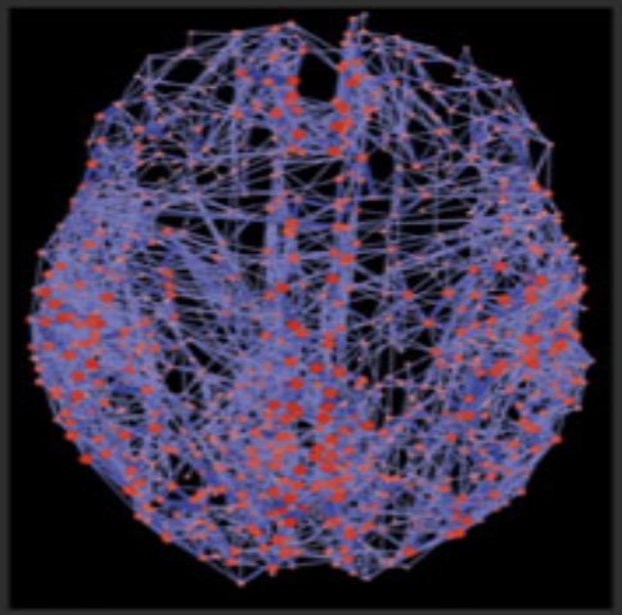

GLN
Glutaminasi
mGlu R
Neurone presinaptico
GLN
GLN sintetasi
EAAT
GLN
GLU
GLU
EAAT
GLU
mGlu R
NMDA
AMPA
mGlu R
EAAT
Chinasi fosfatasi
Astrocita
Neurone postsinaptico


36 weeks gestation
Newborn
3 months
6 months
2 years
4 years
6 years
Synapse formation
Synapse pruning

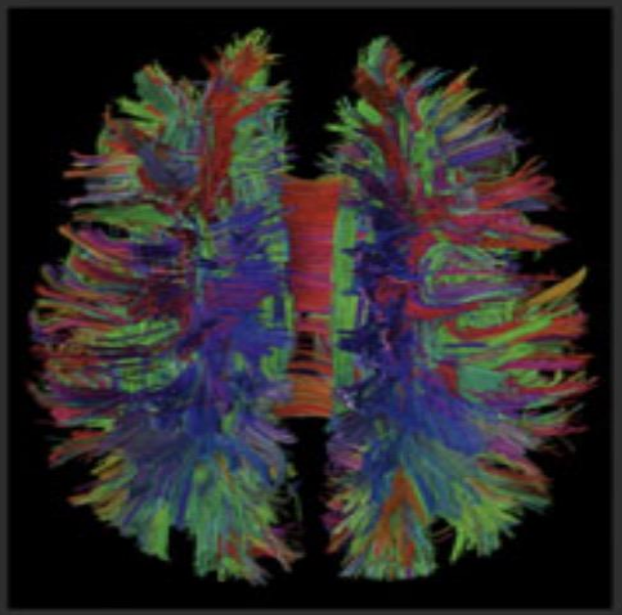
The Human Connectome



Anatomy
Klingler's method for fiber tract dissection uses freezing of brain matter to spread nerve fibers apart. Afterwards, tissue is carefully scratched away to reveal a relief-like surface in which the desired nerve tracts are naturally surrounded by their anatomical brain areas.



Connectome
Shown are the connections of brain regions together with "hubs" that connect signals among different brain areas and a central "core" or backbone of connections, which relays commands for our thoughts and behaviors.

