Independent Research & Further Reading

Guest: David Eagleman

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Contents

Internal Models of the World in the Brain

> *"how the brain, which is locked inside the skull (...) How it constructs this model of the world"*

Research suggests that the brain constructs internal models of the world, using them to predict sensory input and guide behaviour. Predictive or generative frameworks propose that perception and action arise from continuous inference, where the brain updates its expectations based on incoming information.

Evidence from hippocampal–cortical systems shows that the brain forms cognitive maps, encoding spatial and relational structure to support navigation and planning. Behavioural findings, such as the ability to act on objects out of view, further indicate the presence of stable internal representations, linked to regions like the precuneus and superior parietal cortex.

Neuroimaging studies provide direct support for model-based processing. Activity in primary visual cortex (V1) reflects inferred features of unseen parts of a scene, while higher-order regions in the prefrontal cortex and default mode network maintain predictive representations of events and update them through prediction errors. Overall, converging evidence from theory, behaviour, and neuroscience supports the view that the brain actively constructs and updates internal models to support perception, memory, and action.

Alternative Theories

There are several competing or alternative frameworks to the free-energy principle (FEP) and active inference, even among theories that agree the brain is predictive or probabilistic. Some approaches, such as predictive coding and broader Bayesian models, describe how the brain may implement prediction and inference without requiring a global free-energy minimisation principle or an account of action grounded in active inference. Similarly, reinforcement learning frameworks, including successor representations, explain goal-directed behaviour through learned predictive structures without positing full generative models of the world in the FEP sense.

Other theories retain the idea of hierarchical prediction but reject FEP's specific formalism. Predictive processing is sometimes treated as an independent framework centred on prediction-error minimisation, without invoking thermodynamic principles or variational free energy.

Likewise, computational models of hierarchical world representations in the cortex often develop generative architectures without adopting the broader philosophical commitments of FEP.

More critical perspectives emerge from ecological and enactive approaches, which challenge the notion that the brain constructs internal models of a hidden external world. Instead, they emphasise dynamic interactions between brain, body, and environment, arguing that cognition arises through embodied engagement rather than internal representation. Additional critiques question whether FEP offers a distinct explanatory advantage, suggesting it may redescribe general Bayesian principles rather than provide a uniquely constrained or testable theory.

References 1-20

Neuron Count and Synaptic Pruning in the Human Brain

> *"in the brain you've got 86 billion cells called neurons (...) You get this at first, you're born with these 86 billion neurons, and they connect and connect and connect, and it finally becomes like a overgrown garden at the age of two. And from there you're pruning from there, you're taking connections away."*

The commonly cited figure of 86 billion neurons is a useful approximation, but it is not an exact or universal count. Modern estimates are largely based on studies using the isotropic fractionator method, which directly counts cells in brain tissue. One influential study reported approximately 86 billion neurons, and this value has since become widely adopted.

However, broader reviews of the evidence suggest a range rather than a single number. Across different studies and methods, estimates typically fall between about 70 and 90 billion neurons. More recent analyses have highlighted variability in the data, with reported counts spanning roughly 60 to nearly 100 billion, reflecting differences in samples, methods, and individual brains. The most accurate conclusion is that the human brain contains on the order of tens of billions of neurons, with 86 billion serving as a reasonable central estimate rather than a precise biological constant.

Synaptic Pruning Across Development

Research supports the idea that the brain undergoes synaptic pruning, but not in the simplified form suggested by the claim. Early development is characterised by an overproduction of synapses, followed by activity-dependent pruning that helps refine neural circuits. This process does occur in the first years of life, contributing to high levels of early connectivity.

However, evidence shows that pruning does not stop after age two. Instead, it continues across multiple developmental stages, including childhood, adolescence, and into early adulthood. In particular, regions such as the prefrontal cortex, which are involved in higher-order cognition and social functioning, exhibit prolonged periods of synaptic elimination and reorganisation, with synaptic density declining gradually from childhood through the third decade of life.

