Neural plasticity – a gift of evolution

Katalin EGYED

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Who am I? Brain surgeon?

Why shall we (developmental/child psychologists) have to study the brain development?

- to eliminate misconceptions/false theories and to understand human development

2

It helps to eliminate misconceptions/false theories

e.g. autism

Cause: 'refrigerator mom's detached and cold behaviour caused the children's autistic behaviours.

This theory blamed the mothers

Autism Today:

Pervasive developmental disorder

We can help children with autism to develop if we take it a neurodevelopmental disorder.

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What is the difference?

Babies' world

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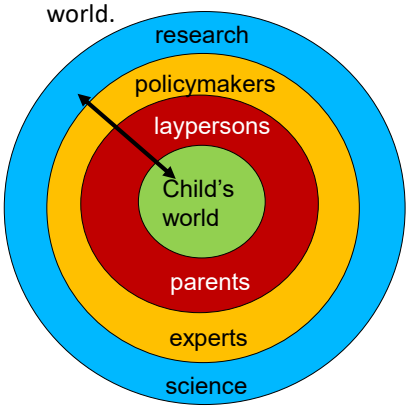
Who am I? Brain surgeon?

Why shall we (developmental/child psychologists) have to study the brain development?

- to eliminate misconceptions/false theories and to understand human development
- First, it was a basic research issue.
- Today: it is „**our business" to understand the role of plasticity** in human development
  - Experts in any services related to infants and children policymakers
  - parents, laypersons, general public

- Although we are far from understanding the brain-human development relationship perfectly,
- the current knowledge has impact on babies' world.



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Research focus

Earlier

- The role of **maturational processes vs. experience** in influencing the course of development.

Today: changes in the brain are inexorably linked to changes in the environment.

- changes in the environment, including **experience, alter the brain**

+

- **changes in the brain alter behaviour**, which in turn ➔ can **change the child's interaction** with the environment.

➔**Focus: on plasticity**, the ability and extent to which the brain can be altered

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In focus: plasticity

- Brain development is not predetermined;
- is shaped through both genetics and the environment,
- prenatally and across the life span.
- **Brain = open system** that is affected by both internal and external influences (from minute to extreme; immediately vs. over time; once or more, persistently)

Key issue for child psychologists:

➔ **The extent of plasticity** of the brain (lost and preserved plasticity in the course of development)

➔ to have some understanding of how the brain develops and functions and **what aspects of the brain can and cannot be altered through experience**. (Then: When? How?...) ➔ „opportunity to change"

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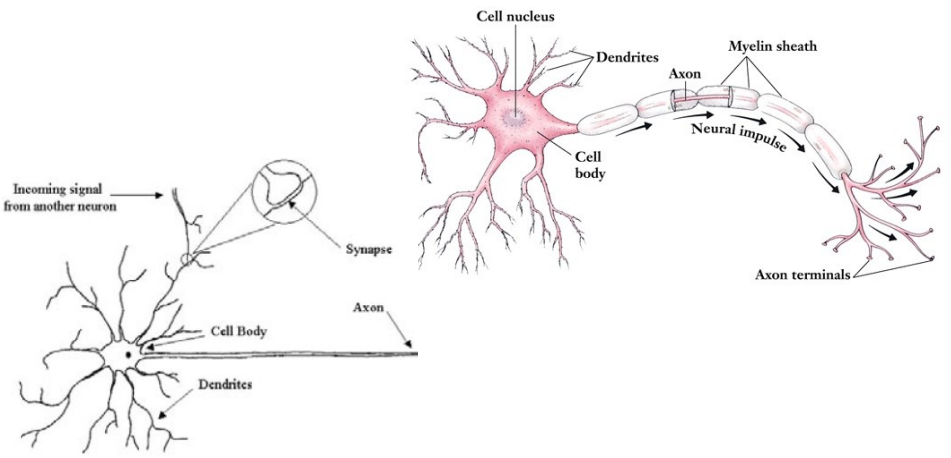
The protagonists



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Brain development

– basic processes, anatomical changes, influencing factors

What kind of processes do you know in brain development from neurogenesis?

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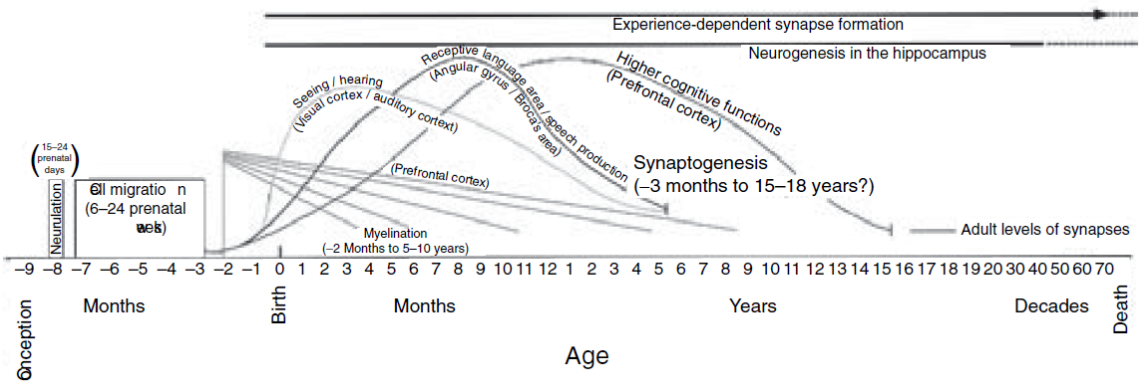
Review figure



