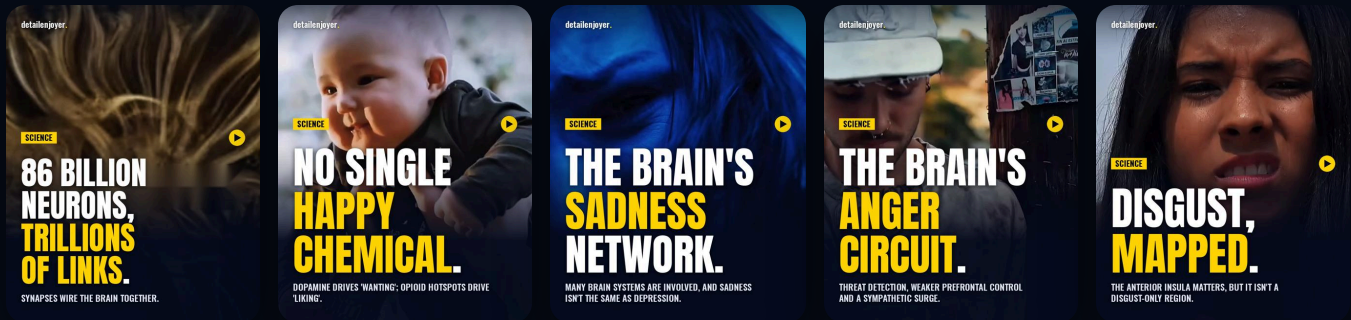




COLLECTION · COL-13

THE BRAIN

Wiring, neurochemistry and emotion: the connections and chemistry behind how you think and feel.



INSIDE

- 01 Connected
- 02 Happiness
- 03 Sadness
- 04 Anger
- 05 Disgust
- 06 Surprise

- 07 Fear
- 08 SSRIs
- 09 Synaptic Transmission
- 10 Seems Familiar
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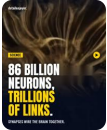
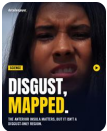
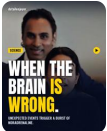
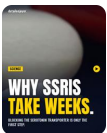
DID YOU KNOW?

11 case files · 22 quiz questions · by DetailEnjoyer

ABOUT THIS COLLECTION

11 case files on how the brain is wired and the circuits and chemicals behind your feelings: its roughly 86 billion neurons and their trillions of connections, then happiness, sadness, anger, disgust, surprise and fear, one at a time, plus the antidepressants that act on serotonin. The locus coeruleus is the brain's main source of noradrenaline. **DD-747** SSRIs work by inhibiting the serotonin transporter, SERT. **DD-749**

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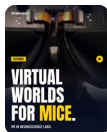
09



Synaptic Transmission

DD-275 · Science / People · Neurons pass signals across a gap called the synaptic cleft.

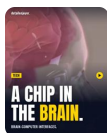
10



Seems Familiar

DD-429 · Science · Scientists build virtual worlds for mice running on treadmills.

11



Neuralink

DD-527 · Tech / Science · Neuralink implants a coin-sized chip with thin threads into the brain.



Collection Quiz and Answer Key

22 questions from every case file

Every quick fact has a stable ID (for example DD-635-F01) and comes from a case file with its sources listed. Online edition with videos and slides: <https://detailenjoyer.com/collections/the-brain/>



DD-635 SCIENCE · 1 OF 11

CONNECTED

The '86 billion neurons' figure comes from a 2009 study by Frederico Azevedo, Suzana Herculano-Houzel and colleagues. They dissolved the brains of four adult men into a uniform 'soup' of cell nuclei and counted samples. They found about 86.1 billion neurons (give or take 8.1 billion), replacing the round 100 billion long quoted in textbooks. Most of those neurons aren't in the wrinkled cortex. About 69 billion are in the cerebellum, and about 16 billion are in the cerebral cortex. Neurons pass signals to each other at synapses. Synapse counts per neuron vary enormously by cell type, so '1,000 to 10,000 per neuron' is only a rough rule of thumb. Whole-brain totals are estimates too. A 2003 stereology study by Bente Pakkenberg and colleagues put the neocortex alone at about 0.15 quadrillion (150 trillion) synapses, which fits within the Reel's range of 100 to 1,000 trillion. No one has counted every synapse in a human brain, so the totals are estimates. The footage is golden, branching animation of neurons.

QUICK FACTS

DD-635-F01

The human brain has about 86 billion neurons.

DD-635-F02

About 69 billion neurons are in the cerebellum.

THE WRITE-UP

Where 86 billion comes from. A 2009 study counted about 86.1 billion neurons in adult male brains, replacing the old 100 billion figure.

Brain soup. The team dissolved brain tissue into a uniform suspension of nuclei and counted samples.

