NEUR 1520 Neuroscience Study Guide for Exam 2: Lecture 14: Synapse Formation and Rearrangement

Sources: the lecture slides and The Mind's Machine, Chapter 4. Nothing else.

Each heading is one book learning objective. [L13 s08] = Lecture 13, slide 8. [MM p124] = book page 124.

(teal italics) = plain meaning of the word before it. ⚠ **Do not mix up = a look-alike pair to keep apart.** **Amber dotted words** = names printed on the figure beside them.

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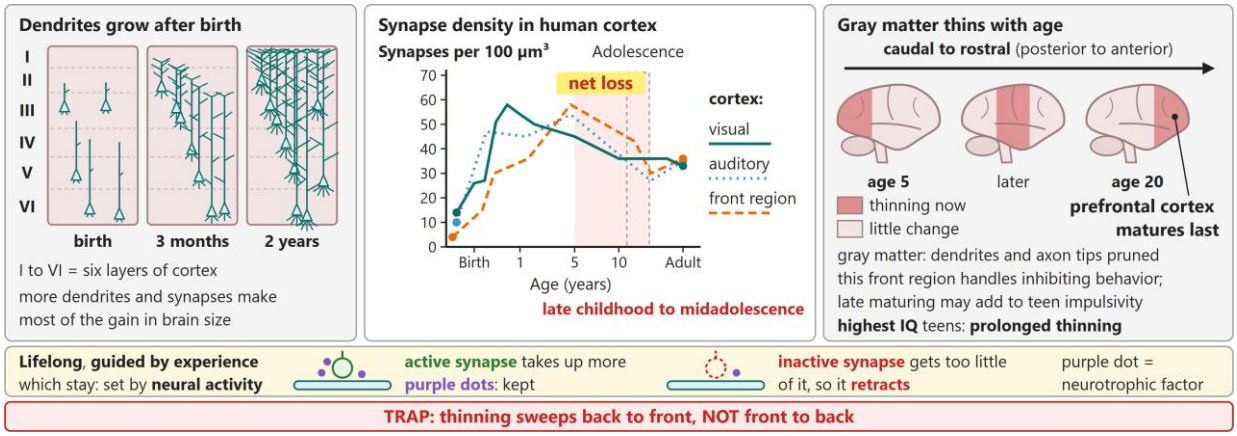
4.2.1 Describe the process of synapse rearrangement, and offer evidence that a net loss of synapses may be adaptive

⚠ **Do not mix up: More synapses is NOT better: fragile X brains keep too many immature spines.** [L14 s08, MM p131]

Review: the six pictured stages are neurogenesis, migration, differentiation, synaptogenesis, cell death, rearrangement. [L14 s02, L14 s03]

Before and after birth, cortical neurons grow longer, more elaborate dendrites jammed with synapses. [MM p129]

This massive **increase** in dendrites and synapses causes most of the brain size **increase** after birth. [MM p129]



Cortex dendrites grow after birth, synapse density rises then falls, gray matter thins back to front [MM p130]

On the figure: six layers · net loss · auditory · gray matter · caudal to rostral · prefrontal cortex · neural activity · neurotrophic factor

Cortex at birth, **3** months, and **2 years** shows dendrites expanding; mature cortex has **six layers**. [MM p129]

Synapse rearrangement (**synaptic remodeling** *(pruning and rebuilding synapses)*): loss of some synapses, development of others. [MM p129]

Synapse rearrangement typically takes place after the period of cell death. [MM p129]

About half of the spinal motor neurons that form later die. [MM p129]

Spinal motor neurons are motor neurons of the **spinal cord** *(CNS tube in the back)*. [MM p129]

Synapse rearrangement continues throughout adulthood, guided by experience. [L14 s05]

After cell death, each motor neuron innervates many muscle fibers; each fiber gets several neurons. [MM p129]

Later, motor neurons retract axon collaterals until each fiber has **only** one motor neuron. [MM p129]

Which synapses stay: competition for trophic factors and/or between **Hebbian synapses** *(synapses strengthened by success)*. [MM p129]

Similar rearrangement occurs in cerebellum, brainstem, visual cortex, and autonomic nervous system. [MM p130]

Human cortex: **net loss** of synapses from late childhood until midadolescence. [L14 s05, MM p130]

Synapse density in visual, **auditory**, **prefrontal cortex** rises after birth, then declines. [L14 s06, MM p130]

Synapse density declines after the first year of life. [L14 s06, MM p130]

Prefrontal cortex synapse density peaks later than visual cortex and keeps falling through adolescence. [L14 s06]