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The Basic Building Blocks

- Neural induction (formation of ectoderm) and neurulation
- Proliferation
- Cellmigration
- Differentiation of cells
- Synaptogensis
- Regressive processes in development:
  - Apoptosis: programmed cell death
  - Pruning

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Neurogenesis

https://www.youtube.com/watch?v=U0L0mYi_ftc 5:10-13

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Neural induction and neurulation

The brain development starts

- 16 days after conception ➔ **neural induction**: from ectoderm to neural plate
- From 21/22nd day: a transformation **from a neural plate to a neural tube** ➔ **neurulation**

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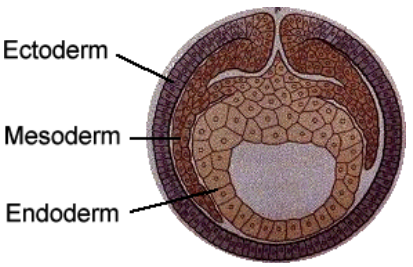
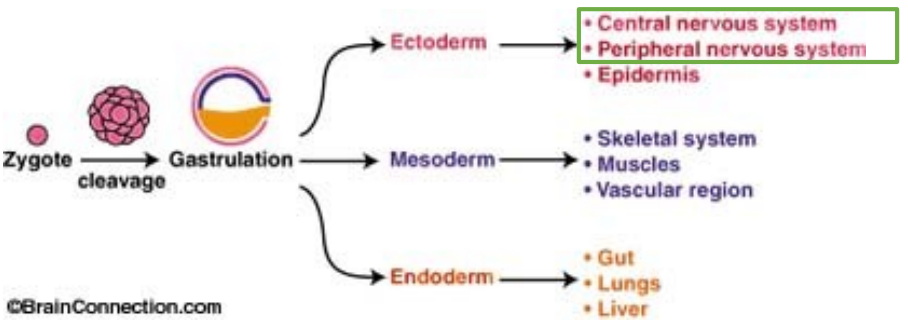


Neural induction: neural plates starts to form from the dorsal side of ectoderm.



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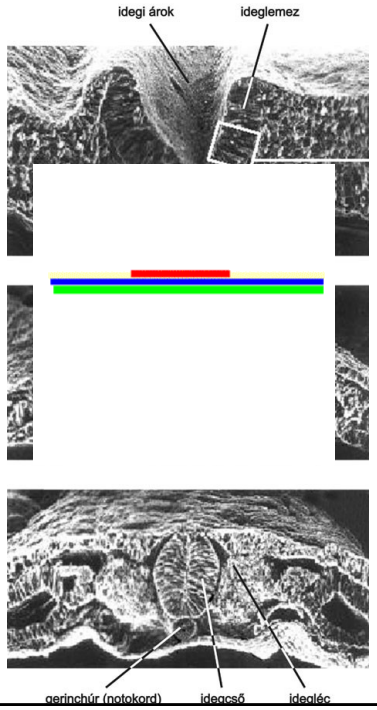
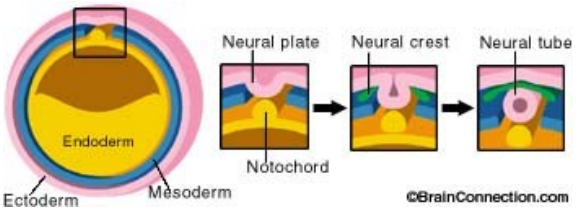


Neurulation: a transformation from a neural plate to a neural tube



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Formation of the neural tube

- Day 21/22: fusing at midsection, closing until the day 25/26
- The structure is ready to develop the brain.

➔ 1. to produce the cells (**proliferation**)

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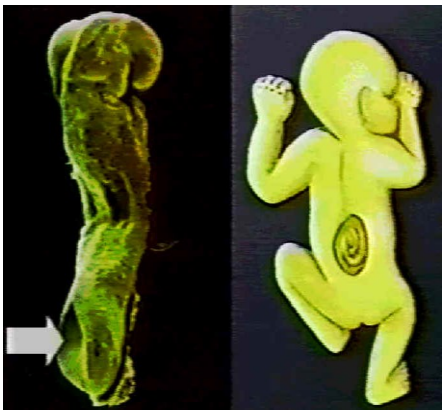
Failures in formation of the neural tube

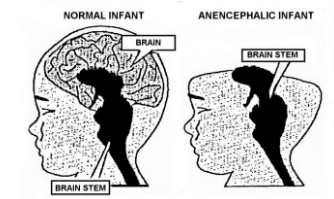
If the posterior portion fails to close

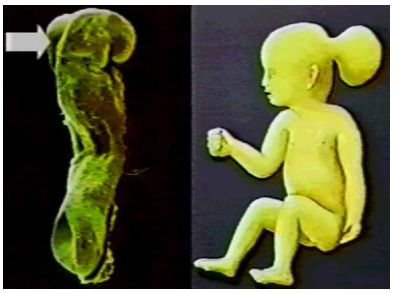
spina bifida



If the anterior

anencephaly





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Proliferation: creation of cells

The birth of cells of the cortex

- From around the day 40
- Day 40-125:
  - The critical part of normal brain development: exponential increase in the number of cells + at extremely high rate.

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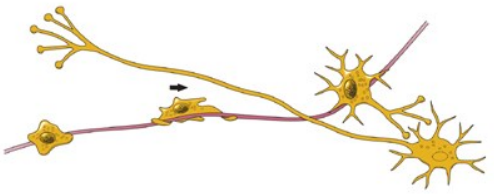
Cellmigration



After the birth of the cells **a journey** to the final home in the brain

- From about the **8th prenatal week**
- In the neocortex until 24th gest. week,
- in other areas even longer process.