Mostly in the cerebellum. About 69 billion neurons are in the cerebellum and about 16 billion in the cerebral cortex.

Counting synapses. The neocortex alone holds an estimated 150 trillion synapses.

Rough numbers. Synapses per neuron vary hugely by cell type, so whole-brain totals are estimates, not counts.

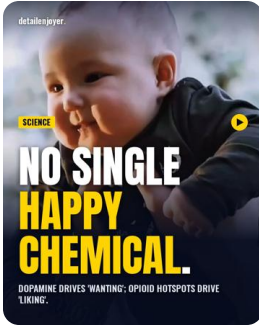
REEL VS. RECORD

- **Note:** 'Approximately 86 billion': correct, but it is a 2009 estimate (86.1 ± 8.1 billion) from the brains of four adult men, not an exact count.
- **Note:** 'Each connecting... 1,000 to 10,000 synapses per neuron': synapse counts vary enormously by cell type, so this is a rough rule of thumb, not a range every neuron falls within.
- **Note:** '100 to 1,000 trillion': a reasonable range of estimates. The neocortex alone has been estimated at about 150 trillion synapses (2003).
- **Missing:** most neurons, about 69 billion, are in the cerebellum, not the cerebral cortex (about 16 billion).
- **Note:** 'Vast processing power': synapse counts alone don't measure what a brain can do. How the connections are organized and how strong they are matters too.

SOURCES

1. Azevedo et al. (2009): Equal numbers of neuronal and nonneuronal cells make the human brain an isometrically scaled-up primate brain, J. Comp. Neurol. (PubMed): <https://pubmed.ncbi.nlm.nih.gov/19226510/>
2. Herculano-Houzel (2009): The human brain in numbers: a linearly scaled-up primate brain, Frontiers in Human Neuroscience (PMC): <https://pmc.ncbi.nlm.nih.gov/articles/PMC2776484/>
3. Pakkenberg et al. (2003): Aging and the human neocortex, Experimental Gerontology (PubMed): <https://pubmed.ncbi.nlm.nih.gov/12543266/>

Permalink: <https://detailenjoyer.com/case/connected/> · Last updated Aug 12, 2025



DD-743 SCIENCE · 2 OF 11

HAPPINESS

Research by Kent Berridge and Morten Kringelbach separates 'wanting' (incentive motivation, in which dopamine plays a major part) from 'liking' (hedonic pleasure). Liking depends on small hedonic hotspots, such as parts of the nucleus accumbens and ventral pallidum, where opioid and endocannabinoid signals amplify pleasure. Raising dopamine can increase wanting without increasing liking. The researchers also distinguish moment-to-moment pleasure from broader happiness, which includes a sense of meaning.

QUICK FACTS

DD-743-F01

Dopamine drives 'wanting' more than 'liking'.

DD-743-F02

Opioid and endocannabinoid signals in small hotspots amplify pleasure.

THE WRITE-UP

No single chemical. Berridge and Kringelbach describe pleasure as arising from a distributed network of brain systems, not from one molecule.

Wanting versus liking. Dopamine is mainly linked to incentive salience ('wanting') and reward learning, not to pleasure itself. Raising dopamine can increase desire without increasing 'liking'.

Hedonic hotspots. 'Liking' reactions depend on small hotspots, including parts of the nucleus accumbens, the ventral pallidum and the parabrachial nucleus in the brainstem. There, opioid and endocannabinoid signals can amplify hedonic responses.

Pleasure versus happiness. Kringelbach and Berridge distinguish pleasure (hedonia) from a broader happiness that also includes meaning and engagement (eudaimonia).

Serotonin. Serotonin's link to mood is real but complex. The Reel's list of mood, appetite and sleep is a broad summary rather than a mechanism of happiness.

REEL VS. RECORD

- **Nuance:** dopamine is mainly about 'wanting' (incentive motivation) and reward learning, not pleasure itself. Raising dopamine can increase desire without increasing 'liking'.
- **Missing context:** pleasure depends on small hedonic hotspots (in the nucleus accumbens, ventral pallidum and brainstem), where opioids and endocannabinoids amplify it.
- **Distinction:** researchers separate momentary pleasure (hedonia) from broader happiness that includes meaning (eudaimonia), so 'happiness' isn't just reward chemistry.
- **Framing:** the serotonin list (mood, appetite, sleep) is a broad generalization, not a mechanism of happiness.
- **Accurate:** the Reel's headline claim that there's no single feel-good chemical matches the research.

