Neuroplasticity

Neuroplasticity, also known as **brain plasticity**, **neuroelasticity**, or **neural plasticity**, is the ability of the brain to change continuously throughout an individual's life, e.g., brain activity associated with a given function can be transferred to a different location, the proportion of grey matter can change, and synapses may strengthen or weaken over time. The aim of neuroplasticity is to optimize the neural networks during phylogenesis, ontogeny, and physiological learning, as well as after a brain injury.[1] Research in the latter half of the 20th century showed that many aspects of the brain can be altered (or are "plastic") even through adulthood.[2][3][4][5] However, the developing brain exhibits a higher degree of plasticity than the adult brain.[6][7]:30

Neuroplasticity can be observed at multiple scales, from microscopic changes in individual neurons to larger-scale changes such as cortical remapping in response to injury.[8] Behavior, environmental stimuli, thought, and emotions may also cause neuroplastic change through activity-dependent plasticity, which has significant implications for healthy development, learning, memory, and recovery from brain damage.[8][9][10] At the single cell level, synaptic plasticity refers to changes in the connections between neurons, whereas non-synaptic plasticity refers to changes in their intrinsic excitability.

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JT Wall and J Xu have traced the mechanisms underlying neuroplasticity. Re-organization is not cortically emergent, but occurs at every level in the processing hierarchy; this produces the map changes observed in the cerebral cortex.[11]

Applications and example

The adult brain is not entirely "hard-wired" with fixed neuronal circuits. There are many instances of cortical and subcortical rewiring of neuronal circuits in response to training as well as in response to injury. There is solid evidence that neurogenesis (birth of brain cells) occurs in the adult, mammalian brain—and such changes can persist well into old age.[4] The evidence for neurogenesis is mainly restricted to the hippocampus and olfactory bulb, but current research has revealed that other parts of the brain, including the cerebellum, may be involved as well.[12] However, the degree of rewiring induced by the integration of new neurons in the established circuits is not known, and such rewiring may well be functionally redundant.[13]

There is now ample evidence for the active, experience-dependent re-organization of the synaptic networks of the brain involving multiple inter-related structures including the cerebral cortex. The specific details of how this process occurs at the molecular and ultrastructural levels are topics of active neuroscience research. The way experience can influence the synaptic organization of the brain is also the basis for a number of theories of brain function including the general theory of mind and Neural Darwinism. The concept of neuroplasticity is also central to theories of memory and learning that are associated with experience-driven alteration of synaptic structure and function in studies of classical conditioning in invertebrate animal models such as Aplysia.

Treatment of brain damage

A surprising consequence of neuroplasticity is that the brain activity associated with a given function can be transferred to a different location; this can result from normal experience and also occurs in the process of recovery from brain injury. Neuroplasticity is the fundamental issue that supports the scientific basis for treatment of acquired brain injury with goal-directed experiential therapeutic programs in the context of rehabilitation approaches to the functional consequences of the injury.

Neuroplasticity is gaining popularity as a theory that, at least in part, explains improvements in functional outcomes with physical therapy post-stroke. Rehabilitation techniques that are supported by evidence which suggest cortical reorganization as the mechanism of change include constraint-induced movement therapy, functional electrical stimulation, treadmill training with body-weight support, and virtual reality therapy. Robot assisted therapy is an emerging technique, which is also hypothesized to work by way of neuroplasticity, though there is currently insufficient evidence to determine the exact mechanisms of change when using this method.[14]

One group has developed a treatment that includes increased levels of progesterone injections in brain-injured patients. "Administration of progesterone after traumatic brain injury[15] (TBI) and stroke reduces edema, inflammation, and neuronal cell death, and enhances spatial reference memory and sensory motor recovery."[16] In a clinical trial, a group of severely injured patients had a 60% reduction in mortality after three days of progesterone injections.[17] However, a study published in the New England Journal of Medicine in 2014 detailing the results of a multi-center NIH-funded phase III clinical trial of 882 patients found that treatment of acute traumatic brain injury with the hormone progesterone provides no significant benefit to patients when compared with placebo.[18]