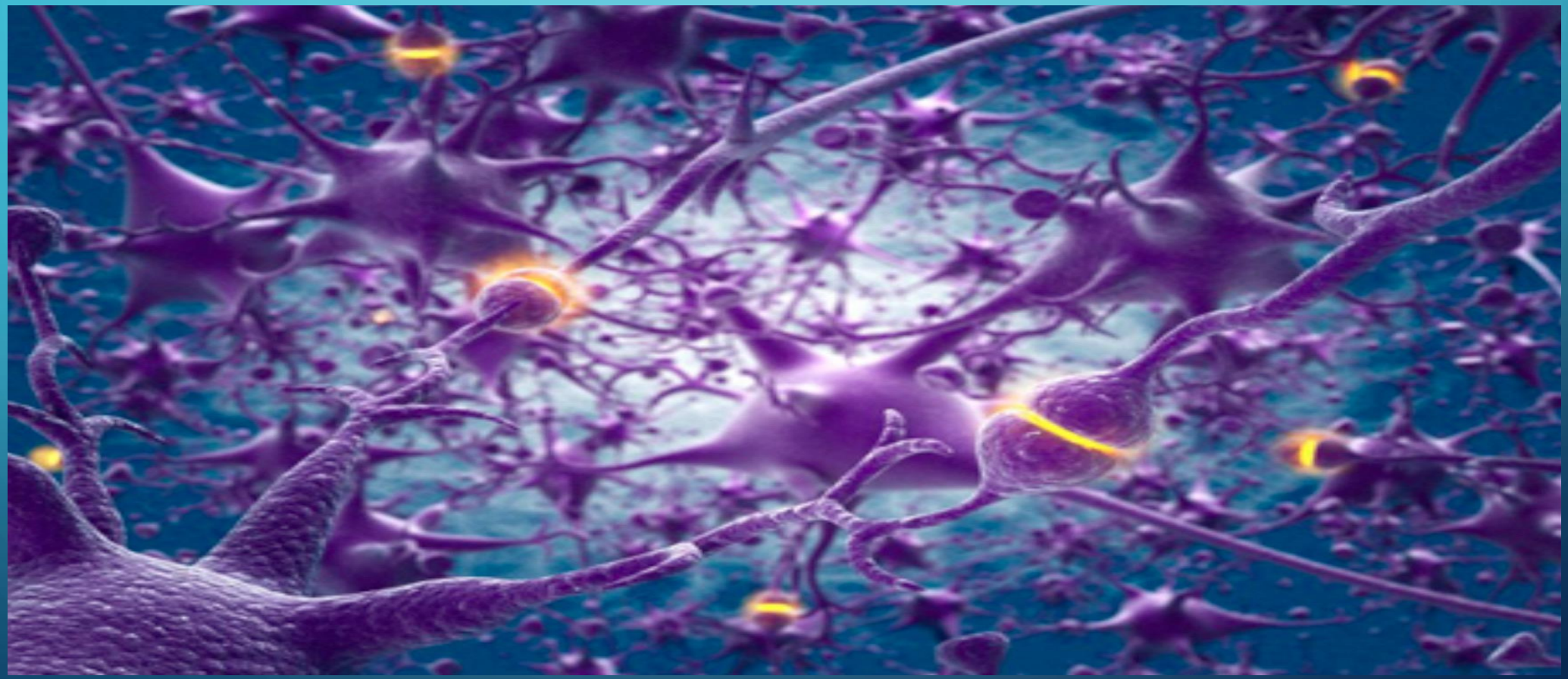


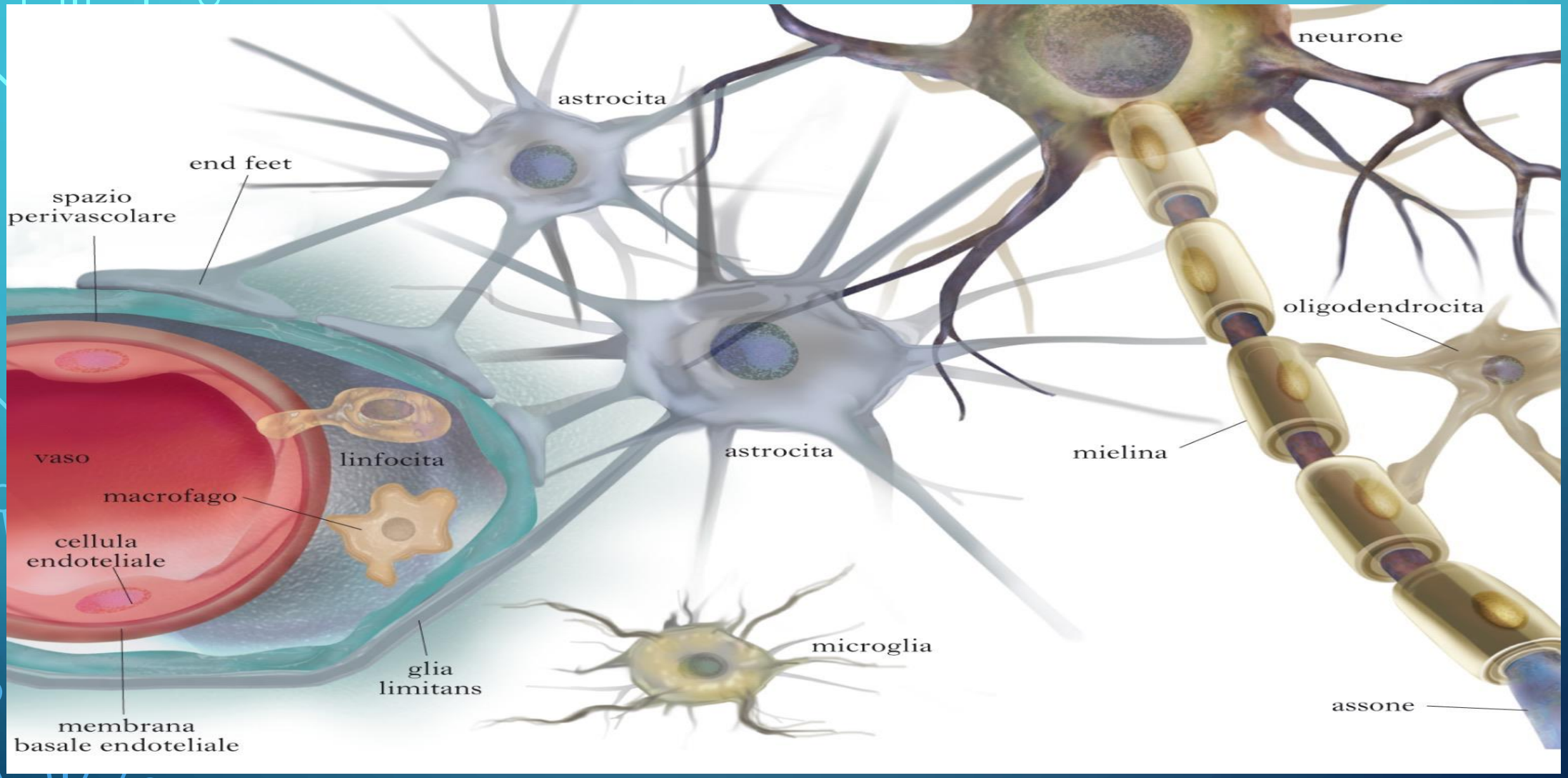
Neuronal Pathways
A new MRI technique called diffusion spectrum imaging (DSI) analyzes how water molecules move along nerve fibers. DSI can show a brain's major neuron pathways and will help neurolo-gists relate structure to function.