Moreover, while the basic structure of brain networks is established early, connectivity continues to be refined over time. This involves not only the elimination of synapses but also the strengthening and integration of remaining connections, leading to more efficient and specialised neural networks. Overall, neural connectivity does not simply peak at age two and then decline. Instead, it undergoes extended, region-specific refinement, with synaptic pruning continuing well beyond early childhood.

References 21-39

The “10% of the Brain” Myth

> *“I hear all the time, this idea that we only use 10% of our brains, that's not true.”*

There is no scientific evidence to support the claim that humans use only 10% of their brains; it is widely recognised as a neuromyth. Neuroscience research, including brain imaging techniques such as fMRI and PET, shows that most regions of the brain are active over the course of normal functioning, even during rest. Different tasks recruit different networks, but virtually all areas of the brain have identifiable functions and are used at various times.

From an evolutionary perspective, the idea is also implausible. The brain is metabolically expensive, and it would be unlikely for such a large proportion of it to remain unused without being reduced over time through natural selection. Clinical evidence further contradicts the myth, as damage to almost any brain region typically produces noticeable functional impairments. The origin

of the 10% claim is likely due to misinterpretations of early neurological findings and later popularisation in media and self-help contexts. Overall, the evidence shows that humans use their entire brain over time, not a small, fixed fraction.

References 40-44

Early Language Deprivation and Lifelong Capacity

> *"you need to learn [the concept of] language in the first several years of your life. If you don't learn language, you can never get the concept of language. Your brain will never figure that out. This happens sometimes with children who are terribly abused. they're known as feral children."*

Evidence shows that failure to acquire language in early childhood has severe and lasting effects, but does not typically result in a complete inability to develop language. Studies of deaf individuals with little or no early language exposure, as well as evidence from so-called feral children and home-signers, consistently show that delayed first-language acquisition leads to persistent deficits, especially in grammar and complex linguistic structures.

Research indicates the presence of sensitive periods, particularly for grammar and morphology, with outcomes worsening the later language exposure begins (often after around 6–8 years). Individuals deprived of early input rarely achieve native-like proficiency in any language. However, they are still able to acquire basic vocabulary, simple sentence structures, and functional communication. Overall, early language deprivation seriously compromises the brain's capacity for full language development, but does not eliminate the ability to acquire language altogether.

Childhood Maltreatment and Language Development

Severe neglect, abuse, or trauma in childhood is strongly associated with delays and impairments in language development, but not with a complete failure to acquire language. Research consistently shows that maltreated children perform worse than non-maltreated peers across receptive (understanding), expressive (speaking), and pragmatic (social use) language domains, with moderate to large effect sizes.

Neglect, particularly when early and prolonged, appears most strongly linked to language difficulties, likely due to reduced caregiver interaction and limited linguistic input. Children raised in neglectful or institutional environments often show lower language and cognitive abilities, with grammar frequently more affected than vocabulary. Greater severity, earlier onset, and longer duration of trauma are associated with more pronounced deficits.

Despite these challenges, maltreated children still develop functional language. They typically produce shorter utterances, show reduced syntactic complexity, and have weaker expressive and receptive skills, but they continue to use language productively. Some patterns may even reflect adaptation rather than total impairment.

References 45-61.

Comparative Brain Structure and Plasticity in Ageing

> *"Here's the thing about brain plasticity. Human beings have a similar brain to all our neighbors in the animal kingdom. If you compare our brain to a horse brain, a dog brain, anything like that, it's the same general structures and stuff. But what we have is much more of the wrinkly outer bit called the cortex (...)*
>
> *we often think that plasticity diminishes as you age, but it's not simply that it's diminishing. It's that you are getting the right answers about how to operate in the world (...) you don't have to change as much. Your brain doesn't require as much change."*

Evidence shows that human brains share the same fundamental structural organisation as other mammals. Across species, there is a conserved layout, including a layered neocortex (typically six layers), similar major lobes, and common brainstem and cerebellar structures. These shared features extend to cellular organisation and circuit patterns, indicating a common evolutionary blueprint.

Where humans differ is primarily in scale and degree rather than basic design. The human neocortex is disproportionately large, comprising a high percentage of total brain mass, and contains a greater number of neurons following primate scaling rules. Human brains are also highly folded

(gyrified), but this reflects general biological principles seen across mammals, where larger brains tend to exhibit more cortical folding due to surface area expansion.

