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Effects of a digitalized childhood on the cognitive and emotional maturation of the brain

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Brain rot. A term that sounds provocative – and yet it describes with frightening precision what I see every day in my practice: a decay of neural structures triggered by excessive, algorithmically controlled digital stimuli. No wonder Oxford Languages chose this very word as its Word of the Year in 2024.¹ It refers to the cognitive and emotional impairment caused by excessive screen use, especially in children.

For the first time in history, children are growing up permanently and intensively exposed to digital stimuli. Smartphones, tablets, social networks – these are no longer just tools. They are digital living spaces in which childhood takes place today.

And the question we all need to ask ourselves is not just a medical or educational one, but a social one: What happens to a brain that matures through two-dimensional, algorithmically controlled stimuli instead of through direct experience, touch, movement, and social interaction? When likes, rapid image sequences, and swiping dominate perception, thinking, and feeling, it is not only the architecture of the brain that changes, but also the basis of what we understand as our “self”.

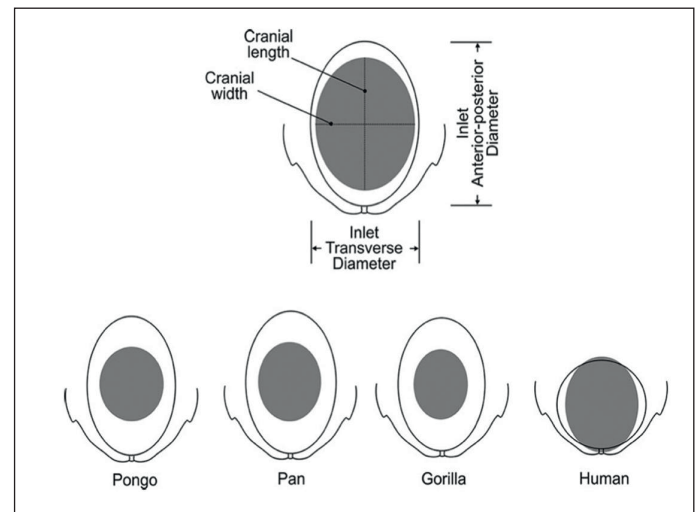
The latest data from the 2025 Common Sense Media study² provides impressive figures on the explosive increase in media use among toddlers and children. Children as young as two to four years old already spend over two hours a day with screen media. Gaming time has increased by 65 percent in the last four years between 2020 and 2024, and almost one in three children uses AI-based applications for learning. By the age of two, four in ten children already have their own tablet, and by the age of four, it is more than half. By the age of eight, almost one in four children has their own smartphone. Short video platforms, originally designed for older children and teenagers, are now increasingly becoming the main source of video content for young children. The majority of parents express great concern about screen time. Daily reading is declining among children aged five to eight. It is also worrying that 20% of children up to the age of eight already use digital devices to calm themselves down, during meals, or to fall asleep.

This article aims to provide insight into our neurobiological research findings and explain how digital environments influence the cognitive and emotional maturation of the brain.

1. Obstetrical dilemma: Evolutionary compromise between pelvic size and brain development

Human evolution is characterized by a remarkable compromise between locomotion and brain development. With the development of upright walking, anatomical structures adapted, especially those of the female pelvis. A pelvis that was too wide would have compromised the stability of upright walking, while one that was too narrow would have made it difficult to give birth to children with relatively

large heads. At the same time, brain volume continued to grow in order to enable complex cognitive abilities such as thinking and language.^{3,4,5} This conflict between pelvic size and head volume led to what is known as the obstetric dilemma.⁶ The evolutionary solution to this dilemma was for human children to be born before their brains were fully developed. As a result, newborns are relatively immature compared to other primates: although they have almost all of their neurons, the formation of functional neural networks occurs predominantly after birth.⁷



2. From upright walking to fine motor skills:

80% of neurons for movement

The growth in brain size led to numerous adaptations that were closely linked to the development of motor skills. Quantitative analyses show that humans have around 86 billion neurons.³⁸ It is striking that around 80% of these neurons are responsible for motor skills and are mainly concentrated in the cerebellum. The high number of motor neurons underscores the central role of movement and sensorimotor integration for the human brain. The cerebellum is primarily responsible for the coordination of movement, fine motor skills, and balance, but at the same time, its extensive connections to other areas of the brain show that physical activity is crucial for the development and function of cognitive networks.