SOURCES

1. Berridge & Kringelbach (2015), Pleasure systems in the brain, Neuron (PMC): <https://ncbi.nlm.nih.gov/pmc/articles/PMC4425246/>
2. Kringelbach & Berridge (2010), The Functional Neuroanatomy of Pleasure and Happiness (PMC): <https://pmc.ncbi.nlm.nih.gov/articles/PMC3008353/>

Permalink: <https://detailenjoyer.com/case/happiness/> · Last updated Sep 22, 2026



DD-744 SCIENCE · 3 OF 11

SADNESS

A 2020 multidisciplinary review describes sadness as a normal emotion with distributed brain correlates. These include the subgenual anterior cingulate cortex, the dorsal periaqueductal gray and changes in frontal activity, along with altered autonomic control of the heart. The review treats ordinary sadness and clinical depression as related but distinct. The Reel's specific neurotransmitter claims are simplifications that these sources don't directly support.

QUICK FACTS

DD-744-F01

Sadness involves distributed circuits, including the subgenual anterior cingulate cortex.

DD-744-F02

Sadness is a normal emotion distinct from clinical depression.

THE WRITE-UP

A normal emotion. Sadness is a basic emotional response to loss and disappointment, and it is distinct from clinical depression. The 2020 review brought together psychology, neuroscience and clinical perspectives on it.

Distributed circuitry. The review links sadness to distributed brain circuits, including the subgenual anterior cingulate cortex and the dorsal periaqueductal gray, along with changes in frontal brain activity.

The body. Sadness is also associated with changes in autonomic control, including the regulation of heart rate.

Reward and pleasure. Kringelbach and Berridge separate 'wanting' (motivation, which dopamine supports) from 'liking' (pleasure, which depends on opioid hotspots). A loss of motivation and a loss of pleasure aren't necessarily the same change.

Simplifications. Statements like 'dopamine decreases' or 'serotonin changes' compress a complicated, still-debated picture. Neurotransmitter effects differ by circuit, context and person.

REEL VS. RECORD

- **Unverified:** I couldn't confirm the general claim that dopamine 'often decreases' during sadness. The 2020 review describes distributed circuits (subgenual anterior cingulate, dorsal periaqueductal gray, frontal changes) rather than a simple dopamine drop.
- **Missing context:** ordinary sadness is a normal emotion and distinct from clinical depression.
- **Nuance:** motivation ('wanting', which dopamine supports) and pleasure ('liking', which depends on opioid hotspots) are separable systems, so 'reduced motivation and pleasure' isn't a single dopamine effect.
- **Missing context:** sadness also changes the body, including autonomic control of the heart.
- **Framing:** the serotonin and norepinephrine claims are broad generalizations, not established mechanisms of sadness.

SOURCES

1. Arias et al. (2020), The neuroscience of sadness: A multidisciplinary synthesis and collaborative review, Neuroscience & Biobehavioral Reviews: <https://www.sciencedirect.com/science/article/pii/S0149763418306146>
2. Kringelbach & Berridge (2010), The Functional Neuroanatomy of Pleasure and Happiness (PMC): <https://pmc.ncbi.nlm.nih.gov/articles/PMC3008353/>
3. Wikipedia: Sadness: <https://en.wikipedia.org/wiki/Sadness>

Permalink: <https://detailenjoyer.com/case/sadness/> · Last updated Sep 26, 2026



DD-745 SCIENCE · 4 OF 11

ANGER

Neuroscience reviews describe reactive anger and aggression as driven by a threat system running from the amygdala through the hypothalamus to the periaqueductal gray (PAG). The prefrontal cortex, especially the ventromedial and orbital regions, helps appraise and regulate these responses, and reduced function there is associated with more reactive aggression. The bodily surge is the sympathetic fight-or-flight response: epinephrine raises heart rate and blood pressure and sends blood to the muscles.

QUICK FACTS

DD-745-F01

Reactive anger runs through an amygdala, hypothalamus and periaqueductal gray threat circuit.

DD-745-F02

Anger (an emotion) is distinct from aggression (a behavior).

THE WRITE-UP

A threat circuit. Reviews describe a basic threat system, running from the amygdala through the hypothalamus to the periaqueductal gray, that mediates reactive aggression. Its response depends on how intense and how close the threat is.

Prefrontal regulation. Regions of the prefrontal cortex, especially the ventromedial and orbital areas, help appraise situations and regulate these responses. Reduced function there is associated with more reactive aggression, though this is a correlation and not the whole story.

The body's surge. The hypothalamus activates the sympathetic nervous system, and the adrenal glands release epinephrine. Heart rate and blood pressure rise, blood is pushed to the muscles, heart and other vital organs, and glucose and fats are released for energy.

Anger is not aggression. Researchers distinguish anger, the emotion, from aggression, the behavior. Anger doesn't always lead to aggression.

Frustration. Blair also links anger to frustration, when an expected reward isn't delivered, as well as to threat. That is broader than the Reel's 'detect threats'.