In addition, humans show pronounced expansion in association cortices, particularly in frontal and parietal regions involved in higher-order cognition. These differences represent elaborations of a shared mammalian structure rather than a fundamentally distinct type of brain.

Brain Plasticity and Ageing

Evidence suggests that age-related changes in brain plasticity reflect both biological reductions and adaptive stabilisation, rather than a simple decline. Ageing is associated with decreases in synaptic plasticity (e.g., long-term potentiation and depression), neurogenesis, and neuronal excitability, particularly in regions such as the hippocampus and prefrontal cortex. These changes are linked to mechanisms including oxidative stress, neuroinflammation, and altered neuromodulation, which can impair learning and memory.

At the same time, older adults often show preserved performance in well-learned skills and knowledge, indicating greater stability of existing neural representations. Neuroimaging studies also show compensatory reorganisation, such as increased bilateral brain activity and network "scaffolding," which may support efficiency and maintain function despite underlying decline. Concepts such as cognitive reserve and compensation further emphasise that ageing involves a balance between loss and adaptation.

Importantly, plasticity is not lost altogether. Training and enriched environments can still produce structural and functional brain changes in older adults, although these effects are typically more constrained and require stronger input. Overall, ageing reflects a shift in plasticity, combining reduced flexibility with increased stability and context-dependent adaptability, rather than a uniform loss of capacity.

References 62-78.

Ulysses Contracts: Self-Binding Agreements

> *“So this is what's known in the literature as a Ulysses contract (...) a Ulysses contract is where you do something now to prevent yourself from behaving badly in the near future”*

A Ulysses contract is an advance, self-binding agreement in which a person authorises others to follow their prior instructions even if they later refuse or resist. The concept comes from Homer’s *Odyssey*, where Odysseus has himself tied to a mast and instructs his crew to ignore his future pleas, ensuring he can hear the Sirens without acting on their lure.

In modern contexts, particularly in psychiatry, Ulysses contracts (often called psychiatric advance directives) are used by individuals with episodic conditions such as bipolar disorder or schizophrenia. While competent, a person specifies future interventions, such as hospitalisation or treatment, that should occur if certain relapse indicators appear, even if they later reject those interventions. These agreements are typically justified as preserving a person’s deeper, long-term autonomy by protecting their considered values against predictable periods of impaired judgement. Overall, a Ulysses contract is a mechanism for committing one’s future self to a predetermined course of action in anticipation of diminished decision-making capacity.

References 79-88.

Alzheimer’s Pathology Without Cognitive Impairment

> *“there's a study that's been going on for decades now called the Religious Orders Study, where a bunch of Catholic nuns agreed to donate their brains for autopsy when they passed away (...) and what the researchers discovered when they look at the brain carefully is that some fraction of these nuns had Alzheimer's disease. Their brains were physically degenerating with the ravages of this dementia, but they didn't show any of the cognitive deficits that one normally has. They didn't seem to be having memory problems and so on.”*

Evidence from longitudinal clinico-pathologic studies, including the Religious Orders Study and related cohorts, shows that individuals can exhibit substantial Alzheimer’s disease (AD) neuropathology without clear clinical symptoms. A significant proportion of participants who died

without cognitive impairment were found to have amyloid plaques, neurofibrillary tangles, or other pathologies typically associated with dementia. In some cases, individuals met diagnostic thresholds for AD pathology yet maintained near-normal cognitive performance, with only subtle deficits in areas such as episodic memory.

This apparent dissociation is largely explained by the concepts of cognitive reserve and neural resilience. Factors such as higher education, intellectual engagement, and occupational complexity are associated with reduced risk of clinical impairment, even in the presence of pathology. At the neural level, differences in brain structure and function, including greater neuron density, synaptic integrity, and efficient network dynamics, may help buffer against the cognitive impact of disease. Overall, these findings demonstrate that Alzheimer's pathology and cognitive decline are not perfectly coupled. Many individuals can tolerate significant brain changes without overt symptoms, likely due to compensatory mechanisms that preserve function until a later threshold is reached.