- Cellproliferation II Cellmigration

http://www.youtube.com/watch?v=ZRF-gKZHINk

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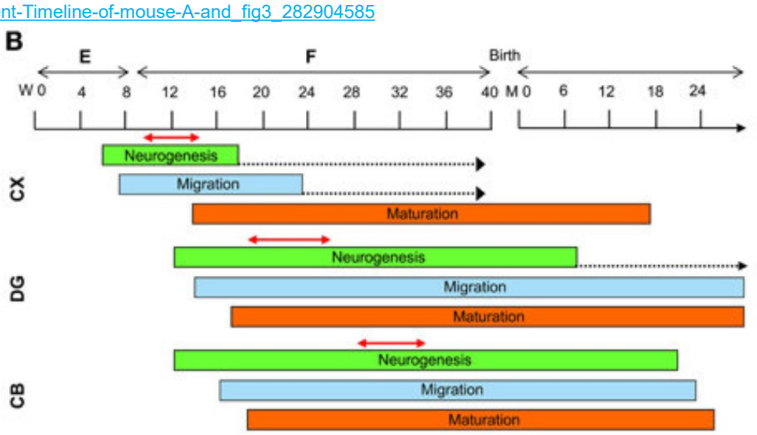
Timeline of brain development

https://www.researchgate.net/figure/Schematic-representation-of-the-timeline-of-brain-development-Timeline-of-mouse-A-and_fig3_282904585



Schematic representation of the timeline of brain development. Timeline of mouse (A) and human (B) brain development. See text for explanations. The dotted arrows indicate a reduction in the rate of neurogenesis. The double-headed red arrows delineate the period of maximum neurogenesis in the different brain regions of the mouse and human brain. Abbreviations: CB, cerebellum; CX, neocortex; DG, dentate gyrys; E, embryonic; F, fetal; M, month; P, post-natal; W, week.

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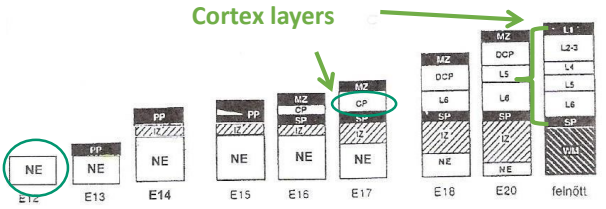
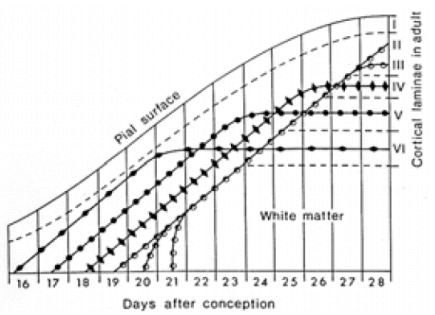
Neocortex



- Migrating neurons inside-out pattern (exce



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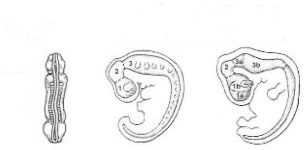
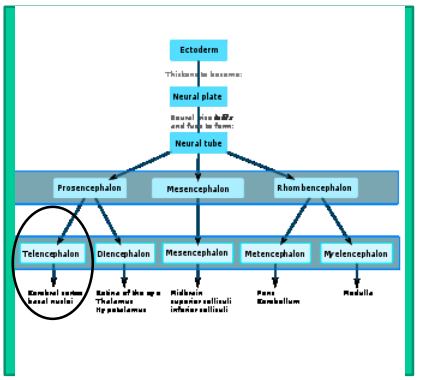
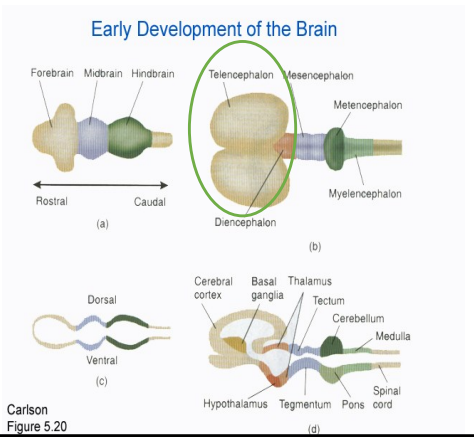
Changes ➔ 7th month: similar shape of an adult brain with 6 layers in the cortex







Carlson
Figure 5.20

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Errors in proliferation and cellmigration

Serious effect

- Errors in proliferation: microencephaly ➔ mental retardation (environmental influence: rubella, HIV, maternal alcoholism etc.)
- Errors in proliferation and/or cellmigration: autism, Down syndrome (unclear relationship)

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Differentiation of cells

- Different types of cell
- Axonal and dendritic growth
  - Development in size and complexity
  - Length of d. in the frontal lobe of adults 30x > in newborns).
  - Different rate in different layers and region
  - depends on stimuli
- Axonal connections (see synaptogenesis, myelination, pruning)

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Errors in axonal and dendritic growth

- Cause: e.g. Genetic, lack of oxygen, damage
- Effect: e.g. learning disability, mental retardation, disorder of movement
- Axonal and dendritic growth continue to some extent after birth ➔ leads to greater plasticity. However, the ability of axons to re-grow is limited.