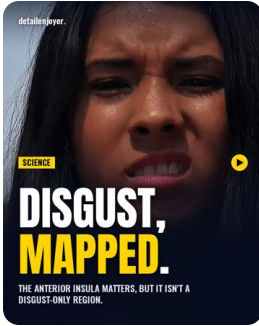
REEL VS. RECORD

- **Missing piece:** reviews place the periaqueductal gray (PAG) in the core threat circuit alongside the amygdala and hypothalamus.
- **Nuance:** reduced prefrontal activity is associated with more reactive aggression, but it's a correlation and not a single cause.
- **Missing context:** researchers distinguish anger, the emotion, from aggression, the behavior.
- **Missing context:** anger is also linked to frustration when an expected reward isn't delivered, not only to threat detection.
- **Simplification:** the bodily surge is the general sympathetic fight-or-flight response, the same one seen in fear, not something unique to anger.

SOURCES

1. Blair (2012), Considering anger from a cognitive neuroscience perspective, WIREs Cognitive Science (PMC): <https://pmc.ncbi.nlm.nih.gov/articles/PMC3260787/>
2. Understanding Brain Mechanisms of Reactive Aggression (2020, PMC): <https://pmc.ncbi.nlm.nih.gov/articles/PMC7661405/>
3. Harvard Health: Understanding the stress response: <https://www.health.harvard.edu/healthy-aging-and-longevity/understanding-the-stress-response>

Permalink: <https://detailenjoyer.com/case/anger/> · Last updated Sep 28, 2026



DD-746 SCIENCE · 5 OF 11

DISGUST

Imaging studies link the anterior insula to disgust. In one study, how strongly people felt disgust tracked anterior insula activity regardless of its form. The insula isn't disgust-specific, though: it represents the body's internal (interoceptive) state in many emotions. A 2022 meta-analysis found that 'core' disgust (contamination) and 'social' disgust (moral) engage different networks but share anterior insula and fusiform activity. Occipital, amygdala and prefrontal regions are involved too.

QUICK FACTS

DD-746-F01

Felt disgust correlated with anterior insula activity across disgust types.

DD-746-F02

Core and social disgust share anterior insula and fusiform activity.

THE WRITE-UP

Feeling disgust. In Harrison et al. (2010), how strongly people felt disgust correlated with anterior insula activity, whatever form the disgust took. This fits earlier findings for disgusting smells and for seeing disgusted faces.

Seeing versus feeling. Lesion and electrode studies suggest that recognizing disgust in others depends on a more ventral part of the insula. In Harrison's data, that ventral region responded when people observed disgust but not when they felt it.

Core versus social. A 2022 meta-analysis found that core disgust, about contamination, engaged threat-detection networks, while social disgust engaged social-cognition networks. Both shared anterior insula and fusiform activity.

A wider network. Across studies, disgust generally engaged occipital, insular, amygdala and prefrontal regions, not just the insula.

The body's state. The insula is thought to represent the body's internal, interoceptive state. Different kinds of disgust produce different bodily responses, which is part of why the insula responds in many emotions, not only disgust.

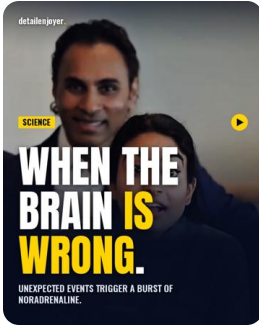
REEL VS. RECORD

- **Specificity:** the anterior insula is important for disgust but isn't disgust-specific. It represents the body's internal state across many emotions.
- **Missing context:** core (contamination) disgust and social (moral) disgust engage partly different networks, threat-detection versus social cognition, though both use the anterior insula.
- **Simplification:** meta-analyses also implicate occipital and prefrontal regions, not just the insula, basal ganglia and amygdala.
- **Nuance:** recognizing disgust in others may depend on a more ventral part of the insula than feeling disgust does.
- **Framing:** 'a neural system that detects contamination' describes disgust's proposed function, not one dedicated circuit.

SOURCES

1. Harrison et al. (2010), The Embodiment of Emotional Feelings in the Brain, Journal of Neuroscience: <https://www.jneurosci.org/content/30/38/12878>
2. Common and distinct neurofunctional representations of core and social disgust in the brain: meta-analyses (2022), Neuroscience & Biobehavioral Reviews: <https://www.sciencedirect.com/science/article/abs/pii/S0149763422000422>
3. Wikipedia: Disgust: <https://en.wikipedia.org/wiki/Disgust>

Permalink: <https://detailenjoyer.com/case/disgust/> · Last updated Oct 2, 2026



DD-747 SCIENCE · 6 OF 11

SURPRISE

The locus coeruleus (LC) is a small nucleus in the brainstem and the brain's main source of noradrenaline (norepinephrine). Brainstem fMRI studies show that deviant, behaviorally relevant stimuli produce brief bursts of LC activity, which spread arousal and attention signals widely through the brain. The LC isn't a surprise-only system, though. It also tracks task relevance and general arousal, and other neuromodulatory systems respond to surprise too.