Children who sit in front of digital devices for long periods of time and have little motor activity do not use this predominant proportion of motor neurons. As a result, much of their neural potential is wasted, which not only limits their motor skills but can also have negative effects on executive functions, learning ability, and emotional regulation.

Physical exercise is therefore essential for promoting neural plasticity, strengthening cognitive skills, and ensuring the development of a functional, adaptive brain.

3. Adolescence as a critical phase of cognitive maturation

Brain development in humans is not complete at birth, but continues into adulthood, approximately until the age of 18–20. It occurs particularly slowly (*extensive neuromaturation*), with maturation processes taking place along various gradients: from back to front (caudal → rostral), from central to peripheral (core areas before cortical areas) and from medial to lateral (medial structures before lateral association cortices), so that each phase serves as the foundation for the next.^{8,9}

The environment and brain maturation interact throughout the entire lifespan. This interaction is particularly intense during the so-called critical phases. Critical phases are developmental windows in which the nervous system exhibits particularly high plasticity and in which environmental influences have a structuring effect.^{11–14} During these periods, neural circuits are reorganized and functionally stabilized through *experience-dependent refinement*, provided that the appropriate sensory or cognitive stimuli are present.^{10,11} If a critical phase is disrupted or the necessary experience is not provided, this can cause long-term and sometimes irreversible impairments in development.⁸

Such critical periods include, for example, early sensorimotor maturation, during which basic motor and perceptual functions develop; the phase of motor learning, which serves to develop gross and fine motor skills; and the phase of language and lateralization development, which is important for reading, writing, and complex cognitive abilities; the maturation of the frontal lobe (PFC, prefrontal cortex), which enables working memory, social behavior, anticipation, and concepts of space and time; and the lifelong development of the limbic system, which acts as a selector for novelty, attention, emotion, and motivation.

According to Larsen and Luna,¹⁵ adolescence as a whole (from birth to adulthood) can even be understood as a large-scale critical phase for the development of higher cognitive functions.

During these phases, the environment has a particularly strong effect by stabilizing frequently used synaptic connections and eliminating rarely used ones (synaptic pruning), which leads to the formation of efficient and specialized neural networks.^{11,13} At the end of the critical phase, plasticity decreases, causing the content to be learned to become structurally and functionally “woven” into the brain, creating stability but at the same time reducing flexibility.¹⁴

Let us note that critical periods are time windows during which the brain is particularly malleable, but also particularly sensitive. This is because during this time, neural connections are stabilized, which later form the basis for complex functions. Early, real-life experiences—movement, orientation, physical contact, diverse sensory

impressions, and social interactions—are crucial for these networks to develop optimally. Digital media can only replace this diversity to a limited extent. If important experiences are missing or are conveyed in a one-sided manner, this can permanently impair the development of central brain functions. Critical periods thus show how fundamental the quality of environmental contacts is for brain development and why active engagement with the real world remains irreplaceable.

4. Lifelong critical phase in the hippocampus

While the critical phase for the maturation of many areas of the brain lasts only until adulthood, it remains lifelong in the hippocampus, as new nerve cells are continuously formed here from an embryonic germ layer.

The hippocampus is a central structure of the medial temporal lobe that plays a key role in processing memory content, spatial orientation, and emotional information. Anatomically, it belongs to the evolutionarily older archicortex and forms a central component of the limbic system. It owes its name to its characteristic shape, which resembles a seahorse. In the human brain, it is elongated and slightly curved, with an anterior part pointing forward and downward and a posterior part pointing backward upwards.

Along this axis, the hippocampus fulfills two central functions,¹⁵ between which there are fluid transitions:

- The anterior hippocampus is more strongly associated with emotional processes, including social behavior and anxiety regulation
- The posterior hippocampus is predominantly associated with cognitive tasks, in particular spatial navigation, figural memory (memory of visual or pictorial information and patterns), and declarative memory (consciously retrievable knowledge of facts and events).

At the same time, the hippocampus distinguishes between familiar and new information, acting as a selector for novelty. Its ability to recognize, select, filter, and integrate relevant signals into memory processes is based on the dynamic interaction of learning processes coupled with attention and motivation. In the Department of Neuroanatomy under Prof. Dr. Teuchert-Noodt, we developed our model of hippocampal plasticity and learning integration, which explains how these mechanisms work.