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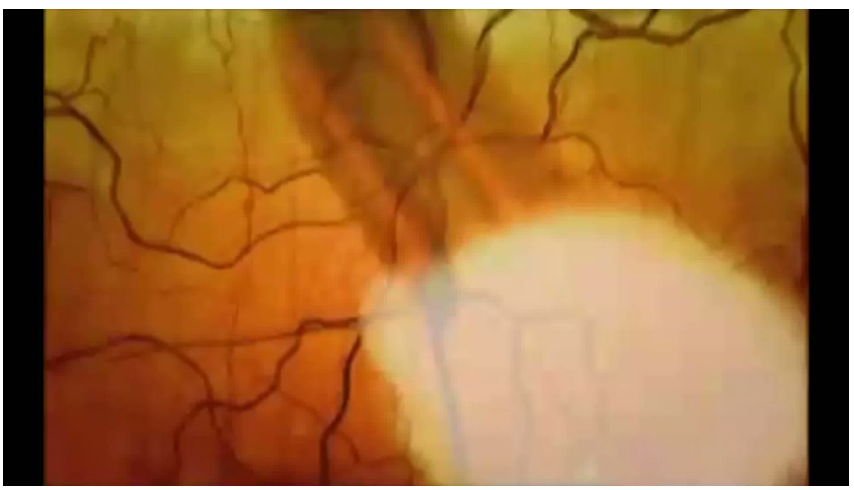
Connections

https://www.youtube.com/watch?v=U0L0mYi_ftc 13



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Synaptogenesis (SG)

- Basis for communication within the brain
- Develop from first weeks to life span
- From the 20th week SG in more and more regions
- Different areas of the brain reach their **synaptic peak at different times**

➔ **overproduction**

- Genetic programming and neural activity trigger development and maturation synapses.

→ **Synapses**: axon-dendrite, axon-axon, dendrite-dendrite, axon- body of neuron

- **First mature** syn. around **23rd week**

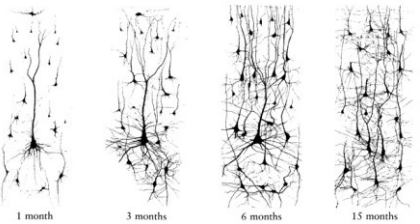

Fig. *First 15 months – increasing number of synapses*

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Errors in synaptogenesis

- Several disorders: e.g. Down, SCH
- Abnormal, lack of, overproduction of synapses
- Secondary effect of errors in axonal and dendritic growth
- Unclear relationship

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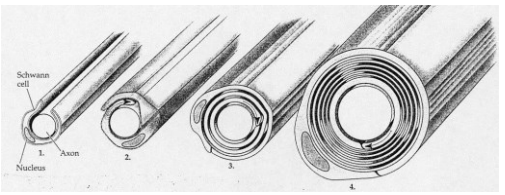
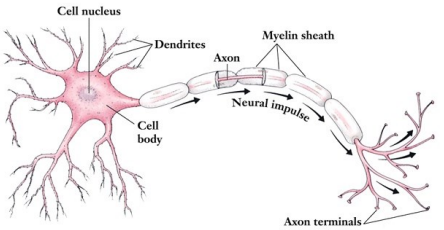
Myelination

by surrounding and **insulating the axons** ➔

- **Increasing speed and efficiency of communication**
- Transferring energy to neurons
- Starts before birth and **goes on after birth**.
- Occurs in **different areas** and reach maturity at **different times**.
- Myelination and other data related to **neural development associate/correlated with development**: e.g. vision, motor development earlier, higher cognitive function later.





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Errors in myelination

- Mental retardation, disorders of movement and sensory system
- e.g. PKU

https://www.youtube.com/watch?v=mBNRuNsDJKU

https://www.youtube.com/watch?v=Ytk5gSRQ54M

A patient: https://www.youtube.com/watch?v=Pk3PN4BHGFI

Mother, babies, PKU: https://www.youtube.com/watch?v=w3L2SPj7alQ

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Regressive processes: Programmed cell death (apoptosis)

- **Large overproduction** of neurons
- A **natural cause of cell death**: in almost all regions of the neural system 30-70% of the cells die due to apoptosis.
  - In proliferation and cellmigration: because of errors in proliferation and eliminating transient areas
  - Changing connections during differentiations of cells and synaptogenesis.

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Errors in apoptosis

- Mental retardation, emotional and behavioural disorders.
- E.g. Down: increased activity in apoptosis ➔ less neurons, less synapses…
- Proliferation is not turned off ➔ macrocephaly ➔ mental retardation, seizures

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Regressive processes: pruning

- Overproduction of neurons and synapses significantly contribute to plasticity.
- synaptic proliferation **II** Pruning ➔ **„Less is more"**
- The process of pruning is **selective**.
- **No** significant **difference** between the **size** of the brain in children and adolescents; however, the **difference is significant in connections**.

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Regressive processes: pruning

- 1. overproduction of connections. 2. pruning
- Pruning: start in the **first year lasts until adolescence**
- - 40% less synapses
- **Different timing in different areas**
- E.g. hearing until 12 y; higher cognitive function even later

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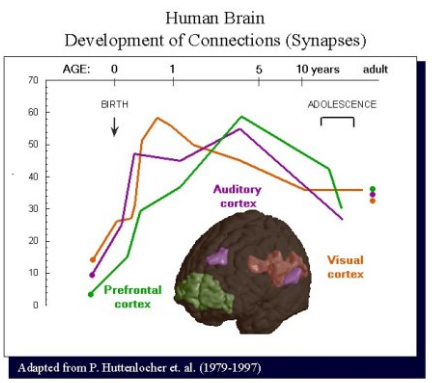
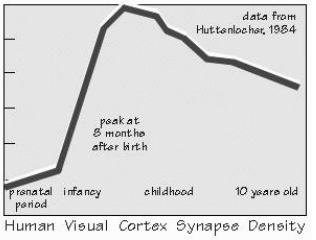
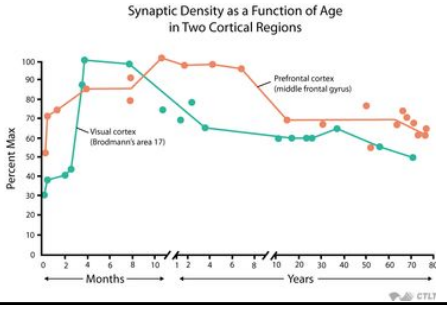
Different areas – different synaptic peaks and timing in pruning







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Errors in pruning

- E.g. SCH
- Fewer disorders have been linked to pruning errors versus errors in synaptogenesis.