QUICK FACTS

DD-747-F01

The locus coeruleus is the brain's main source of noradrenaline.

DD-747-F02

Noradrenaline and norepinephrine are the same molecule.

THE WRITE-UP

The locus coeruleus. The LC is a small cluster of neurons in the brainstem's pons. It is the main source of the brain's noradrenaline, and its axons reach most of the brain.

Bursts for the unexpected. Studies of deviant stimuli show brief bursts of LC activation, with noradrenaline released across the brain. The 2023 fMRI study tested whether different kinds of deviant stimuli all engage the LC, or whether other neuromodulatory systems respond too.

Not surprise-only. LC activity tracks how behaviorally relevant a deviant stimulus is, and it is involved in arousal, attention and the sleep-wake cycle generally. It is not a dedicated 'surprise center'.

One name, two words. Noradrenaline and norepinephrine are the same molecule. The Reel uses the American name.

Prediction. The Reel's idea of comparing incoming information with predictions reflects a common way of modeling surprise as a mismatch, or prediction error, between expected and actual input.

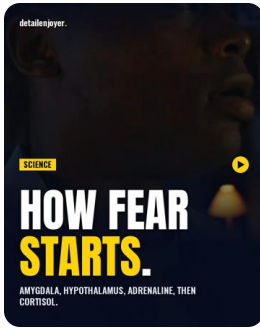
REEL VS. RECORD

- **Simplification:** the locus coeruleus isn't a surprise-specific system. It responds to behaviorally relevant deviant stimuli and also governs general arousal and attention.
- **Missing context:** other neuromodulatory systems besides the LC may also respond to unexpected stimuli, which is a question brainstem fMRI research is still testing.
- **Terminology:** norepinephrine and noradrenaline are the same molecule.
- **Missing context:** the LC is a small brainstem nucleus whose projections reach most of the brain, which is why a brief burst can raise arousal broadly.
- **Framing:** 'comparing information with predictions' is a model of surprise (prediction error), not a single identified circuit.

SOURCES

1. Brainstem fMRI signaling of surprise across different types of deviant stimuli (2023, PMC): <https://pmc.ncbi.nlm.nih.gov/articles/PMC10698303/>
2. Wikipedia: Locus coeruleus: https://en.wikipedia.org/wiki/Locus_coeruleus

Permalink: <https://detailenjoyer.com/case/surprise/> · Last updated Oct 5, 2026



FEAR

When the amygdala perceives danger, it signals the hypothalamus. The hypothalamus activates the sympathetic nervous system, and the adrenal glands release epinephrine (adrenaline), raising heart rate, blood pressure and blood sugar. If the threat persists, the HPA axis takes over: CRH from the hypothalamus triggers ACTH from the pituitary, which prompts the adrenal cortex to release cortisol. The amygdala is central to fear of external threats, but a 2013 study showed that people without functioning amygdalae can still feel fear and panic triggered from inside the body.

QUICK FACTS

DD-748-F01

Cortisol comes from the adrenal cortex, the last step of the HPA chain.

DD-748-F02

People with bilateral amygdala damage still felt fear when they inhaled carbon dioxide.

THE WRITE-UP

The alarm. Information from the eyes and ears reaches the amygdala. When it perceives danger, it sends a distress signal to the hypothalamus. This cascade can begin before the brain's visual centers have fully processed what is happening.

The fast wave. The hypothalamus activates the sympathetic nervous system through nerves to the adrenal glands, which release epinephrine (adrenaline). Heart rate, blood pressure and breathing go up, and glucose and fats are released for energy.

The slow wave. As the initial epinephrine surge subsides, the HPA axis takes over. The hypothalamus releases CRH, the anterior pituitary releases ACTH, and the adrenal cortex releases cortisol. Cortisol then feeds back to shut the response down.

Not only the amygdala. Patient SM and two others with bilateral amygdala damage felt fear, and had panic attacks, after inhaling 35% carbon dioxide. The authors concluded the amygdala is not required for fear triggered from inside the body.

The brake. When the threat passes, cortisol falls and the parasympathetic nervous system dampens the stress response.

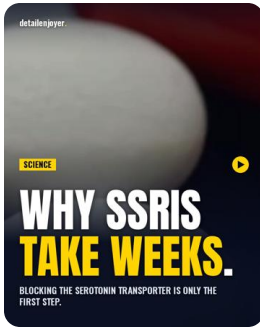
REEL VS. RECORD

- **Mechanism:** the HPA 'axis' doesn't release cortisol in one step. The hypothalamus releases CRH, the pituitary releases ACTH, and the adrenal cortex then releases cortisol.
- **Timing:** the HPA response isn't simultaneous. Harvard Health describes it as a second wave that follows as the initial epinephrine surge subsides.
- **Simplification:** the amygdala is central to fear of external threats but not essential to all fear. Amygdala-damaged patients still felt fear and panic after inhaling carbon dioxide (Feinstein et al., 2013).
- **Missing context:** the amygdala and hypothalamus can start the cascade before the brain's visual centers have fully processed the scene.
- **Missing context:** the parasympathetic nervous system acts as the 'brake', calming the body once the threat passes.