Remodeling shows as thinning of cortical **gray matter** *(surface layer of cortex)* as dendrites and terminals are pruned. [L14 s07, MM p130]

Thinning proceeds **caudal to rostral** *(back to front, posterior to anterior)*, so prefrontal cortex matures last. [L14 s05, L14 s07, MM p130]

Prefrontal cortex is important for inhibiting behavior. [MM p130]

Late prefrontal maturation may contribute to teenagers' impulsivity and relative lack of control. [L14 s05, MM p130]

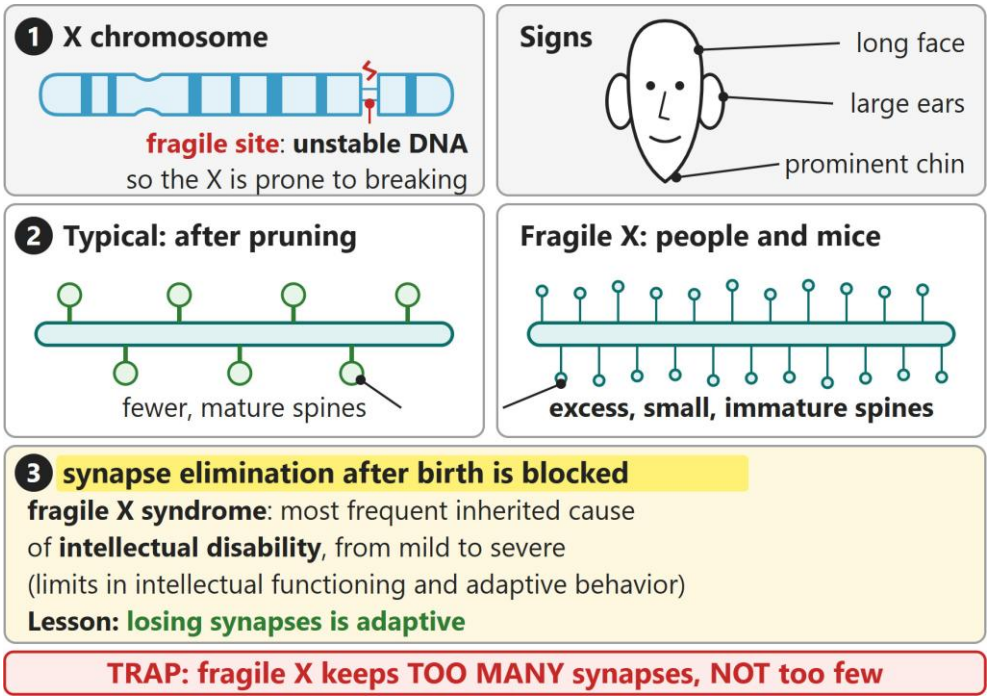
Neural activity is one important influence on which synapses are kept. [MM p130]

Theory: active synapses take up **neurotrophic factor** that keeps them; inactive ones get too little. [MM p130]

Intellectual stimulation probably helps: highest-IQ teenagers show especially prolonged cortical thinning. [MM p130]

Genetic disorders can have a profound effect on the developing brain. [L14 s08]

Intellectual disability: significant limits in intellectual functioning and adaptive behavior. [MM p130]



Fragile X: unstable X site, typical signs, and excess immature spines [MM p131]

On the figure: fragile X syndrome · intellectual disability · fragile site · prominent chin · immature

Fragile X syndrome: most frequent inherited cause of **intellectual disability**, mild to severe. [L14 s08, MM p130, MM p131]

Cause: a **fragile site** *(unstable DNA spot)* makes the X chromosome prone to breaking. [MM p130, MM p131]

Fragile X appearance: elongated face, large prominent ears, **prominent chin**. [MM p131]

Humans and mice with fragile X: cortical neurons have excess small, **immature** **dendritic spines** *(tiny dendrite bumps holding synapses)*. [L14 s08, MM p131]