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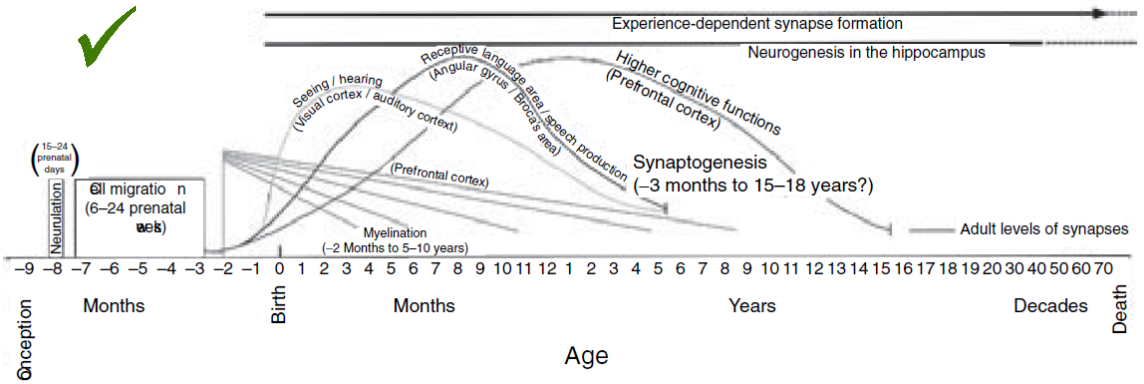
Review figure



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Experience vs. Pre-programmed brain development

https://www.youtube.com/watch?v=U0L0mYi_ftc 25-29min

depends on the experience

- Development depends on the individual environment
- This would provide more flexibility in the adaptation to individual environment

?

preprogrammed

- would provide **evolutionary advantage** of **quick adaptation**
- Arguments – e.g. language acquisition:
  - Quickly, effortless development
  - Milestones, error patterns – similar in typical development
  - Independent from culture

    …
- **Disadvantage**: less flexible adaptation

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Mechanisms of plasticity

We are evolved to have processes in brain development that are

- 1. **highly gene-driven** (like neurogenesis, proliferation, and migration)
  - primarily prenatally
  - less plastic
- 2. **primarily environment-driven** and depends on the interaction with the environment (like synaptogenesis and pruning which are involved in communication within the brain).
  - Two paths through which it occurs: 1. experience-**expectant**, 2. **experience-dependent plasticity**

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Experience-expectant plasticity and sensitive periods

(Greenough et al., 1987)

- It is **useless to suppose a pre-programmed brain** if the necessary stimulus (conditions) are in the environment („genetically less expensive") ➔instead: the **brain expects a species-typical experience** ➔ in order to develop normally, the developing brain requires specific environmental input (**no/low risk** because the expected environment is assumed to be **common to all members of species**).
- Additionally, it is necessary for this input to occur during **a window of time ➔ sensitive periods**
- The **primary mechanism**: overproductions of synapses and pruning
- The combination of environmental and genetic mechanisms that occurs in experience-expectant plasticity results in change that brain is **less plastic later** in development.
- The consequence of not-preprogrammed brain: **If** the normal **expected** experience is **not present** (and it is unknown) ➔ **atypical development**➔ relatively stable, not changeable

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Experience-dependent plasticity

- The brain's adaptation to information in the environment that is **unique to individuals.**
- **Primary mechanisms**: ongoing synaptogenesis and pruning
- Occur throughout the **lifespan** (learning)
- Depends on prior development

- **EE and ED** do not occur in isolation; rather **they interact** across development.

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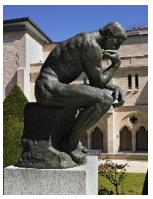
Preterm babies

Als, H.

https://www.youtube.com/watch?v=U0L0mYi_ftc&list=PLq-WhlcqSobf5JdW5wM-zT_iFBGjI9UHE 3- , 16:30

https://www.youtube.com/watch?v=VFx6k80wxOI 1:20-



NIDCAP

means

Neonatal Individualized Developmental Care Assessment Program

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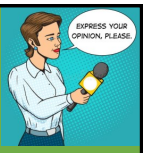
NIDCAP (13p) https://www.youtube.com/watch?v=VFx6k80wxOI



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NIDCAP

- Task-oriented to relationship-based
- Being based upon staff schedules to following the infant's rhythm
- Technology focused to person or process focused
- Action and reaction oriented to reflection based
- Crisis oriented to developmentally oriented
- Focus on organ by subspecialty to whole person, holistic (mind, body and spirit) oriented
- Deficit repair to strength-based nurturing
- ICU environment to family environment
- "Doing to" becomes "engaging with"

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Experience-expectant plasticity
– in which functions?
What does the brain expect?
Input - easy to assure

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When the brain is expecting in vain…

e.g. when the baby
cannot see ➔ baby with cataract
cannot hear ➔ deaf baby
no individualised caring ➔ early years in institutions

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Vision

e.g. if the baby

- cannot see (cataract) ➔ consequences?