SOURCES

1. Harvard Health: Understanding the stress response: <https://www.health.harvard.edu/healthy-aging-and-longevity/understanding-the-stress-response>
2. Cleveland Clinic: Hypothalamic-Pituitary-Adrenal (HPA) Axis: <https://my.clevelandclinic.org/health/body/hypothalamic-pituitary-adrenal-hpa-axis>
3. Feinstein et al. (2013), Fear and panic in humans with bilateral amygdala damage, Nature Neuroscience (PMC): <https://ncbi.nlm.nih.gov/pmc/articles/PMC3739474/>

Permalink: <https://detailenjoyer.com/case/fear/> · Last updated Oct 7, 2026



DD-749 SCIENCE · 8 OF 11

SSRIS

SSRIs inhibit the serotonin transporter (SERT) on presynaptic neurons, so serotonin stays in the synaptic cleft longer. They act on SERT within hours, but the NHS says benefits commonly appear after 1 to 2 weeks and can take longer. The delay is thought to reflect slower adaptations in receptors and circuits, and the idea that low serotonin causes depression (the monoamine hypothesis) is a postulate rather than settled fact. Regulators warn about withdrawal symptoms when stopping, and about monitoring young people early in treatment. This case is general information, not medical advice.

QUICK FACTS

DD-749-F01

SSRIs work by inhibiting the serotonin transporter, SERT.

DD-749-F02

NHS guidance says SSRI benefits commonly start after 1 to 2 weeks.

THE WRITE-UP

The mechanism. SSRIs inhibit the serotonin transporter (SERT) at the presynaptic axon terminal, so more serotonin stays in the synaptic cleft and keeps stimulating postsynaptic receptors for longer. Compared with older antidepressant classes, they have little effect on dopamine or norepinephrine.

A hypothesis, not a settled cause. StatPearls describes the rationale for SSRIs as based on the monoamine hypothesis, which researchers postulate attributes depression to deficient serotonin. How SSRIs actually relieve symptoms is still researched and debated.

Why it takes time. According to the NHS, people commonly notice benefits after 1 to 2 weeks, and the full effect can take longer. The Reel's explanation, that receptors and circuits adapt over weeks, is a common scientific account of this delay.

Stopping. The NHS and the UK medicines regulator note that SSRIs can cause withdrawal symptoms when the dose is reduced or the drug is stopped. Symptoms can include dizziness, anxiety, sleep problems and 'brain zaps'. The NHS advises not stopping suddenly without talking to a doctor.

The five named drugs. Fluoxetine, sertraline, citalopram, escitalopram and paroxetine are all SSRIs.

REEL VS. RECORD

- **Timing:** the transporter is blocked quickly, but benefits typically appear after 1 to 2 weeks and the full effect can take longer (NHS). Why it takes this long is still being researched.
- **Missing context:** the low-serotonin (monoamine) explanation of depression is a hypothesis researchers postulate, not settled fact (StatPearls).
- **Missing context:** SSRIs can cause withdrawal symptoms when reduced or stopped. The NHS says not to stop suddenly without speaking to a doctor.
- **Precision:** SSRIs are called 'selective' because, unlike older antidepressant classes, they have little effect on dopamine or norepinephrine (StatPearls).
- **Missing context:** UK guidance calls for monitoring young adults for suicidal thoughts early in treatment.

SOURCES

1. StatPearls (NCBI Bookshelf): Selective Serotonin Reuptake Inhibitors: <https://ncbi.nlm.nih.gov/books/NBK554406/>
2. NHS: Antidepressants: <https://www.nhs.uk/medicines/antidepressants/>
3. GOV.UK (MHRA): SSRIs and SNRIs, use and safety: <https://www.gov.uk/government/publications/ssris-and-snr-is-use-and-safety/selective-serotonin-reuptake-inhibitors-ssris-and-serotonin-and-noradrenaline-reuptake-inhibitors-snr-is-use-and-safety>

Permalink: <https://detailenjoyer.com/case/ssris/> · Last updated Oct 9, 2026



DD-275 SCIENCE / PEOPLE · 9 OF 11

SYNAPTIC TRANSMISSION

Neurons communicate at synapses, junctions between two nerve cells. When an action potential reaches the end of the presynaptic neuron, it triggers the release of neurotransmitters from vesicles. They cross the synaptic cleft and bind to receptors on the postsynaptic neuron, opening ion channels.