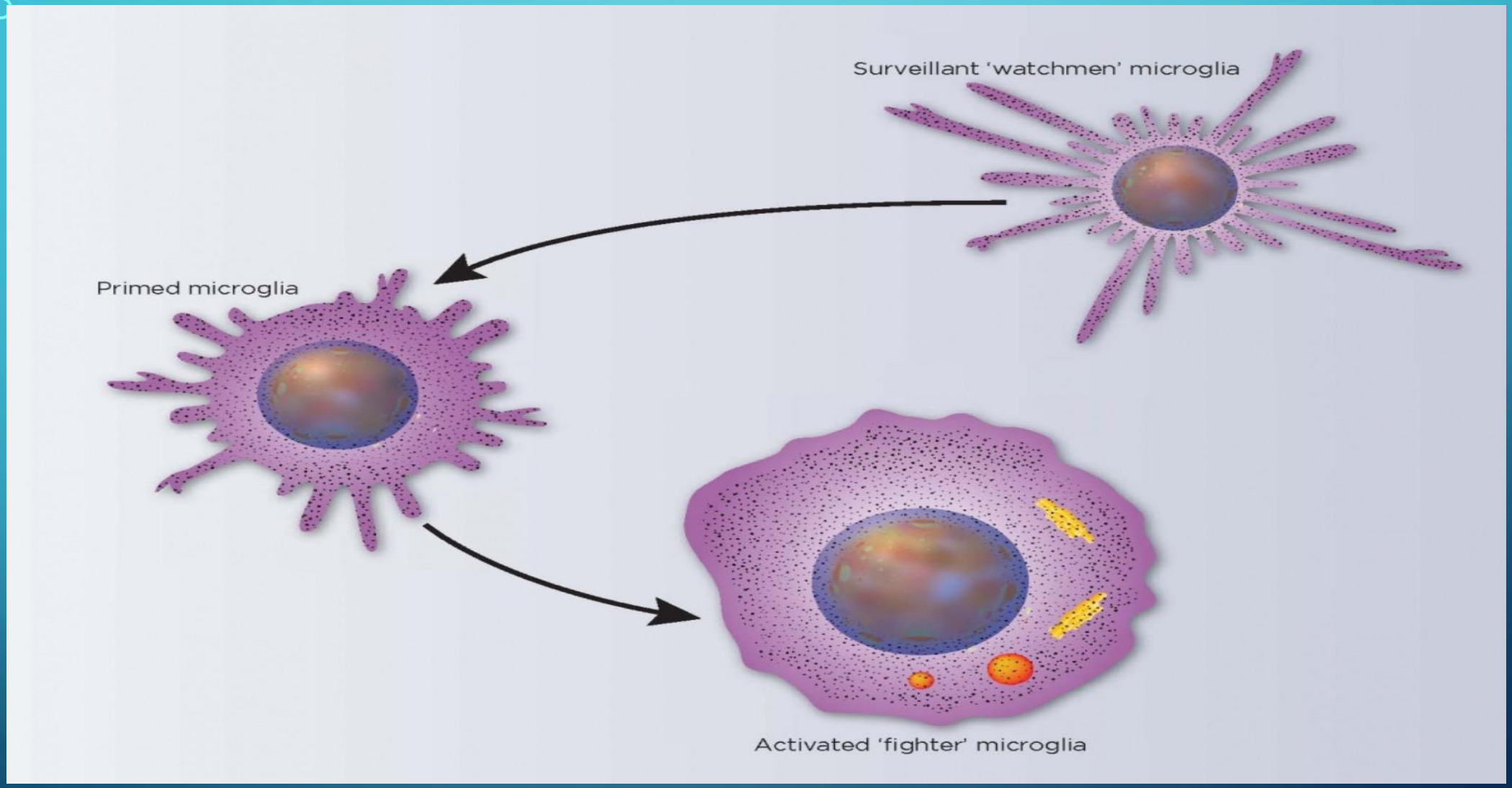
DISTURBI DEL NEUROSVILUPPO

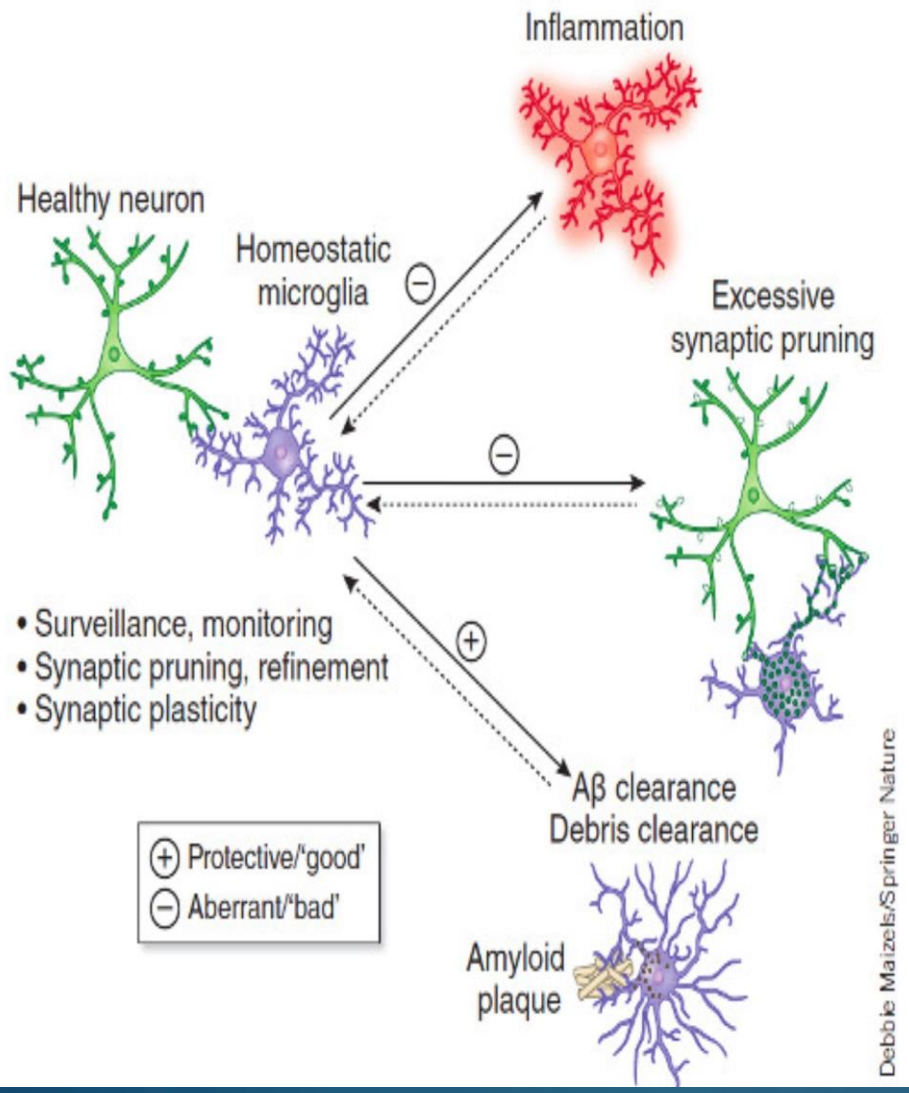
DSA, ADHD, DISLESSIA

DISTURBI DEL CONNETTOMA




neurone
astrocita
end feet
spazio
perivascolare
oligodendrocita
vaso
linfocita
astrocita
mielina
macrofago
cellula
endoteliale
microglia
glia
limitans
assone
membrana
basale endoteliale


