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Neuroplasticity

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Neuroplasticity is the capacity of the brain to change as a result of external influence and internal activity. It underpins learning from the environment, from others and from our own thoughts. Neuroplasticity occurs during sensory and cultural learning, adaptation to bodily differences (e.g., blindness, deafness, limb loss) and in response to nervous system damage (e.g., stroke). The term encompasses a wide variety of neurobiological mechanisms at different scales, from strengthening a single synapse to enlarging a fiber pathway. Anatomical neuroplasticity involves structural change, such as enhancement of axonal fibers or synaptic pruning. In functional neuroplasticity, neural structures change their response properties and behavioral role, sometimes without correspondingly large changes in anatomy, for example, by differential use of preexisting anatomical pathways. The potential for neuroplasticity peaks during development but continues throughout the lifespan.

History

William James used the term “plasticity” to describe the capacity of the nervous system to change during learning ([James, 1890](#)). At the time, little was known about how the nervous system supports information processing. At the end of the 19th century work by neurophysiologists, such as Santiago Ramon y Cajal, established that the brain is comprised of individuated neurons transmitting signals to each other ([Ramón y Cajal, 1904](#)). This paved the way for Donald Hebb’s articulation of activity-dependent synaptic plasticity: when one neuron causes another to fire repeatedly, the connection between these neurons is strengthened ([Hebb, 1949](#)).

A major advance in understanding neural information processing and neuroplasticity came from [Hubel & Weisel’s \(1970\)](#) experiments on the visual system of cats and monkeys. They observed that closure of one eye during the first months of life causes primary visual cortex neurons to become dominated by the undeprived eye, suggesting a “critical period” of sensitivity ([Hubel & Weisel, 1970](#), see section on Critical Periods below.) Analogous periods of heightened sensitivity to experience have been documented in humans with early cataracts ([Maurer, 2017](#); [Fine et al., 2003](#)). Research on language acquisition by Elizabeth Bates, Elissa Newport and Rachel Mayberry, among others, has demonstrated similar effects in higher-order cognition ([Johnson & Newport, 1991](#); [Bates et al., 1997](#); [Mayberry et al., 2002](#)) [see [Language Acquisition](#)].

Core concepts

Experience-based plasticity

Experience-based plasticity occurs when the brain changes in response to information from the environment and encompasses all learning, from episodic memory to language acquisition, learning of cultural skills (e.g., reading), and adapting to sensory loss. In humans, neuroplasticity is studied by comparing brain function and behavior across subpopulations with naturally occurring differences in experience (e.g., blind vs. sighted people) or by testing the same individuals before and after learning

([Sadato et al., 1996](#); [Dehaene-Lambertz et al., 2018](#)). “Controlled rearing” studies with animals manipulate experience (e.g., raising animals without visual access to faces) and measure resulting changes in the nervous system and behavior ([Sugita, 2008](#); [Arcaro et al., 2017](#)).

Critical periods

Critical periods are periods early in life when neurocognitive systems are especially receptive to environmental effects (e.g., [Hubel & Weisel, 1970](#)). The term “sensitive period” is sometimes used to indicate a more gradual and incomplete waning in plasticity over time but is also used synonymously with critical periods by some scholars. Cellular and molecular mechanisms governing the opening and closing of critical periods have been identified in sensory systems ([Hensch, 2005](#)). “Expected” experiences during critical periods are sometimes required for typical development (e.g., coordinated input from the two eyes, seeing faces, language) (e.g., [Huber et al., 2015](#); [Greenough et al., 1987](#)). The timing of critical periods differs widely across neurocognitive systems ([Maurer, 2017](#)). Not all forms of learning require critical period plasticity: we can learn new faces, stories, facts, and words throughout our lives.

Anatomical plasticity

Anatomical plasticity at the largest scale involves growth or pruning of long-range fiber tracts, myelination and changes in cortical thickness. At the smallest scale, learning occurs through synaptic plasticity i.e., the creation, elimination and modification of synaptic connections between neurons ([Feldman, 2009](#)).

Functional plasticity

Functional plasticity refers to changes in cognitive function supported by a given neural structure e.g., a neural population, cortical area, or fiber pathway. For example, visual cortices taking on non-visual functions in blindness. All functional plasticity is supported by some form of anatomical change but not always at similar scales e.g., synaptic plasticity (small-scale anatomical change) might enable a cortical area to substantially change function (large-scale functional change). The term functional plasticity is often used when anatomical substrates of functional change are not known or suspected to be small-scale.