Vision

For decades, researchers assumed that humans had to acquire binocular vision, in particular stereopsis, in early childhood or they would never gain it. In recent years, however, successful improvements in persons with amblyopia, convergence insufficiency or other stereo vision anomalies have become prime examples of neuroplasticity; binocular vision improvements and stereopsis recovery are now active areas of scientific and clinical research.[19][20][21]

Brain training

Several companies have offered so-called cognitive training software programs for various purposes that claim to work via neuroplasticity; one example is Fast ForWord which is marketed to help children with learning disabilities.[22] A systematic meta-analytic review found that "There is no evidence from the analysis carried out that Fast ForWord is effective as a treatment for children's oral language or reading difficulties".[22] A 2016 review found very little evidence supporting any of the claims of Fast ForWord and other commercial products, as their task-specific effects fail to generalise to other tasks.[23]

Sensory prostheses

Neuroplasticity is involved in the development of sensory function. The brain is born immature and it adapts to sensory inputs after birth. In the auditory system, congenital hearing impairment, a rather frequent inborn condition affecting 1 of 1000 newborns, has been shown to affect auditory development, and implantation of a sensory prostheses activating the auditory system has prevented the deficits and induced functional maturation of the auditory system.[24] Due to a sensitive period for plasticity, there is also a sensitive period for such intervention within the first 2–4 years of life. Consequently, in prelingually deaf children, early cochlear implantation, as a rule, allows the children to learn the mother language and acquire acoustic communication.[25]

Phantom limbs

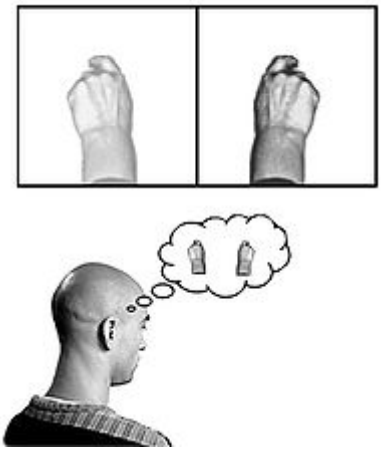
In the phenomenon of phantom limb sensation, a person continues to feel pain or sensation within a part of their body that has been amputated. This is strangely common, occurring in 60–80% of amputees.[26] An explanation for this is based on the concept of neuroplasticity, as the cortical maps of the removed limbs are believed to have become engaged with the area around them in the postcentral gyrus. This results in activity within the surrounding area of the cortex being misinterpreted by the area of the cortex formerly responsible for the amputated limb.

The relationship between phantom limb sensation and neuroplasticity is a complex one. In the early 1990s V.S. Ramachandran theorized that phantom limbs were the result of cortical remapping. However, in 1995 Herta Flor and her colleagues demonstrated that cortical remapping occurs only in patients who have phantom pain.[27] Her research showed that phantom limb pain (rather than referred sensations) was the perceptual correlate of cortical reorganization.[28] This phenomenon is sometimes referred to as maladaptive plasticity.

In 2009 Lorimer Moseley and Peter Brugger carried out a remarkable experiment in which they encouraged arm amputee subjects to use visual imagery to contort their phantom limbs into impossible configurations. Four of the seven subjects succeeded in performing impossible movements of the phantom limb. This experiment suggests that the subjects had modified the neural representation of their phantom limbs and generated the motor commands needed to execute impossible movements in the absence of feedback from the body.[29] The authors stated that: "In fact, this finding extends our understanding of the brain's plasticity because it is evidence that profound changes in the mental representation of the body can be induced purely by internal brain mechanisms—the brain truly does change itself."