5. Model of the core mechanisms of learning in the hippocampus

Let's dive into the heart of learning. Our model can be divided into three central mechanisms that together enable the selection and integration of new learning content.

1. Synaptic and molecular mechanisms of learning reinforcement through Hebbian learning synapses in the hippocampus

Sensory information from the environment reaches the hippocampus primarily via the medial and lateral entorhinal cortex (EC): The medial EC mainly encodes spatial information, while the lateral EC transmits object-relevant information.^{20,21} These signals reach the granule cells of the dentate gyrus, a part of the hippocampus, where they activate glutamatergic synapses in the upper two-thirds of the cell layer.^{20,22} This is precisely where learning takes place, as described by Hebb's rule of learning: “Neurons that fire together, wire together”.^{22,23}

This occurs molecularly via two types of receptors – AMPA and NMDA.²³ When the presynaptic neuron becomes active, it releases glutamate. This initially binds to the AMPA receptors of the postsynaptic membrane, causing sodium ions (Na^+) to flow into the cell and depolarizing the membrane.²³ The NMDA receptor also responds to glutamate, but is initially blocked by a magnesium ion (Mg^{2+}), which acts like a “plug” to prevent the flow of ions.^{23,24}

Only when the postsynapse itself is active and depolarized is the Mg^{2+} block removed and the NMDA receptor can open. When glutamate then binds to the open NMDA receptor, calcium (Ca^{2+}) flows into the postsynapse. This calcium is the decisive signal for synaptic amplification, known as long-term potentiation (LTP). The presynapse and postsynapse fire almost simultaneously. The NMDA receptor acts like an “AND switch” – only when both are active is the connection strengthened.^{23,24}

There are two forms of LTP: *early* LTP strengthens the synapse within minutes for several hours by incorporating AMPA receptors. *Late* LTP acts at the structural level, leading to new protein synthesis and synapse formation over hours to days and ensuring lasting strengthening.^{23,24}

This system is specifically geared towards learning: information from the entorhinal cortex, especially spatial information, specifically strengthens the synapses and consolidates learning processes. If this information is missing, there is no strengthening and no learning takes place. What’s more, unused connections atrophy – according to the principle of “use it or lose it”.^{20–24}

2. Neurogenesis: Permanent restlessness forces reorganization

While sensory inputs in the molecular layer of the dentate gyrus are initially processed in the upper two-thirds, a different synaptic connection takes place in the lower third. Immediately below the granule cell layer lies a lifelong active embryonic germ layer – the so-called subgranular zone (SGZ).^{25,26} New nerve cells are continuously produced here: approximately 1600 cells per mm^2 in children and around 700 cells per mm^2 in adults.

In order for the newly formed granule cells to be integrated into the existing network, continuous structural and functional reorganization through synaptic adaptations is required, particularly in the inner third of the molecular layer.^{25,26} In our model, this process proceeds as follows: During the ingrowth of new nerve cells, the young neurons send out neurotrophic factors that stimulate neighboring circuits to connect with them. Existing synapses detach from their previous partners and connect with the new neurons.^{25,26} This leads to a state of constant dynamic instability—a kind of “functional restlessness”—that keeps the network flexible and adaptable.^{25,26}

This permanent restructuring creates the basis for high adaptability: it enables the rapid encoding and evaluation of sensory information, but at the same time contributes to the transience of memory traces.²⁷ Information can be efficiently evaluated and categorized in the short term, but fades away if it is not transferred to long-term memory processes. This corresponds to the functioning of short-term memory.²⁷

3. Theta rhythm and the coupling of emotion and cognition through movement

A central feature of the hippocampal system is its timing by the theta rhythm (4–7 Hz). This rhythm is generated by the medial septum and acts as a “neuronal metronome” that precisely controls the activity of the hippocampus in a phase-dependent manner.^{15,28} Theta timing coordinates the flow of information between the anterior hippocampus, which is primarily responsible for emotional and social processes—including anxiety regulation, affective evaluation, and motivational states—and the posterior hippocampus, which specializes in cognitive tasks such as spatial navigation, contextual memory, and declarative memory.^{15,29,30}

In a resting state, the theta rhythm is not normally active. It is only activated during active movement, exploration, focused attention, or REM sleep.^{28,29} During these phases, theta timing synchronizes neural activity in a phase-dependent manner so that learning content can be processed in a temporally and spatially organized manner. This allows new information to be encoded efficiently and emotion/motivation and cognition to be dynamically coupled.^{15,28,29}