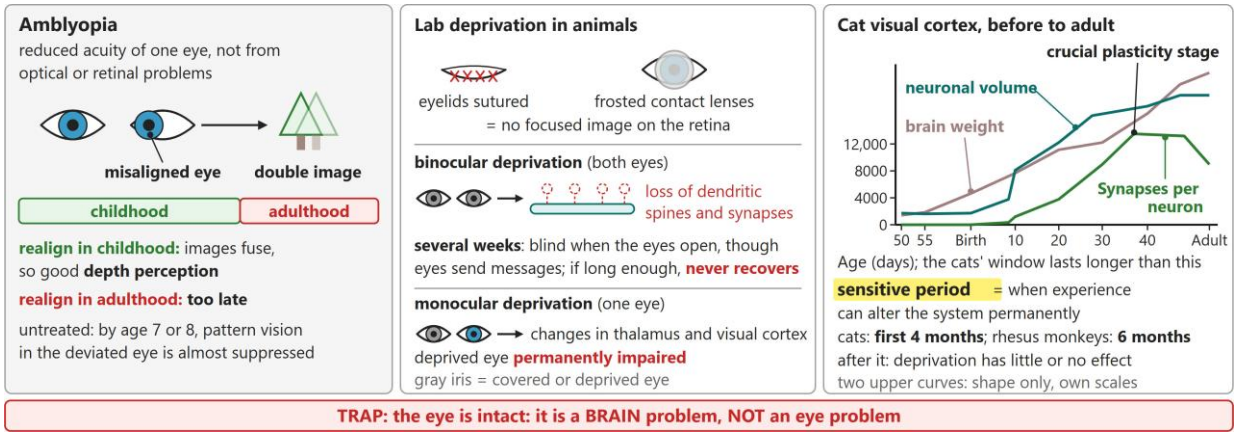
Suggests fragile X blocks the normal elimination of synapses after birth. [L14 s08, MM p131]

Evidence net loss is adaptive: losing synapses helps the brain function better. [MM p130, MM p131]

Touch cortex rearranges synapses throughout life; learning strengthens, weakens, or rearranges synapses. [MM p131]

4.2.2 Describe studies showing that visual experience early in life is required to develop normal vision

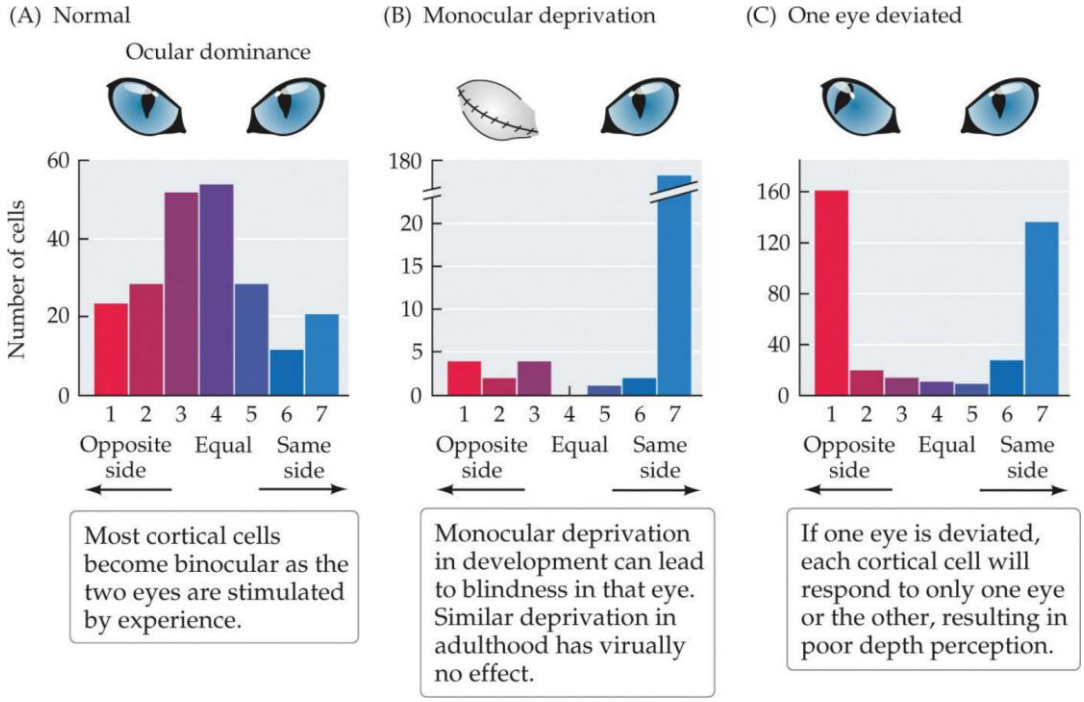
⚠ Do not mix up: Monocular deprivation in adulthood has virtually NO effect: only the sensitive period matters.
[L14 s14, MM p132, MM p133]



Amblyopia, animal deprivation methods, and the cat synapse curve with its sensitive period [MM p131]

On the figure: amblyopia · double image · binocular deprivation · monocular deprivation · sensitive period · synapses per neuron