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Experience-expectant plasticity – Vision (acuity)

https://www.youtube.com/watch?v=U0L0mYi_ftc 36-47

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Visual development

- **Newborn**'s visual acuity: short sighted (40x worse than adult vision in preferential looking paradigm)
- **First six months**: quick development (8x worse) (motion, depth, integration of parts of faces and objects)
- **4-6y**: slower development
- **Acuity at 6-7y: like adults**
- Some aspects **develop further** (adolescence – face, motion)

➔ What is the role of experience?

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Visual plasticity, sensitive periods in visual development

(Maurer & Lewis, 2001, 2013; Lewis & Maurer, 2005)

Investigated groups:

- Normal developing
- Cataract – at birth, with time
- One eye, both eyes
- Surgery: 1 week – 1 year

The effect of deprivation:

- yes/no
- timing: start, duration

…

- Development at once and quickly after birth BUT: **no development without experience** (cataract).
- **Sleeper effect**: from 2y worse vision than normal.
- Unilateral: worse because of the **competition** ➔ cover is needed
- **Cover is more effective if**:
  - Several years, several hours a day
  - Surgery before 6-8m
- The later the deprivation occurs, the better development
- **Deprivation** has **effect** even **from 7 to 10y too.**
- After 10y: no effect of deprivation.

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Visual plasticity, sensitive periods in visual development

(Maurer & Lewis, 2001, 2013; Lewis & Maurer, 2005)

3 sensitive periods for visual acuity:

- Normal development **driven by visual input**: 10 days **- 7y**
- System **can be damaged** by the absence of visual input: **10y**
- System **can recover** from earlier deprivation:
  - Relatively long, **surgery at 12y might improve vision** that influences the quality of life
  - **Strong limitations**

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Conclusion?

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When the developing brain expects experience – and is waiting in vain

- If no language because of extreme neglect: e.g. Genie

- **Deafness** in a hearing-speaking environment, deprived language input ➔ serious effect on language acquisition

Genie: https://www.youtube.com/watch?v=VjZolHCrC8E 8-

Tommy:

https://www.youtube.com/watch?v=SCzl4kuWLw0

2- beginning

10 choice; 14 poor speech

30-33: Tommy cannot speak, his parents do not understand him, and he does not understand them

https://www.youtube.com/watch?v=XhceEJ1R_vY&t=94s 3:57-hearing parents 8:02 deaf father

https://www.youtube.com/watch?v=B7FK0Ni6W1g&t=864s Sound and Fury

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When the brain expects individual child-carer relationship

- Psychosocial deprivation ➔ the effect of insitutionalized care

Nelson:

https://www.youtube.com/watch?v=ptZ-L3uAXXA

https://www.youtube.com/watch?v=Hqh47no-IRw

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Charles NELSON & BEIP (5m)

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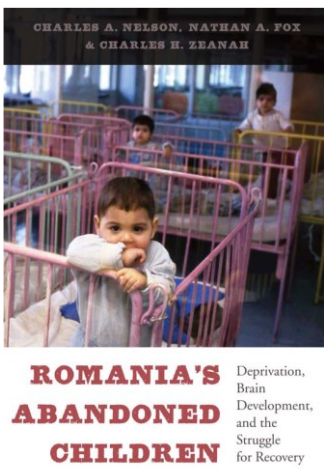
Bucharest Early Intervention Project (BEIP)

- Long suspected:
  - early life in an orphanage could have adverse consequences.
  - Better upbringing in foster care as an alternative form (or in inst.?)

  ➔ Empirical studies: selection bias
- Romanian government supported the project
- Many experts in child protection system in Romania believed that inst. was better (⇔ e.g. U.S. foster care system)



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Bucharest Early Intervention Project (BEIP)

- **Goal**: to investigate the effect of institut.zed care (timing, sensitive periods)
- Launched **in 2000**, in cooperation with Romanian government
- **Firs-ever randomized controlled** study (regarding this effect)
- Relationship between the brain and behaviour, development
- Results ➔ could be convincing case for reform, shaping the practice
- 6 institutions, 136 children
- No neurological, genetic disorder, perinat. damage
- To orphanage at 22m (average) (range: few weeks – 31m)

Groups

- 50% in institutions (**care as usual**)
- 50% in **foster care** (53 families, 68 children) + program for the families (training, salary, encourage to commitment).
- **Never** been inst.zed
- High ethical standards
- After 8 years 68/15 children were in inst.

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Bucharest Early Intervention Project (BEIP)

- The effect of early instituionalized care ➔ Biological, neurological, psychological, behavioural level, long-term effects
- Sensitive periods? ➔ In which domains?

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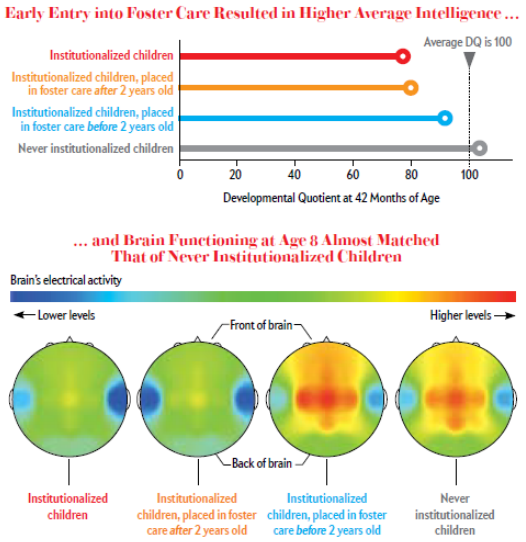

IQ (see next slide):