Together, these three mechanisms form the basis for the hippocampus to function as a central interface for learning. It does not simply receive information passively, but constantly checks it for novelty, filters it specifically, and even coordinates it on different time levels. This makes it an important integration and selection instance in the brain, ensuring that learning processes can be maintained flexibly, adaptively, and throughout life. If rich, multisensory experiences are lacking and are replaced by strongly visual digital stimuli, this can hinder the formation of stable memory traces and disrupt emotional self-regulation. Teuchert-Noodt aptly describes this effect as “functional decoupling of the limbic system”.³¹

6. Overstimulation of the hippocampus by digital media and constant sensory stimuli

Today’s ubiquitous use of digital media leads to chronic overstimulation and thus to a “functional decoupling of the limbic system.” Endless reels, constant notifications, and multisensory stimuli create an uninterrupted stream of information. Not only light, color, and movement on screens activate the hippocampus, but also the rapid succession of visual, auditory, and other sensory stimuli.

Thermal effects such as blue light and overheating, but also athermal effects—such as rapid image changes or algorithmically controlled sensory overload—create a state of permanent activation. Neural networks are under constant stress, cannot find rest, and increasingly lose their ability to separate relevant from irrelevant information.^{32,33}

Studies provide alarming findings: children and adolescents who use media extensively show changes in areas responsible for emotional regulation, reward processing, and cognitive control.^{32,34} Animal models show that *excessive sensory stimulation* (ESS) during the early developmental phase promotes hyperactivity and attention deficits and disrupts learning and memory processes.^{33,36} Even young children who use screens intensively show significant deficits in writing and language.^{32,34}

In the context of our model, this means that if the hippocampus is constantly flooded with unfiltered, intense sensory input—with-

out real, three-dimensional spaces for experience, without coupled motor skills, without breaks and repetition phases—then the three central mechanisms become unbalanced:

- Hebbian learning synapses cannot be selectively strengthened
- Lifelong neurogenesis loses its structural effect
- Theta rhythm synchronization is disrupted^{33,35,36}

The result: the system overloads itself. Network stability declines, learning and memory performance deteriorate, and the ability to process information and learn is weakened.^{32–36}

We are not talking about an abstract danger here, but rather about very concrete, measurable neurological consequences for children and adolescents whose brains are still in critical stages of development. This constant digital overload acts like continuous fire on the brain's central learning system—with consequences that we can already see today and whose long-term effects we can only guess at.^{32–36}

7. Activation of the reward system and influence on the prefrontal cortex

Permanent overstimulation of the hippocampal system has an effect far beyond the hippocampus and influences the mesocortical dopamine pathway in particular. This projection is central to the development and function of the prefrontal cortex (PFC), the region responsible for working memory, impulse control, decision-making processes, and long-term planning. Research by the Teuchert-Noodt working group has shown that the mesocortical dopamine innervation of the PFC is particularly sensitive to environmental influences during early development. Even slight dopaminergic overstimulation during sensitive phases can lead to profound structural changes. The findings show that a single dose of methamphetamine during the early developmental window can reduce subsequent dopamine fiber density in the PFC by more than fifty percent.^{25,26} This result illustrates how vulnerable the system is and how sensitive it is to early childhood stimulation – a mechanism that is also relevant to modern digital overstimulation in a figurative sense.

However, the mesocortical dopamine pathway is also permanently stimulated in adulthood. The resulting continuous release of dopamine leads to chronic activation of prefrontal networks. Instead of a finely modulated, context-dependent dopamine effect, overstimulation occurs. At the same time, GABAergic interneurons in the PFC increasingly take over the modulation of dopaminergic fibers. This reorganization represents an attempt by the system to compensate for chronic overstimulation, but leads to dysregulation of the prefrontal network. The result is a significant impairment of executive functions: working memory, decision-making ability, impulse control, and goal-oriented planning lose stability and efficiency. The brain reacts increasingly impulsively, seeks new stimuli more quickly, and loses the ability to maintain attention over longer periods of time.