Chronic pain

Individuals who suffer from chronic pain experience prolonged pain at sites that may have been previously injured, yet are otherwise currently healthy. This phenomenon is related to neuroplasticity due to a maladaptive reorganization of the nervous system, both peripherally and centrally. During the period of tissue damage, noxious stimuli and inflammation cause an elevation of nociceptive input from the periphery to the central nervous system. Prolonged nociception from the periphery then elicits a neuroplastic response at the cortical level to change its somatotopic organization for the painful site, inducing central sensitization.[30] For instance, individuals experiencing complex regional pain syndrome demonstrate a diminished cortical somatotopic representation of the hand contralaterally as well as a decreased spacing between the hand and the mouth.[31] Additionally, chronic pain has been reported to significantly reduce the volume of grey matter in the brain globally, and more specifically at the prefrontal cortex and right thalamus.[32] However, following treatment, these abnormalities in cortical reorganization and grey matter volume are resolved, as well as their symptoms. Similar results have been reported for phantom limb pain,[33] chronic low back pain[34] and carpal tunnel syndrome.[35]



A diagrammatic explanation of the mirror box. The patient places the intact limb into one side of the box (in this case the right hand) and the amputated limb into the other side. Due to the mirror, the patient sees a reflection of the intact hand where the missing limb would be (indicated in lower contrast). The patient thus receives artificial visual feedback that the "resurrected" limb is now moving when they move the good hand.

Meditation

A number of studies have linked meditation practice to differences in cortical thickness or density of gray matter.[5][36][37][38] One of the most well-known studies to demonstrate this was led by Sara Lazar, from Harvard University, in 2000.[39] Richard Davidson, a neuroscientist at the University of Wisconsin, has led experiments in cooperation with the Dalai Lama on effects of meditation on the brain. His results suggest that long-term or short-term practice of meditation results in different levels of activity in brain regions associated with such qualities as attention, anxiety, depression, fear, anger, and the ability of the body to heal itself. These functional changes may be caused by changes in the physical structure of the brain.[40][41][42][43]

Fitness and exercise

Aerobic exercise promotes adult neurogenesis by increasing the production of neurotrophic factors (compounds that promote growth or survival of neurons), such as brain-derived neurotrophic factor (BDNF), insulin-like growth factor 1 (IGF-1), and vascular endothelial growth factor (VEGF).[44][45][46] Exercise-induced neurogenesis in the hippocampus is associated with measurable improvements in spatial memory.[47][48][49][50] Consistent aerobic exercise over a period of several months induces marked clinically significant improvements in executive function (i.e., the "cognitive control" of behavior) and increased gray matter volume in multiple brain regions, particularly those that give rise to cognitive control.[46][47][51][52] The brain structures that show the greatest improvements in gray matter volume in response to aerobic exercise are the prefrontal cortex and hippocampus;[46][47][48] moderate improvements are seen in the anterior cingulate cortex, parietal cortex, cerebellum, caudate nucleus, and nucleus accumbens.[46][47][48] Higher physical fitness scores (measured by VO_2 max) are associated with better executive function, faster processing speed, and greater volume of the hippocampus, caudate nucleus, and nucleus accumbens.[47]

Human echolocation

Human echolocation is a learned ability for humans to sense their environment from echoes. This ability is used by some blind people to navigate their environment and sense their surroundings in detail. Studies in 2010[53] and 2011[54] using functional magnetic resonance imaging techniques have shown that parts of the brain associated with visual processing are adapted for the new skill of echolocation. Studies with blind patients, for example, suggest that the click-echoes heard by these patients were processed by brain regions devoted to vision rather than audition.[54]

ADHD stimulants

Reviews of MRI studies on individuals with ADHD suggest that the long-term treatment of attention deficit hyperactivity disorder (ADHD) with stimulants, such as amphetamine or methylphenidate, decreases abnormalities in brain structure and function found in subjects with ADHD, and improves function in several parts of the brain, such as the right caudate nucleus of the basal ganglia.[55][56][57]

In children

Neuroplasticity is most active in childhood as a part of normal human development, and can also be seen as an especially important mechanism for children in terms of risk and resiliency.[58] Trauma is considered a great risk as it negatively affects many areas of the brain and puts strain on the sympathetic nervous system from constant activation. Trauma thus alters the brain's connections such that children who have experienced trauma may be hyper vigilant or overly aroused.[59] However a child's brain can cope with these adverse effects through the actions of neuroplasticity.[60]

In animals

In a single lifespan, individuals of an animal species may encounter various changes in brain morphology. Many of these differences are caused by the release of hormones in the brain; others are the product of evolutionary factors or developmental stages.[61][62][63][64] Some changes occur seasonally in species to enhance or generate response behaviors.