Cross-modal plasticity

Cross-modal plasticity is an example of experience-based functional plasticity, where a sensory region that is typically dominated by one sensory modality (e.g., primary visual cortex, V1), shows heightened responses to input from other modalities in people with sensory loss (e.g., blindness) ([Bavelier & Neville, 2002](#); [Pascual-Leone et al., 2005](#)). In people who are born blind, “visual” cortices of the occipital lobe, including V1, respond to auditory, tactile and higher-order cognitive tasks e.g., braille reading, auditory sentence processing, verbal memory ([Sadato et al., 1996](#); [Röder et al., 2002](#); [Bedny, 2011](#)). Analogously in people born deaf, auditory cortices participate in non-auditory functions ([Finney et al., 2001](#)). Large-scale cross-modal reorganization is restricted to congenital sensory loss (from birth), as opposed to acquired sensory loss (in adulthood) ([Cohen et al., 1999](#)).

Questions, controversies, and new developments

Flexibility and behavioral relevance

Whether cortical areas can support cognitive functions for which they did not evolve remains debated. In the case of cross-modal plasticity in blindness, one view suggests that visual cortices have entirely changed function, i.e., from low-level vision to language ([Saccone et al., 2024](#)). Other perspectives suggest maintenance of the original computations, but a change in the modality of input ([Collignon et al., 2007](#)). The behavioral relevance of neuroplasticity is also debated, and appears variable across different examples (e.g., congenital blindness vs. acquired limb-loss) ([Cohen et al., 1997](#); [Makin et al., 2015](#)).

Plasticity in cultural learning

Cultural skills, such as literacy, numeracy, and musical expertise, are acquired through a combination of reuse and modification of existing cortical circuits. Different accounts emphasize reuse of innate domain-specific systems (*cultural-recycling hypothesis*) or de novo learning and domain general mechanisms (*cognitive gadgets*) ([Dehaene & Cohen, 2007](#); [Herholz & Zatorre, 2012](#); [Heyes, 2018](#); see [Hauptman et al., \(in press\)](#)). Literacy is a textbook example of *recycling*, it reuses and modifies neural circuits that evolved for visual object recognition and their connectivity with the language system ([Saygin et al., 2016](#); [Dehaene & Cohen, 2007](#)).

Critical/sensitive periods in higher cognition

Language is an example of a higher-cognitive capacity subject to critical or sensitive periods. For example, language deprivation, in deaf children without access to visuo-manual language, is associated with reduced proficiency in phonology and morphosyntax as well as altered neural basis of language ([Johnson & Newport, 1991](#); [Bates et al., 1997](#); [Mayberry et al., 2002](#)). The neurobiological mechanisms that open and close critical/sensitive periods in higher-cognitive systems are not known.

Plasticity in brain damage

The brain functionally reorganizes in response to damage ([Luria, 1963](#)). For example, damage to the left hemisphere language network leads to activity increases in right homologue areas and in perilesional tissue ([Hartwigsen & Saur, 2019](#)). In some domains, such as language, potential for behavioral recovery is much greater in childhood ([Bates et al., 1997](#)) (for a counterexample in memory, see [Vargha-Khadem et al., 1997](#)). Children with extensive early damage to the left hemisphere show little or no change in linguistic behavior in adulthood and show activity in right homologue frontotemporal areas during language tasks ([Bates et al., 1997](#)). Similar brain damage in adulthood leads to language impairment (i.e., aphasia) ([Hartwigsen & Saur, 2019](#)).

Edge cases: is this plasticity?

Some definitions of plasticity include neural change of any kind, including maturation. Most consider plasticity to be neural changes resulting from neural activity or effects of the environment on the brain.

There are “edge” cases that may or may not count as neuroplasticity: “Retinal waves” are innate patterns of activity in the retina that propagate into the cortex before onset of vision ([Wong, 1999](#)). Since these waves themselves are innately programmed, it is unclear whether this should count as plasticity.

Broader connections

Research on neuroplasticity is connected to work in artificial intelligence and machine learning. In neural network models, plasticity occurs when connection weights among units are updated during learning ([Smolensky, 1988](#)). Neuroplasticity research in cognitive science has focused on large-scale functional changes in humans (but connects to plasticity work at cellular and molecular levels, see [Feldman, 2009](#)). Research on plasticity connects to developmental psychology, cognitive psychology, comparative psychology, anthropology and philosophy. For example, experience-based change is relevant to nature/nurture debates within developmental psychology, evolutionary biology and philosophy ([Carey & Spelke, 1996](#)). Neuroplasticity is also relevant to cultural anthropology and archeology, which examine adaptation of humans to varying cultural and environmental contexts ([Boyd & Richerson, 2005](#); [Tomasello, 2009](#); [Roberts & Stewart, 2018](#)).

Further reading

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