Overall, the neurobiological findings suggest that constant digital stimulation is a form of chronic dopaminergic stress that has a particularly profound effect on the functioning of the prefrontal cortex. Since this area continues to develop well into young adulthood, such disruptive factors affect a system that remains malleable and vulnerable for years. The permanent activation of the reward system

thus not only destabilizes the hippocampus, but also weakens in the long term those prefrontal functions that are irreplaceable for self-control, reflective thinking, and sustainable learning.

Conclusion – Digital cocaine for the brain

For our children, the constant sensory overload from digital media is like a neurobiological barrage, a form of “digital cocaine”. Every click, every new image, every like releases small dopamine kicks – the reward system is constantly on high alert. The hippocampus works incessantly, the prefrontal cortex is under constant stress.

The brain learns to respond only to quick clicks – instead of deep-rooted, sustainable learning. Concentration, patience, long-term goal pursuit – all of these are weakened. Emotions and motivation become increasingly disconnected from cognition and reflection.

What remains is a system that constantly demands new stimuli but finds less and less real satisfaction. Digital overstimulation is no substitute for real experience, touch, or social interaction. It is usually low in movement, rendering around 80% of the brain, which is intended for motor functions, virtually inoperable. The brain is thus systematically robbed of its natural learning and action capabilities. In short, the constant overstimulation of the digital world puts our brains into an artificial state—addicted to novelty but lacking in depth.

Responsibility and opportunity

We all bear responsibility for this: as parents, teachers, scientists, doctors, and therapists. The particularly long postnatal brain development – due to the compromise between pelvic width and brain size – as well as the disproportionately high density of motor neurons in the brain (80%) necessitate real, three-dimensional experiences: movement, grasping, feeling, and social closeness. Only in this way can stable cortical and limbic networks develop, which form the basis for cognitive, emotional, and social skills.

Similar to dealing with alcohol or driving a car, we need rules, structures, and protective measures to prevent our children's brains from rotting – to prevent *brain rot* from becoming the norm.

It is up to us to critically examine digital environments, recognize algorithmic control, and create conditions in which children can grow up to be resilient, self-determined, and empathetic.

We are at a crossroads: we can shape and control the risks of the digital world, or we can leave our children to an endless stream of stimuli.

Digital media can significantly impair children's cognitive and emotional development. We must actively support our children, provide them with guidance, and safely navigate them through this digitally-driven world so that their brains can develop to their full potential.

There is no plan B. There is only this one childhood. Let us protect it. Because what we preserve today determines who our children will be tomorrow.