Surveillant 'watchmen' microglia
Primed microglia
Activated 'fighter' microglia



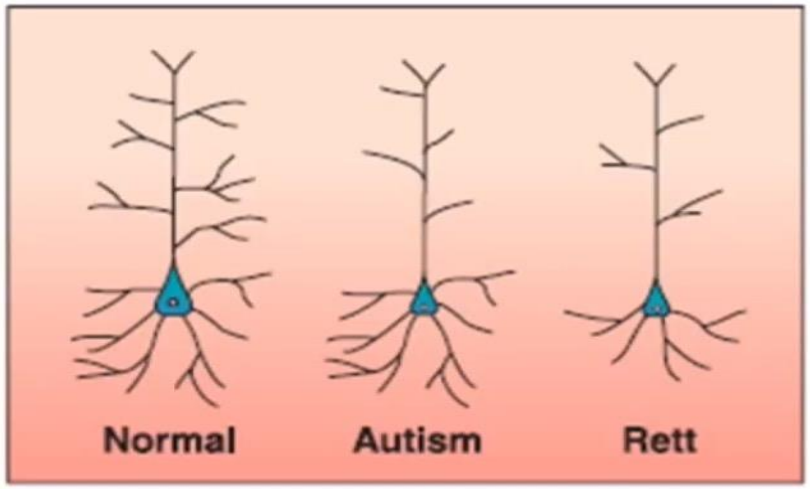
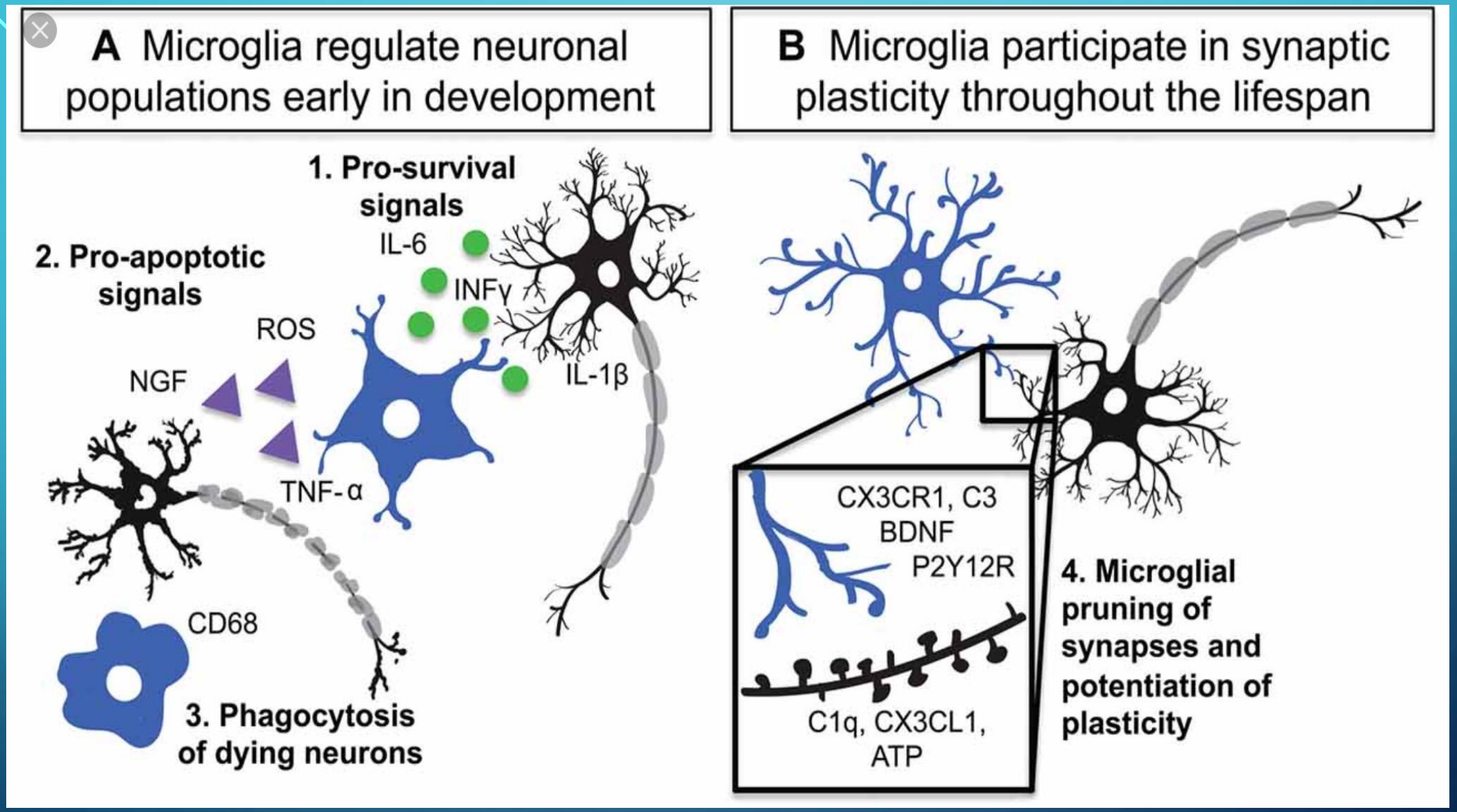


Fig. 2. Schematic representation of pyramidal neurons from control, autism, and Rett brains. In autism, the cell body is small and there is reduced dendritic branching. Similar changes occur in Rett, along with reduction in basilar dendritic branching. The reported changes are subtle and apply to a few neurons in selected brain regions in each disorder (*50, 81*).

Postnatal Neurodevelopmental Disorders: Meeting at the Synapse?
Huda Y. Zoghbi, *et al.*
Science **302**, 826 (2003);


A Microglia regulate neuronal populations early in development
B Microglia participate in synaptic plasticity throughout the lifespan
1. Pro-survival signals
IL-6
INFγ
IL-1β
2. Pro-apoptotic signals
ROS
NGF
TNF-α
CD68
3. Phagocytosis of dying neurons
CX3CR1, C3
BDNF
P2Y12R
C1q, CX3CL1,
ATP
4. Microglial pruning of synapses and potentiation of plasticity

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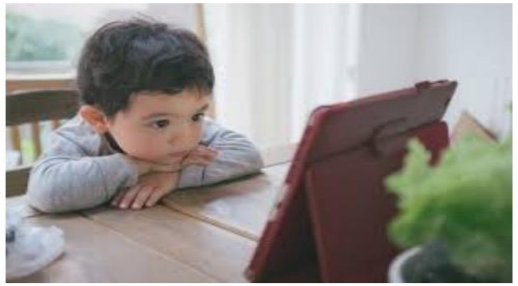

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