- 30m-8y: differences in each assessment
- Placement:
  - before **2y** IQ is higher than after;
  - 8y: no timing effect (long-term experience could overwrite;
  - long-term group diff.s

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Table 12.1 Timing effects for foster care placement evident in assessments at 42 months, 54 months, and 8 years

	42 months		54 months		8 years	
	Intervention effects	Timing effects	Intervention effects	Timing effects	Intervention effects	Timing effects
EEG power	no	no	n/a	n/a	yes	yes
IQ	yes	yes	yes	yes	yes	no
Language	yes	yes	n/a	n/a	yes	yes
Stereotypies	yes	no	yes	yes	n/a	n/a
Social skills	n/a	n/a	n/a	n/a	yes	yes
Selective attachment	yes	yes	n/a	n/a	n/a	n/a
Height and weight	yes	yes	n/a	n/a	n/a	n/a

Sleeper effect

Timing effect disappeared

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Stress effect

- Bio.cal **toll of stress** (telomere length: a mark of accelerated cellular aging): those who had spent **any time in an institution** had **shorter telomeres** than those who had not.

Language

- Delays in language acquisition
- Placement before 15-16m: language is normal.
- the later children were placed, the further behind they fell

Brain power

- EEG activity: **delayed brain maturation**.
- **8y**: placed in foster care **before 2y could not be distinguished** from that of those who had **never** passed time **in an institution**; **after 2y = never left** the inst.
- **Inst. group**: MRI – large reduction volume of grey and white matter, **smaller brain** volume
- **No sensitive period in grey matter**
- **White matter** volume: foster care **>** inst.

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Attachment (see the details: Gervai Judit's lecture)

- Early assessment: overwhelming majority in **inst.**: incompletely formed and aberrant relationships with their caregivers
- 42m:
  - In foster care: dramatic improvement – almost 50% secure
  - Inst.: **18% secure**
  - Control: 65%
- **Placement of foster care before 24m: more likely to form secure attachment.**

Mental health problems

- ever been in institution: at **1.5y: 53%** psych.tric diagn.; **only in family: 20%**
- 5y: Inst.: **62% diagn.**: 44% anxiety, depression, 23% ADHD
- Anxiety, depression – reducing incidence in family; ADHD, conduct disorder – no
- sensitive periods – not detectable
- **The quality of relationship**: the better quality, secure attachment between a child and foster parent, the greater probability that the child's symptoms would diminish.

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Bucharest Early Intervention Project (BEIP)

To explore:

- the effect of early insitutionalized care
- sensitive periods
- several aspects/functions

- Deprivation ➔ serious and persistent differences between the groups: biological, neurological, psychological development.
- in family ➔ an opportunity to improve, normal range
- sensitive periods in many domains but not all
- sleeper effects

➔ **policy**: in family – the sooner the better

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According to **Rutter**:

- If **genes** specify **only** the **rudiments** of the brain-to-be, the **experience** will bear **responsibility for** the major proportion of **development**
- ➔ Institutional environment is inadequate and in some cases damaging (a very extreme form of neglect)
- ➔ **experience-adaptive programming**: a behaviour that is abnormal outside the institution, is adaptive within the institution, environment (hugging a complete stranger).

The families could provide for the child's **expectable needs**: The children were

- talked to
- played with
- held when needed
- engaged with
- encouraged to master the world
- respected
- loved

➔ Some of the things our species has come to expect

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Neural plasticity
– the gift of evolution is a two-edged weapon

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Does matter:

- Experience (several aspects)
- The timing of (lack of) experience

➔ Effect, consequences: on biological, neurological, psychological, behavioural level

➔ Understanding the opportunities and limitations of plasticity ➔ can make different experts' work more efficient.

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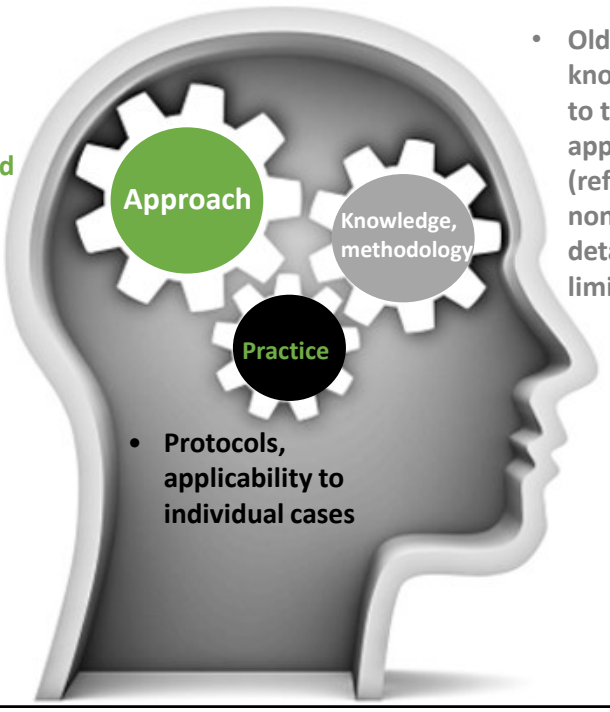
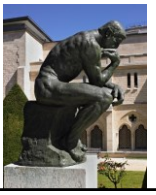
- **Relationship between the human development and brain plasticity**



- **Old and new knowledge fit to the approach (reflection on non-fitting details and limitations)**