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Literature

- 1 Oxford University Press. Oxford University Press; [(accessed on December 2, 2024)]. Brain Rot Named Oxford Word of the Year 2024. Available online: <https://corp.oup.com/news/brain-rot-named-oxford-word-of-the-year-2024/>
- 2 Common Sense Media (2025). The Common Sense Census: Media Use by Kids Age Zero to Eight 2025. Common Sense Media. Available online: <https://www.common SenseMedia.org/sites/default/files/research/report/2025-common-sense-census-web-2.pdf>
- 3 Rosenberg, K., & Trevathan, W. (2005). Bipedalism and human birth: The obstetrical dilemma revisited. *Evolutionary Anthropology: Issues, News, and Reviews*, 14(5), 161–168. <https://doi.org/10.1002/evan.1360040506>
- 4 Washburn, S. L. (1960). Tools and human evolution. *Scientific American*, 203(3), 63–75.
- 5 Kuzawa, C. W., Chugani, H. T., Grossman, L. I., Lipovich, L., Muzik, O., Hof, P. R., Wildman, D. E., Sherwood, C. C., Leonard, W. R., & Lange, N. (2014). Metabolic costs and evolutionary implications of human brain development. *Proceedings of the National Academy of Sciences of the United States of America*, 111(36), 13010–13015. <https://doi.org/10.1073/pnas.1323099111>
- 6 Wittman, A. B., & Wall, L. L. (2007). The evolutionary origins of obstructed labor: Bipedalism, encephalization, and the human obstetric dilemma. *Obstetrics & Gynecology Survey*, 62(11), 739–748. <https://doi.org/10.1097/01.ogx.0000286584.04310.5c>
- 7 Teuchert-Noodt, G. (2016). The development of the child's brain is subject to the triad of activity, dynamics, and compensation. *Trauma – Journal for Psychotraumatology and its Applications*, 14(2).
- 8 Hensch, T. K. (2004). Critical period regulation. *Annual Review of Neuroscience*, 27, 549–579. <https://doi.org/10.1146/annurev-neuro.27.070203.144327>
- 9 Knudsen, E. I. (2004). Sensitive periods in the development of the brain and behavior. *Cognitive Neuroscience*, 16(8), 1412–1425. <https://doi.org/10.1162/0898929042304796>
- 10 Levelt, C. N., & Hübener, M. (2012). Critical-period plasticity in the visual cortex. *Annual Review of Neuroscience*, 35, 309–330. <https://doi.org/10.1146/annurev-neuro-061010-113813>
- 11 Hooks, B. M., & Chen, C. (2020). Circuitry underlying experience-dependent plasticity in the mouse visual system. *Neuron*, 106(1), 21–36. <https://doi.org/10.1016/j.neuron.2020.01.031>
- 12 Petanjek, Z., et al. (2011). Extraordinary neoteny of synaptic spines in the human prefrontal cortex. *Proceedings of the National Academy of Sciences*, 108(32), 13281–13286.
- 13 Takesian, A. E., & Hensch, T. K. (2013). Balancing plasticity/stability across brain development. *Progress in Brain Research*, 207, 3–34.
- 14 Larsen, B., & Luna, B. (2018). Adolescence as a neurobiological critical period for the development of higher-order cognition. *Neuroscience & Biobehavioral Reviews*, 94, 179–195. <https://doi.org/10.1016/j.neubiorev.2018.09.005>
- 15 Strange, B. A., Witter, M. P., Lein, E. S., & Moser, E. I. (2014). Functional organization of the hippocampal longitudinal axis. *Nature Reviews Neuroscience*, 15(10), 655–669. <https://doi.org/10.1038/nrn3785>
- 16 Strange, B. A., Fletcher, P. C., Henson, R. N., Friston, K. J., & Dolan, R. J. (1999). Segregating the functions of human hippocampus. *Proceedings of the National Academy of Sciences of the USA*, 96(7), 4034–4039. <https://doi.org/10.1073/pnas.96.7.4034>
- 17 Kaplan, R., Horner, A. J., Bandettini, P. A., Doeller, C. F., & Burgess, N. (2014). Human hippocampal processing of environmental novelty during spatial navigation. *Hippocampus*, 24(7), 740–750. <https://doi.org/10.1002/hipo.22264>
- 18 Tacikowski, P., Kalender, G., Ciliberti, D., & Fried, I. (2024). Human hippocampal and entorhinal neurons encode the temporal structure of experience. *Nature*, 635(8037), 160–167. <https://doi.org/10.1038/s41586-024-07973-1>
- 19 Gómez-Ocádiz, R., Trippa, M., Zhang, C.-L., Posani, L., Cocco, S., Monasson, R., & Schmidt-Hieber, C. (2022). A synaptic signal for novelty processing in the hippocampus. *Nature Communications*, 13(1), 4122. <https://doi.org/10.1038/s41467-022-31775-6>
- 20 Strauch, C., Böge, J., Shchyglo, O., Dubovyk, V., & Manahan-Vaughan, D. (2025). The suprapyramidal and infrapyramidal blades of the dentate gyrus exhibit different GluN subunit content and dissimilar frequency-dependent synaptic plasticity in vivo. *Hippocampus*, 35(2), e70002. <https://doi.org/10.1002/hipo.70002>