References 89-98.

Retirement, Mortality, and Cognitive Decline

> *"**Steven: is there data to support that, that when you retire, if you retire early, or if you retire, say in your sixties, it increases your probability of an earlier death or cognitive decline?***
> *David: Almost certainly with cognitive decline because (...) you're just not getting the challenge at that point."*

Evidence on earlier retirement and its effects on mortality and cognition is mixed, and strongly influenced by prior health, job conditions, and post-retirement lifestyle. Some studies find that retiring earlier is associated with higher mortality, while others show no effect once factors such as baseline health and socioeconomic status are controlled. In many cases, higher mortality among early retirees appears to reflect selection effects, where individuals in poorer health retire earlier.

For cognitive outcomes, there is more consistent evidence linking retirement to faster cognitive decline, often described as a "mental retirement" effect. Reduced cognitive, social, and goal-directed engagement after leaving work may contribute to this pattern. However, findings are not uniform. In some populations, particularly those in physically demanding or lower-skill jobs,

earlier retirement can be neutral or even beneficial for cognitive health. Overall, the relationship is not straightforward. Retirement itself is not inherently harmful, but its effects depend on context. Continued engagement in mentally, socially, and physically stimulating activities appears to be a key factor in maintaining cognitive function, regardless of retirement age.

References 99-112

Social Engagement and Brain Health

> *"it turns out social life is one of the most important things that we can do for our brains"*

Evidence consistently shows that social engagement is an important factor in maintaining brain health and is associated with a lower risk of cognitive decline and dementia. Large longitudinal studies and meta-analyses report that individuals with higher levels of social interaction and participation have a reduced risk of dementia, while social isolation and weak social networks are linked to increased risk.

Regular social contact, group activities, and socially engaging occupations are associated with better cognitive outcomes and higher dementia-free survival. Some intervention studies also show modest improvements in cognitive functions, particularly executive function, following increased social interaction.

These benefits are thought to arise through multiple mechanisms, including increased cognitive reserve, reduced stress and inflammation, improved mental health, and healthier behaviours. While much of the evidence is observational and causal proof remains limited, the overall pattern strongly supports social engagement as a significant and modifiable contributor to cognitive health.

References 113-119

Brain Activity in Early Learning vs Expertise

> *“when I'm in novice at something, my brain is using much more activity. Not just the anterior mid cingulate, but tons of activity all over because I'm trying to figure out the rules (...) as an expert (...) You don't need to burn much activity.”*

Evidence supports the claim that early stages of skill learning involve more widespread and often higher brain activity, including the anterior/mid cingulate cortex (ACC/mid-cingulate). Neuroimaging and meta-analytic studies show that novice learners recruit broad cortical and subcortical networks, including prefrontal, parietal, cerebellar, striatal, and cingulate regions, reflecting increased demands on attention, control, and error monitoring.

The anterior and dorsal cingulate cortex, in particular, plays a prominent role during early learning. It is associated with cognitive control, performance monitoring, and adaptation, and shows stronger engagement during initial skill acquisition. As learning progresses and performance becomes more automatic, activity in these regions typically decreases, alongside a reduction in overall brain-wide activation. With expertise, neural activity becomes more efficient and specialised, shifting toward more focal sensorimotor and task-specific circuits. Overall, the evidence supports a transition from widespread, effortful processing in early learning to more efficient, streamlined neural activity with skill mastery.

References 120-127.

Musical Training and Motor Cortex Plasticity

> *“if you are a pianist, if you play piano, then we can actually see physical changes in your motor cortex (...) you actually get a bigger loop of tissue here than you do in a normal brain. Why? Because you're doing so much fine motor activity with your fingers, with both hands, okay? In contrast, if you're a violinist, you're only really doing that kind of detailed activity with one hand and the other hand's just bowing. And so you only get that activity here in one half of the brain for violinists. So I can look at a brain and tell, Hey, is the person a pianist or a violinist or neither?”*

Evidence shows that intensive musical training leads to measurable structural changes in the motor cortex and related sensorimotor networks. Studies consistently find increased grey matter, altered cortical thickness, and changes in white-matter pathways (e.g., corticospinal tract), with effects often scaling with years and intensity of practice. Longitudinal research further confirms that such changes can emerge within months of training.