Depending on the neurotransmitter and receptor, the effect excites or inhibits the next neuron. The footage is an animation of a synapse.

QUICK FACTS

DD-275-F01

Neurons meet at synapses.

DD-275-F02

Neurotransmitters are stored in vesicles.

THE WRITE-UP

Synapse. Neurons meet at synapses.

Vesicles. Neurotransmitters are stored in vesicles.

Cleft. They cross the synaptic cleft.

Receptors. They bind to receptors.

Excite or inhibit. They can excite or inhibit.

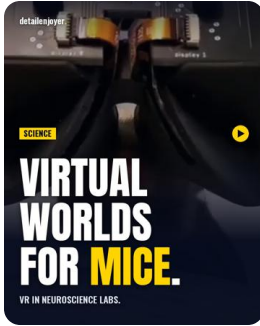
REEL VS. RECORD

- **Note:** None needed.

SOURCES

1. Wikipedia: Chemical synapse: https://en.wikipedia.org/wiki/Chemical_synapse
2. Wikipedia: Neurotransmitter: <https://en.wikipedia.org/wiki/Neurotransmitter>

Permalink: <https://detailenjoyer.com/case/synaptic-transmission/> · Last updated Jul 20, 2024



DD-429 SCIENCE · 10 OF 11

SEEMS FAMILIAR

Researchers place mice and rats in virtual reality setups, typically with the animal on a treadmill or floating ball surrounded by screens that update as it walks. Because the animal stays in place, scientists can record neurons during navigation, learning and decision-making. Newer systems use tiny headsets. The footage shows a mouse in a VR rig.

QUICK FACTS

DD-429-F01

Treadmill or ball.

DD-429-F02

Update with movement.

THE WRITE-UP

Setup. Treadmill or ball.

Screens. Update with movement.

Recording. Neurons during behavior.

Topics. Navigation and learning.

Headsets. Tiny goggles now exist.

REEL VS. RECORD

- **Note:** None needed.

SOURCES

1. Wikipedia: Virtual reality: https://en.wikipedia.org/wiki/Virtual_reality

Permalink: <https://detailenjoyer.com/case/seems-familiar/> · Last updated Dec 16, 2024



NEURALINK

Neuralink, co-founded by Elon Musk in 2016, develops implantable brain-computer interfaces (BCIs): a small chip with ultra-thin electrode threads that records and can stimulate brain activity. Its near-term aim is helping people with paralysis control devices; its stated long-term goal is human-AI symbiosis. The first human implant was in January 2024. The footage, credited to Neuralink, ABC News and Royalty Free Content, shows the implant and animations.

QUICK FACTS

DD-527-F01

2016.

DD-527-F02

Implantable chip.

THE WRITE-UP

Founded. 2016.

Device. Implantable chip.

Threads. Thin electrodes.

Aim. Help paralysis.

First human. January 2024.

REEL VS. RECORD

- **Note:** None needed.

SOURCES

1. Wikipedia: Neuralink: <https://en.wikipedia.org/wiki/Neuralink>

Permalink: <https://detailenjoyer.com/case/neuralink/> · Last updated Mar 28, 2025

COLLECTION QUIZ

Test Your Knowledge: 22 questions from The Brain. Answers are on the next page.

1. About how many neurons are in the human brain? (DD-635 · Connected)

- A. 86 billion
- B. 8.6 million
- C. 860 billion
- D. 86 trillion

2. What figure did the 86 billion estimate replace? (DD-635 · Connected)

- A. 100 billion
- B. 10 billion
- C. 1 trillion
- D. 50 billion

3. According to Berridge, dopamine is mainly linked to... (DD-743 · Happiness)

- A. 'Liking'
- B. 'Wanting'
- C. Vision
- D. Bone strength

4. Where are 'hedonic hotspots' found? (DD-743 · Happiness)

- A. Nucleus accumbens and ventral pallidum
- B. Fingertips
- C. Liver
- D. Retina

5. Is everyday sadness the same as clinical depression? (DD-744 · Sadness)

- A. Yes
- B. No, it's a normal emotion distinct from depression
- C. Only in winter
- D. Only in children

6. Which brain region does the 2020 review link to sadness? (DD-744 · Sadness)

- A. Subgenual anterior cingulate cortex
- B. Cerebellar vermis only
- C. Olfactory bulb
- D. Retina

7. Which structure completes the amygdala-hypothalamus threat circuit? (DD-745 · Anger)

- A. Periaqueductal gray
- B. Cerebellum
- C. Retina
- D. Olfactory bulb

8. Which brain area helps regulate reactive aggression? (DD-745 · Anger)

- A. Ventromedial prefrontal cortex
- B. Visual cortex
- C. Medulla only
- D. Spinal cord