- 21 Wilson, D. I. G., Watanabe, S., Milner, H., & Ainge, J. A. (2013). Lateral entorhinal cortex is necessary for associative but not nonassociative recognition memory. *Hippocampus*, 23(12), 1280–1290. <https://doi.org/10.1002/hipo.22165>
- 22 Lee, I., & Kesner, R. P. (2002). Differential contribution of NMDA receptors in hippocampal subregions to spatial working memory. *Nature Neuroscience*, 5(2), 162–168. <https://doi.org/10.1038/nn790>
- 23 Mikulasch, F. A., Rudelt, L., & Priesemann, V. (2021). Local dendritic balance enables learning of efficient representations in networks of spiking neurons. *Proceedings of the National Academy of Sciences of the United States of America*, 118(50), e2021925118. <https://doi.org/10.1073/pnas.2021925118>
- 24 Yamaguchi, M., & Mori, K. (2005). Critical period for sensory experience-dependent survival of newly generated granule cells in the adult mouse olfactory bulb. *Proceedings of the National Academy of Sciences of the United States of America*, 102(27), 9697–9702. <https://doi.org/10.1073/pnas.0406082102>
- 25 Dawirs, R., Hildebrandt, K., & Teuchert-Noodt, G. (1998). Adult treatment with haloperidol increases dentate granule cell proliferation in the gerbil hippocampus. *Journal of Neural Transmission*, 105(3–4), 317–327. <https://doi.org/10.1007/s007020050061>
- 26 Hildebrandt, K., Teuchert-Noodt, G., & Dawirs, R. (1999). A single neonatal dose of methamphetamine affects granule cell proliferation in the gerbil hippocampus. *Journal of Neural Transmission*, 106(7–8), 689–701. <https://doi.org/10.1007/s007020050061>
- 27 Zhao, C., Teng, E. M., Summers, R. G., Ming, G.-L., & Gage, F. H. (2006). Distinct morphological stages of dentate granule neuron maturation in the adult mouse hippocampus. *The Journal of Neuroscience*, 26(1), 3–11. <https://doi.org/10.1523/JNEUROSCI.3648-05.2006>
- 28 Buzsáki, G. (2002). Theta oscillations in the hippocampus. *Neuron*, 33(3), 325–340. [https://doi.org/10.1016/S0896-6273\(02\)00586-X](https://doi.org/10.1016/S0896-6273(02)00586-X)
- 29 Colgin, L. L. (2013). Mechanisms and functions of theta rhythms. *Annual Review of Neuroscience*, 36, 295–312. <https://doi.org/10.1146/annurev-neuro-062012-170330>
- 30 Eichenbaum, H. (2017). Memory: Organization and control. *Annual Review of Psychology*, 68, 19–45. <https://doi.org/10.1146/annurev-psych-010416-044131>
- 31 Teuchert-Noodt, G. (2018). Risks of neuroplastic adaptation of the perception of space and time in the context of media effectiveness. In: J. Weinzi, P. Lutzka, P. Heusser (Eds.), *Significance and endangerment of the senses in the digital age* (Witten Colloquium 5), Würzburg 2017. <https://www.erziehungskunst.de/artikel/das-wird-eine-ganze-generation-in-die-steinzeit-zurueckwerfen>
- 32 Twenge, J. M., Joiner, T. E., Rogers, M. L., & Martin, G. N. (2018). Increases in depressive symptoms, suicide-related outcomes, and suicide rates among U.S. adolescents after 2010 and links to increased new media screen time. *Clinical Psychological Science*, 6(1), 3–17. <https://doi.org/10.1177/2167702617723376>
- 33 Hutton, J. S., Dudley, J., Horowitz-Kraus, T., DeWitt, T., & Holland, S. K. (2020). Associations between screen-based media use and brain white matter integrity in preschool-aged children. *JAMA Pediatrics*, 174(1), e193869. <https://doi.org/10.1001/jamapediatrics.2019.3869>
- 34 Christakis, D. A., Ramirez, J. S. B., Ferguson, S. M., & Ramirez, J.-M. (2018). How early media exposure may affect cognitive function: A review of results from observations in humans and experiments in mice. *Proceedings of the National Academy of Sciences*, 115(40), 9851–9858. <https://doi.org/10.1073/pnas.171154811>
- 35 Orben, A., & Przybylski, A. (2019). Screens, Teens, and Psychological Well-Being: Evidence From Three Time-Use Diary Studies. *Psychological Science*, 30, 095679761983032. <https://doi.org/10.1177/0956797619830329>
- 36 Volkow, N. D., Wang, G.-J., Fowler, J. S., Tomasi, D., & Telang, F. (2011). Addiction: Beyond dopamine reward circuitry. *Proceedings of the National Academy of Sciences*, 108(37), 15037–15042. <https://doi.org/10.1073/pnas.1010654108>
- 37 Owens, J., Adolescent Sleep Working Group, & Committee on Adolescence. (2014). Insufficient sleep in adolescents: Causes, consequences, and treatment strategies. *Pediatrics*, 134(3), e921–e932. <https://doi.org/10.1542/peds.2014-1696>
- 38 Prati, J. M., Pontes-Silva, A., & Gianlorenço, A. C. L. (2024). The cerebellum and its connections to other brain structures involved in motor and non-motor functions: A comprehensive review. *Behavioural Brain Research*, 465, 114933. <https://doi.org/10.1016/j.bbr.2024.114933>