There is also evidence of instrument- and effector-specific adaptations. For example, violinists tend to show greater representation of the left hand in motor and somatosensory cortex, while pianists often exhibit more bilateral changes reflecting the use of both hands. Other differences, such as hemispheric asymmetries and connectivity patterns, have been observed across instrument types. However, these differences are subtle and overlapping at the individual level. While group-level patterns can distinguish categories of musicians, current evidence does not support reliably identifying a person's specific instrument from motor cortex structure alone.

References 128-138.

Benefits of Exercise for the Brain

> *"the general story is exercise is really important for the brain."*

Exercise produces consistent, small-to-moderate improvements in cognitive function and brain health across the lifespan. The strongest effects are seen in memory and executive function (e.g. attention, planning, decision-making), alongside reductions in depression and anxiety, which further support cognitive performance.

At a structural level, regular exercise is associated with increased hippocampal volume (a key memory region), improved grey matter in frontal and temporal areas, and better white matter integrity. It also enhances cerebral blood flow and functional connectivity, supporting more efficient brain function.

These effects are driven by multiple biological mechanisms. Exercise promotes neuroplasticity (the brain's ability to change), including neurogenesis (the formation of new neurons) and synaptogenesis (formation of new connections), particularly in the hippocampus. It also increases neurotrophic factors such as BDNF and IGF-1, improves vascular and metabolic function,

and reduces inflammation and stress-related processes. Overall, exercise supports cognition, mood, and long-term brain resilience, although the optimal type and amount of exercise remain areas of ongoing research.

References 139-150.

Effort and Willingness to Pay

> *"it turns out there are psychology studies where (...) You'll pay much more for the thing that looks like it took a lot of effort."*

Evidence shows that people are often willing to pay more for products that appear to require greater effort to make, but this effect depends on how that effort is interpreted. When effort signals care, authenticity, or craftsmanship, consumers tend to value products more highly. For example, items described as handmade or artisanal are typically associated with higher willingness to pay, even when identical to machine-made alternatives.

Experimental studies also show that when firms visibly invest effort in producing or presenting a product, consumers respond more positively, often due to perceived sincerity or gratitude. Similarly, design features that suggest creativity and labour can increase perceived value. However, effort is not always rewarded. If it is seen as irrelevant, inefficient, or manipulative, it may have no effect or even reduce perceived value. Overall, effort increases willingness to pay primarily when it is viewed as meaningful and tied to quality or authenticity.

References 151-159.

AI and the Human Brain: Similarities and Differences

> *"**Steve: When you think about the brain and how it's built, and then you think about the exact technology that they've used to create AI, isn't it very, very similar? And if so, if it is similar, what does that say about human's role in the future.** David: Yeah. it's similar, but it's not the same, which is why with AI you get what we call jagged intelligence, meaning that it can do something so extraordinarily smart and then in the next moment given an answer that's weird and doesn't make any sense (...) they both have converged on something that we would call intelligence, but it's a pretty different structure. This was even though AI was inspired by the brain (...)"*

Evidence shows that modern AI systems, particularly deep neural networks, share some functional similarities with the human brain, but differ fundamentally in structure and mechanism. In domains such as vision and language, AI models can develop internal representations that correlate with patterns of brain activity, and both systems process information hierarchically, from simple features to more abstract representations.

However, these similarities are limited. The underlying structure and learning mechanisms are markedly different. Biological brains rely on local synaptic plasticity, continuous learning, and embodied interaction with the environment, whereas AI systems depend on non-local optimisation methods (e.g. backpropagation), large datasets, and lack true biological integration.

At a cognitive level, humans exhibit flexible generalisation, rich memory, and adaptive behaviour across contexts, while AI systems are more constrained, excelling in specific tasks but lacking the same breadth and robustness. Overall, AI and the brain show partial functional convergence in certain tasks, but remain deeply different in structure, learning processes, and overall form of intelligence.