Seasonal brain changes

Changing brain behavior and morphology to suit other seasonal behaviors is relatively common in animals.[65] These changes can improve the chances of mating during breeding season.[61][62][63][65][66][67] Examples of seasonal brain morphology change can be found within many classes and species.

Within the class Aves, black-capped chickadees experience an increase in the volume of their hippocampus and strength of neural connections to the hippocampus during fall months.[68][69] These morphological changes within the hippocampus which are related to spatial memory are not limited to birds, as they can also be observed in rodents and amphibians.[65] In songbirds, many song control nuclei in the brain increase in size during mating season.[65] Among birds, changes in brain morphology to influence song patterns, frequency, and volume are common.[70] Gonadotropin-releasing hormone (GnRH) immunoreactivity, or the reception of the hormone, is lowered in European starlings exposed to longer periods of light during the day.[61][62]

The California sea hare, a gastropod, has more successful inhibition of egg-laying hormones outside of mating season due to increased effectiveness of inhibitors in the brain.[63] Changes to the inhibitory nature of regions of the brain can also be found in humans and other mammals.[64] In the amphibian Bufo japonicus, part of the amygdala is larger before breeding and during hibernation than it is after breeding.[66]

Seasonal brain variation occurs within many mammals. Part of the hypothalamus of the common ewe is more receptive to GnRH during breeding season than at other times of the year.[67] Humans experience a change in the "size of the hypothalamic suprachiasmatic nucleus and vasopressin-immunoreactive neurons within it"[64] during the fall, when these parts are larger. In the spring, both reduce in size.[71]

Traumatic brain injury research

Randy Nudo's group found that if a small stroke (an infarction) is induced by obstruction of blood flow to a portion of a monkey's motor cortex, the part of the body that responds by movement moves when areas adjacent to the damaged brain area are stimulated. In one study, intracortical microstimulation (ICMS) mapping techniques were used in nine normal monkeys. Some underwent ischemic-infarction procedures and the others, ICMS procedures. The monkeys with ischemic infarctions retained more finger flexion during food retrieval and after several months this deficit returned to preoperative levels.[72] With respect to the distal forelimb representation, "postinfarction mapping procedures revealed that movement representations underwent reorganization throughout the adjacent, undamaged cortex."[72] Understanding of interaction between the damaged and undamaged areas provides a basis for better treatment plans in stroke patients. Current research includes the tracking of changes that occur in the motor areas of the cerebral cortex as a result of a stroke. Thus, events that occur in the reorganization process of the brain can be ascertained. Nudo is also involved in studying the treatment plans that may enhance recovery from strokes, such as physiotherapy, pharmacotherapy, and electrical-stimulation therapy.

Jon Kaas, a professor at Vanderbilt University, has been able to show "how somatosensory area 3b and ventroposterior (VP) nucleus of the thalamus are affected by longstanding unilateral dorsal-column lesions at cervical levels in macaque monkeys."[73] Adult brains have the ability to change as a result of injury but the extent of the reorganization depends on the extent of the injury. His recent research focuses on the somatosensory system, which involves a sense of the body and its movements using many senses. Usually, damage of the somatosensory cortex results in impairment of the body perception. Kaas' research project is focused on how these systems (somatosensory, cognitive, motor systems) respond with plastic changes resulting from injury.[73]