Amblyopia *(dull vision in one eye)*: reduced acuity in one eye, not from optical or retinal damage. [L14 s09, MM p131]
Amblyopia comes from the Greek amblys, dull, and ops, eye. [L14 s09, MM p131]
Amblyopia: the eye is intact and a sharp image focuses on the retina. [MM p131]
Some people with amblyopia have one eye misaligned: turned inward (cross-eyed) or outward. [L14 s09, MM p131]
Children born misaligned see a **double image**, not a single fused image. [MM p131]
Untreated, by age **7** or **8,** pattern vision in the deviated eye is almost completely suppressed. [MM p131]
Realigned in childhood: the two images fuse and depth perception is good. [MM p131]
Not realigned in childhood: visual acuity in the turned eye cannot be reestablished. [L14 s09, MM p131]
Realignment in adulthood is **too late** to restore acute vision to the turned eye. [MM p131]
Visual deprivation experiments in animals provide the data on amblyopia. [L14 s10, MM p131]
Methods: eyelids reversibly sutured shut, or frosted contact lenses, prevent focused images on the retina. [MM p131]
Binocular deprivation *(blocking sight in both eyes)*: loss of dendritic spines and fewer synapses in visual cortex. [L14 s10, MM p131]
Binocular deprivation for several weeks in development: when eyes open, the animal is blind. [MM p131]
Light enters and the eyes send messages, but the brain seems to ignore them. [MM p131]
If deprivation lasts long enough, sight can never be restored. [L14 s10, MM p131]
Sensitive period *(window when experience causes lasting change)*: when experience or treatment permanently alters the organism. [L14 s10, MM p131]
Effects are biggest during early synapse development in visual cortex; afterward little or none. [MM p131]
Cat visual cortex: **synapses per neuron** climb steeply after birth, peak, then drop by adulthood. [L14 s11, MM p131]
Cat brain weight and neuron volume keep rising while synapses per neuron later fall. [L14 s11, MM p131]
Experience decides which synapses are kept: a crucial stage of **neural plasticity** *(brain change with experience)*. [MM p131]
Monocular deprivation *(blocking light to one eye)*: in the sensitive period, the deprived eye later fails to respond. [L14 s12, MM p132]
Monocular deprivation changes structure and function in the thalamus and visual cortex. [MM p132]



Ocular dominance histograms: normal, one eye covered, and one eye deviated [L14 s14]

On the figure: ocular dominance · number of cells · monocular deprivation · binocular · depth perception

Ocular dominance histogram *(graph of which eye drives each neuron)*: neuron response strength to left versus right eye. [L14 s12, MM p132]

Histogram classes: **1 only** opposite eye, **4** both eyes equally, **7 only** same-side eye. [L14 s14, MM p132]

Ocular dominance histogram axes: x is eye class **1** to **7,** y is **number of cells**. [MM p132]

Normally most cortical neurons, except layer IV, respond equally to either eye. [L14 s12, MM p132]

Normal: most cortical cells become **binocular** as both eyes are stimulated by experience. [L14 s14, MM p132]

Monocular deprivation: most cortical neurons respond **only** to the non-deprived eye. [L14 s13, MM p132, MM p133]

Similar deprivation in adulthood has virtually no effect. [L14 s14, MM p132]

Sensitive period for this effect: cats, first **4** months; rhesus monkeys, to age **6** months. [MM p133]

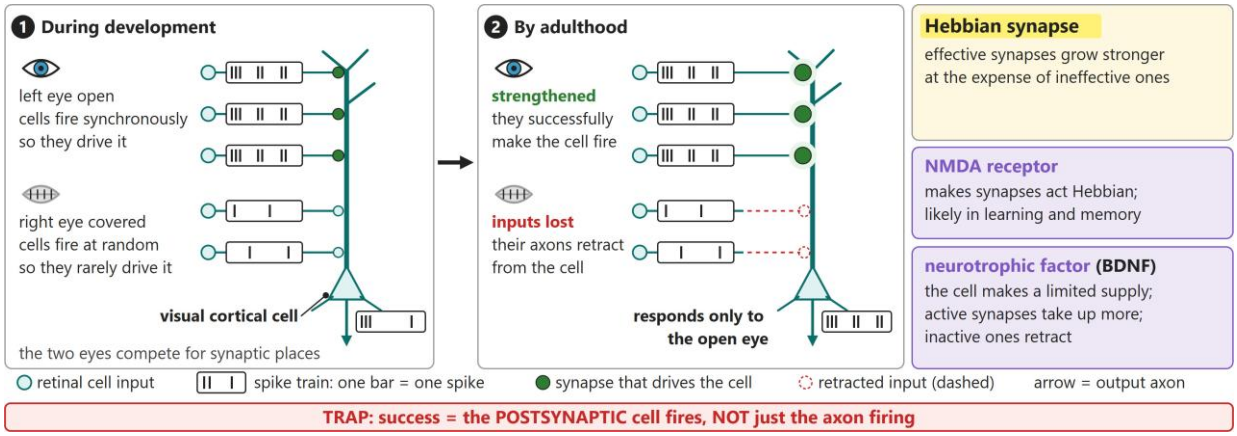
One eye deviated: each cortical cell usually responds to one eye **only**; poor **depth perception**. [L14 s13, L14 s14, MM p132]