Jagged Intelligence in Modern AI

Evidence strongly supports the idea that modern AI systems exhibit "jagged intelligence," meaning highly uneven performance across tasks. Studies show that AI can perform exceptionally well on certain tasks while failing or even producing misleading outputs on others that appear similar in domain or difficulty. For example, in real-world experiments, AI assistance can significantly

improve speed and quality on some knowledge-work tasks, yet reduce accuracy on others outside its effective range. Similarly, across multimodal systems, small changes in task type can lead to inconsistent or contradictory outputs, and multi-task models often perform unevenly due to competing optimisation demands. In addition, more advanced models can produce confident but incorrect answers, indicating that increased capability does not necessarily translate into reliable performance across contexts. Overall, AI systems demonstrate strong but fragmented competence, excelling in specific areas while remaining unstable or error-prone in others.

References 160-168.

Aphantasia, Hyperphantasia, and Drawing Ability

> *"hyperphantasia, which means you have very rich visualization (...) it turns out that if you are an aphantasia kid, you're gonna become better at drawing because you have to really pay attention to the subject out there and really have a dialogue with the page with your pencil. Whereas a kid who's hyperphantasia might say, oh, I know what a horse looks like, and just draws it (...)*
>
> *So we've done lots of studies about what this translates to in terms of your capacities in the world. Nothing. Why does it translate to nothing? It's because you can accomplish tasks in a hundred different ways (...) there's nothing obvious"*

Evidence suggests that individuals with aphantasia differ from those with typical or vivid imagery primarily in *how* they draw from memory, rather than in overall drawing ability. In memory-based drawing tasks, people with aphantasia tend to include fewer object details, use less colour, and rely more on verbal or symbolic strategies (e.g. labels), while maintaining accurate spatial layouts. They also tend to make fewer false additions, indicating a more conservative, detail-limited recall style. However, when drawing from direct observation, there are no significant differences in performance between individuals with aphantasia and those with typical imagery. This indicates that basic drawing skill, effort, and perceptual accuracy are largely intact.

Direct evidence comparing aphantasia and hyperphantasia in drawing is limited. Some findings suggest that individuals with more vivid imagery may perform better on object-based imagery tasks, but clear differences in drawing ability have not been robustly established. Overall,

aphantasia does not confer a general advantage or disadvantage in drawing skill, but it is associated with distinct strategies and differences in memory-based visual recall.

Imagery Vividness and Task Performance

Evidence shows that differences in visual imagery vividness (from aphantasia to hyperphantasia) do influence task performance, but the effects are selective rather than global. The clearest differences appear in domains such as autobiographical memory, imagination, and visual recognition, where performance tends to scale with imagery vividness (hyperphantasia > typical > aphantasia). Individuals with aphantasia often recall fewer visual details (e.g. objects, colours) and may show mild deficits in recognition tasks.

However, on many standard cognitive tasks, overall accuracy is similar across groups. Differences more often emerge in strategy and efficiency rather than raw ability. For example, individuals with aphantasia may rely more on verbal or analytic strategies, sometimes performing as accurately but more slowly, while those with vivid imagery may respond faster or use more image-based processing. Overall, imagery vividness affects how tasks are performed, particularly in memory richness, recognition, and processing style, but many cognitive outcomes remain comparable due to the use of alternative strategies.

References 169-178.

Synesthesia

> *"Synesthesia is having a blending of the senses (...) At least 3% of the population has this. It's not a disease or a disorder, it's just an alternative perceptual reality."*

Synesthesia is a perceptual phenomenon in which a stimulus in one sensory or cognitive domain automatically and consistently triggers an additional experience in another domain. For example, individuals may perceive specific colours when seeing letters or numbers (grapheme–colour synesthesia), experience tastes when hearing words, or mentally map numbers in spatial layouts. These experiences are stable over time, involuntary, and typically feel percept-like rather than imagined. Synesthesia is generally considered a benign variation in cognition rather than a disorder.

Estimates of its prevalence vary depending on how broadly it is defined and which subtypes are included. More specific forms, such as grapheme–colour synesthesia, are estimated to occur in around 1% of the population. When a wider range of synesthetic experiences is considered, prevalence estimates increase, with many studies suggesting that at least around 4% of people have some form of synesthesia, and some estimates reaching higher when broader criteria are used. Overall, while precise global prevalence remains uncertain due to methodological differences and limited cross-cultural data, synesthesia appears to be a relatively common cognitive variation affecting several percent of the population.