9. Which brain region is most consistently linked to disgust? (DD-746 · Disgust)

- A. Anterior insula
- B. Cerebellum
- C. Visual cortex alone
- D. Spinal cord

10. Is the insula disgust-specific? (DD-746 · Disgust)

- A. Yes, only disgust
- B. No, it represents the body's internal state in many emotions
- C. It only processes vision
- D. It only controls movement

11. Where is the locus coeruleus? (DD-747 · Surprise)

- A. Brainstem
- B. Retina
- C. Spinal cord base
- D. Frontal lobe surface

12. Which chemical does the locus coeruleus mainly release? (DD-747 · Surprise)

- A. Dopamine
- B. Noradrenaline (norepinephrine)
- C. Insulin
- D. Serotonin

13. Which brain region sends the first distress signal to the hypothalamus? (DD-748 · Fear)

- A. Cerebellum
- B. Amygdala
- C. Hippocampus
- D. Occipital lobe

14. Which hormone causes the fast surge in heart rate? (DD-748 · Fear)

- A. Insulin
- B. Epinephrine (adrenaline)
- C. Melatonin
- D. Oxytocin

15. What do SSRIs block? (DD-749 · SSRIs)

- A. The dopamine receptor
- B. The serotonin transporter (SERT)
- C. Serotonin production
- D. The blood-brain barrier

16. What does the 'S' for selective refer to? (DD-749 · SSRIs)

- A. Little effect on dopamine or norepinephrine
- B. Prescription only
- C. Taken once a day
- D. Made in a lab

17. Where do neurons communicate? (DD-275 · Synaptic Transmission)

- A. At synapses
- B. In bones
- C. In blood
- D. In skin

18. What triggers release? (DD-275 · Synaptic Transmission)

- A. An action potential
- B. Light
- C. Heat
- D. Sound

19. What does the mouse run on? (DD-429 · Seems Familiar)

- A. A treadmill or ball
- B. A wheel only
- C. Sand
- D. Water

20. Why VR? (DD-429 · Seems Familiar)

- A. To record the brain
- B. For fun
- C. To exercise
- D. To sell

21. Founded? (DD-527 · Neuralink)

- A. 2016
- B. 2002
- C. 2020
- D. 2010

22. BCI means? (DD-527 · Neuralink)

- A. Brain-computer interface
- B. Basic chip input
- C. Bionic cell implant
- D. None

ANSWER KEY

1. **A. 86 billion** Now You Know: About 86 billion, per a 2009 study. (DD-635)
2. **A. 100 billion** Now You Know: The textbook 100 billion. (DD-635)
3. **B. 'Wanting'** Now You Know: Dopamine supports incentive 'wanting' and reward learning. (DD-743)
4. **A. Nucleus accumbens and ventral pallidum** Now You Know: Small hotspots there amplify pleasure. (DD-743)
5. **B. No, it's a normal emotion distinct from depression** Now You Know: Sadness is a normal emotion; depression is a clinical condition. (DD-744)
6. **A. Subgenual anterior cingulate cortex** Now You Know: It also links the dorsal periaqueductal gray and frontal changes. (DD-744)
7. **A. Periaqueductal gray** Now You Know: The basic threat system runs from the amygdala through the hypothalamus to the PAG. (DD-745)
8. **A. Ventromedial prefrontal cortex** Now You Know: Ventromedial and orbital frontal regions help regulate the response. (DD-745)
9. **A. Anterior insula** Now You Know: Felt disgust correlates with anterior insula activity. (DD-746)
10. **B. No, it represents the body's internal state in many emotions** Now You Know: The insula is involved in interoception broadly. (DD-746)
11. **A. Brainstem** Now You Know: It is a small nucleus in the brainstem's pons. (DD-747)
12. **B. Noradrenaline (norepinephrine)** Now You Know: It is the brain's main source of noradrenaline. (DD-747)
13. **B. Amygdala** Now You Know: The amygdala signals the hypothalamus when it perceives danger. (DD-748)
14. **B. Epinephrine (adrenaline)** Now You Know: The adrenal glands pump epinephrine into the blood. (DD-748)
15. **B. The serotonin transporter (SERT)** Now You Know: They inhibit SERT, so serotonin stays in the synaptic cleft longer. (DD-749)
16. **A. Little effect on dopamine or norepinephrine** Now You Know: SSRIs act mainly on serotonin reuptake. (DD-749)
17. **A. At synapses** Now You Know: Junctions. (DD-275)
18. **A. An action potential** Now You Know: An electrical impulse. (DD-275)
19. **A. A treadmill or ball** Now You Know: Stays in place. (DD-429)
20. **A. To record the brain** Now You Know: Neuroscience. (DD-429)
21. **A. 2016** Now You Know: Musk. (DD-527)
22. **A. Brain-computer interface** Now You Know: Tech. (DD-527)

NOW YOU KNOW!