Kittens with eyes surgically diverged: normal binocular response of cortical cells greatly reduced. [MM p133]

Why: misaligned eyes no longer give simultaneous, convergent input to visual cortex cells. [MM p133]

Axons from each eye compete for synaptic places *(targets)*; active synapses predominate. [L14 s15, MM p133]

If one eye is silenced, its synapses retract; the other eye's synapses are maintained. [MM p133]



Hebbian synapses: inputs that drive the cell grow stronger, ineffective inputs retract [MM p132]

On the figure: Hebbian synapse · NMDA receptor · neurotrophic factor · BDNF · strengthened

Hebbian synapse *(synapse strengthened by success)*: **strengthened** when it successfully drives the postsynaptic cell. [L14 s15, MM p133]

Hebbian synapses grow stronger or weaker depending on ability to drive the target cell. [L14 s15, MM p133]

Effective synapses grow stronger at the expense of ineffective synapses. [MM p133]

1. Open eye: neighboring retinal cells fire synchronously and drive the postsynaptic neuron. [L14 s16, MM p132]
2. Covered eye: cells fire at random and rarely make the postsynaptic neuron fire. [MM p132]
3. Effective inputs strengthen; ineffective inputs are lost. [L14 s16, MM p132]
4. By adulthood, the cortical cell responds **only** to signals from the open eye. [MM p132]

The **NMDA receptor** *(a glutamate receptor)* makes synapses act Hebbian; likely involved in learning and memory. [MM p133]

Neurotrophic factors may play a role: active synapses take up more of the factor. [L14 s15, MM p133]

Postsynaptic cells make limited factor, so inactive axons may retract for lack of it. [MM p133]

BDNF *(brain-derived neurotrophic factor)* is implicated as the factor competed for in visual cortex. [MM p133]

Babies with **cataracts** *(cloudy lenses)* removed a few months after birth have good vision. [MM p133]

Cataracts removed in adulthood: vision comes slowly and **only** to a limited extent. [MM p133]

Cataracts blocking vision just the first **6** months: face recognition impaired **9 years** later. [MM p133]

Early visual experience is especially crucial for learning to perceive faces. [MM p133]

These effects probably come from synapse rearrangement in visual cortex, as in kittens. [MM p133]

Michael May: blinded at age **3** by chemical explosion; right eye sight restored at **46.** [MM p121]

Images focused properly on the retina, yet vision stayed poor. [MM p121]

Years later, Michael May could not recognize a face, even the wife's, unless the person spoke. [MM p121]

Could not recognize **3**D objects like cubes or spheres unless they were moving. [MM p121]

Skiing: had to close the eyes; seeing was more distracting than helpful. [MM p121]

Had the accident happened in adulthood, surgery would have restored normal vision. [L14 s17, MM p133]

Cause: deprived of form vision from childhood for about **40 years**. [L14 s17, MM p133]

Visual cortex synapses were not strengthened by patterns of light moving across the retina. [L14 s17, MM p133]

Without patterned stimulation, eye-to-brain synapses languished and disappeared. [L14 s17, MM p133]

Normal form vision for the first **3.5 years** may have kept some **residual synapses**, allowing some sight. [MM p133, MM p134]

Soon after surgery, objects recognized under **30%** of the time; no significant gain **10 years** later. [MM p134]

After more than a decade of adult sight, still trouble with **3**D objects and faces. [MM p134]

Other people who first gain sight as adults have similar trouble with objects and faces. [MM p134]

Binocular vs monocular deprivation

	Binocular deprivation	Monocular deprivation
Eyes deprived	Both eyes	One eye
Cortex change	Fewer spines and synapses	Most cells answer **only** the open eye
Vision result	Blind if long enough	Deprived eye permanently impaired

Table source: [L14 s10, L14 s12, L14 s13, MM p131, MM p132]

Ocular dominance histograms

Condition	Histogram	Vision result
Normal	Most cells answer both eyes	Binocular cells
Monocular deprivation	Most cells answer open eye **only**	Deprived eye blind
One eye deviated	Each cell answers one eye	Poor depth perception

Table source: [L14 s12, L14 s13, L14 s14, MM p132, MM p133]