References 179-183.

Visual Cortex Reorganisation in Blindness

> *"if you go blind, the visual cortex of the back of the brain gets taken over by hearing and by touch and by other things, and it's no longer visual cortex."*

Evidence indicates that the visual cortex is repurposed for processing other sensory modalities, such as touch and hearing, in individuals who are blind, particularly in cases of congenital or early blindness. Neuroimaging studies show that occipital regions, typically associated with vision, are actively recruited during tasks such as Braille reading, tactile object recognition, and auditory processing (e.g. sound localisation and pitch discrimination). Importantly, this activity is not merely incidental. Disruption of the visual cortex using transcranial magnetic stimulation (TMS) impairs tactile tasks like Braille reading in blind individuals, indicating that these regions play a functional role in non-visual processing. Additionally, studies using sensory substitution devices demonstrate that visual cortex can respond robustly to auditory or tactile inputs.

While the cortex becomes responsive to new modalities, it often retains some functional specialisation. For example, regions associated with motion or shape processing continue to perform analogous roles, but using non-visual input. This reorganisation is most pronounced when blindness occurs early in life, suggesting a sensitive developmental window, though some degree of plasticity is also observed in adults. Overall, the visual cortex in blind individuals is not inactive but reorganised to support other sensory and cognitive functions.

References 184-190.

Dreaming and the “Defensive Activation” Hypothesis

> *“The purpose of dreaming is to defend the visual territory from takeover from the other senses. So every 90 minutes you've got this very ancient thing in your midbrain that shoots random activity into the visual system and only the visual system, only this very tiny part of the visual system. Every 90 minutes, you just blast random activity in here. And the reason is you are just defending that territory against takeover. Now, the reason that all this came together is because our colleagues at Harvard did an experiment where they took normally sighted people and they blindfolded them tightly for 60 minutes, and it turns out that 60 minutes was sufficient for the visual cortex to start responding to sound and to touch (...)”*

The evidence does not establish that dreaming evolved to defend the visual cortex from takeover by other senses, although some findings are consistent with this idea. The Defensive Activation Theory (DAT) proposes that REM sleep periodically activates visual cortex to prevent cross-modal reorganisation during visual deprivation. Supporting observations include correlations between REM sleep and neuroplasticity across species, age-related declines in both REM and plasticity, and well-established evidence that visual cortex can be rapidly recruited for other modalities under deprivation.

However, this evidence is indirect and not specific to the theory. While REM sleep robustly activates visual cortex and plays a role in neural plasticity, these effects are also consistent with broader functions of sleep, such as memory consolidation, synaptic homeostasis, and emotional processing. Importantly, some comparative data challenge the theory, for example findings that blind species exhibit similar REM sleep proportions to sighted ones. Overall, the hypothesis that dreaming serves to protect visual cortex is plausible but remains speculative. Current evidence shows that REM sleep engages and shapes visual systems, but does not demonstrate that its primary function is to prevent sensory takeover.

Cross-Modal Activation of Visual Cortex After Short-Term Blindfolding

Research shows that short-term blindfolding in sighted individuals can lead to rapid cross-modal activation of the visual cortex. Within 45–90 minutes, studies report increased occipital excitability and tactile enhancements, indicating early plastic changes. After a few hours, neuroimaging findings show altered activity and connectivity in intermediate visual areas during tactile tasks, and by around 6 hours, some individuals exhibit auditory-like responses in occipital cortex.

With longer deprivation (e.g. several days combined with tactile training), visual cortex becomes strongly and causally involved in non-visual processing, as demonstrated by disruption of Braille performance following occipital stimulation.

These effects are reversible once vision is restored. The underlying mechanism is best explained by the unmasking of pre-existing multisensory connections rather than rapid structural rewiring, and there is substantial variability between individuals. Overall, evidence supports that even short-term blindfolding can induce genuine, though graded, cross-modal recruitment of visual cortex.

References 191-200.

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