- **Protocols, applicability to individual cases**



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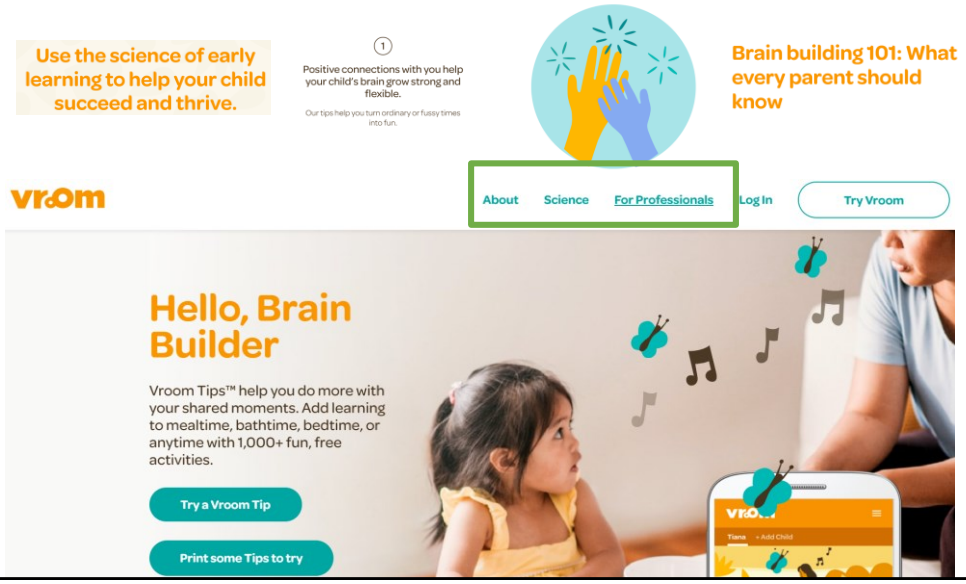
For parents

www.vroom.org
https://www.zerotothree.org/espanol/brain-development



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For parents

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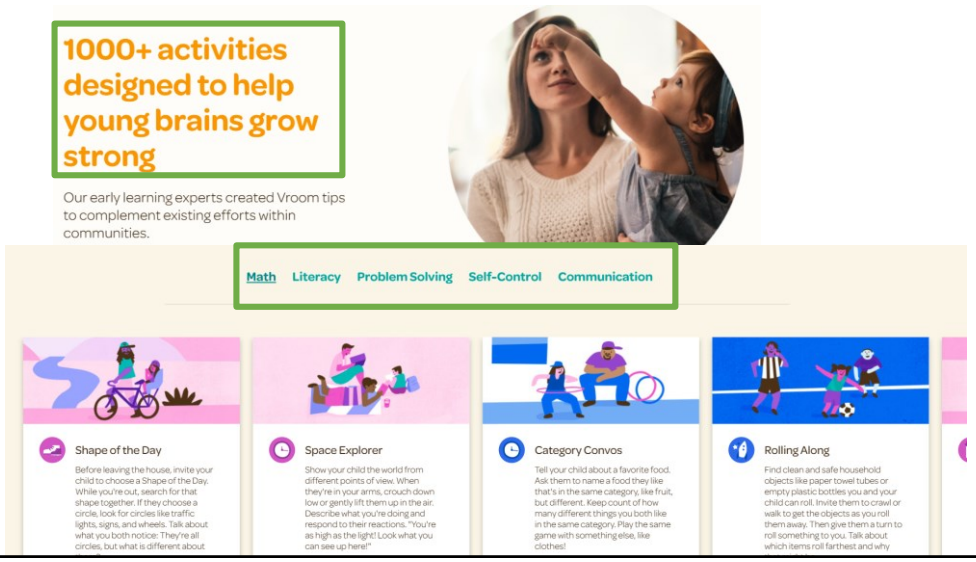

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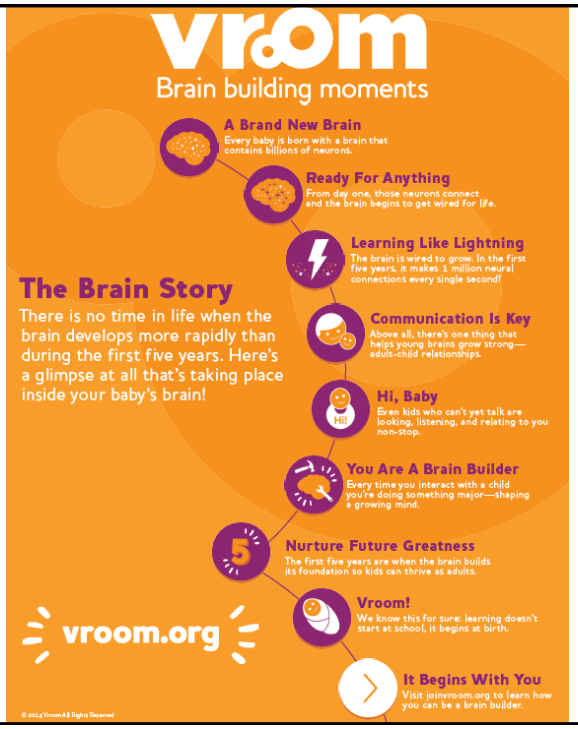
For parents

So simple
Vroom, 2014



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Readings

- **Couperus, J.W. & Nelson, C.A**. (2006). Early Brain Development and Plasticity In: McCartney, K. & Phillips, D. (eds.) The Blackwell -Handbook of Early Childhood Development. Malden, MA: Blackwell Publishing, 86-104 – **compulsory**
- **Shonkoff, J. P. and PhilFrom, D. A.** (2000): Neurons to Neighbourhoods: The Science of Early Childhood Development. NATIONAL ACADEMY PRESS, Washington, D.C. Chapter 8: The Developing Brain. 182-219 – **compulsory**
- Useful: The Secret Life of the Brain https://www.youtube.com/watch?v=U0L0mYi_ftc

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