One recent study of neuroplasticity involves work done by a team of doctors and researchers at Emory University, specifically Dr. Donald Stein[74] and Dr. David Wright. This is the first treatment in 40 years that has significant results in treating traumatic brain injuries while also incurring no known side effects and being cheap to administer.[17] Dr. Stein noticed that female mice seemed to recover from brain injuries better than male mice, and that at certain points in the estrus cycle, females recovered even better. This difference may be attributed to different levels of progesterone, with higher levels of progesterone leading to the faster recovery from brain injury in mice. However, clinical trials showed progesterone offers no significant benefit for traumatic brain injury human patients.[75]

Aging

Transcriptional profiling of the frontal cortex of persons ranging from 26 to 106 years of age defined a set of genes with reduced expression after age 40, and especially after age 70.[76] Genes that play central roles in synaptic plasticity were the most significantly affected by age, generally showing reduced expression over time. There was also a marked increase in cortical DNA damage, likely oxidative DNA damage, in gene promoters with aging.[76]

Reactive oxygen species appear to have a significant role in the regulation of synaptic plasticity and cognitive function.[77] However age-related increases in reactive oxygen species may also lead to impairments in these functions.

History

Origin

The term "plasticity" was first applied to behavior in 1890 by William James in The Principles of Psychology.[78] The first person to use the term neural plasticity appears to have been the Polish neuroscientist Jerzy Konorski.[3][79]

In 1793, Italian anatomist Michele Vicenzo Malacarne described experiments in which he paired animals, trained one of the pair extensively for years, and then dissected both. He discovered that the cerebellums of the trained animals were substantially larger. But these findings were eventually forgotten.[80] The idea that the brain and its function are not fixed throughout adulthood was proposed in 1890 by William James in The Principles of Psychology, though the idea was largely neglected.[78] Until around the 1970s, neuroscientists believed that the brain's structure and function was essentially fixed throughout adulthood.[81]

The term has since seen broadly applied:

> Given the central importance of neuroplasticity, an outsider would be forgiven for assuming that it was well defined and that a basic and universal framework served to direct current and future hypotheses and experimentation. Sadly, however, this is not the case. While many neuroscientists use the word neuroplasticity as an umbrella term it means different things to different researchers in different subfields ... In brief, a mutually agreed upon framework does not appear to exist.[82]

Research and discovery

In 1923, Karl Lashley conducted experiments on rhesus monkeys that demonstrated changes in neuronal pathways, which he concluded were evidence of plasticity. Despite this, and other research that suggested plasticity took place, neuroscientists did not widely accept the idea of neuroplasticity.

In 1945, Justo Gonzalo concluded from his research of brain dynamics, that, contrary to the activity of the projection areas, the "central" cortical mass (more or less equidistant from the visual, tactile and auditive projection areas), would be a "maneuvering mass", rather unspecific or multisensory, with capacity to increase neural excitability and re-organize the activity by means of plasticity properties.[83] He gives as a first example of adaptation, to see upright with reversing glasses in the Stratton experiment,[84] and specially, several first-hand brain injuries cases in which he observed dynamic and adaptive properties in their disorders, in particular in the inverted perception disorder [e.g., see pp 260–62 Vol. I (1945), p 696 Vol. II (1950)].[83] He stated that a sensory signal in a projection area would be only an inverted and constricted outline that would be magnified due to the increase in recruited cerebral mass, and re-inverted due to some effect of brain plasticity, in more central areas, following a spiral growth.[85]

Marian Diamond of the University of California, Berkeley, produced the first scientific evidence of anatomical brain plasticity, publishing her research in 1964.[86][2]

Other significant evidence was produced in the 1960s and after, notably from scientists including Paul Bach-y-Rita, Michael Merzenich along with Jon Kaas, as well as several others.[81][87]

In the 1960s, Paul Bach-y-Rita invented a device that was tested on a small number of people, and involved a person sitting in a chair, in which were embedded nubs that were made to vibrate in ways that translated images received in a camera, allowing a form of vision via sensory substitution.[88][89]

Studies in people recovering from stroke also provided support for neuroplasticity, as regions of the brain remained healthy could sometimes take over, at least in part, functions that had been destroyed; Shepherd Ivory Franz did work in this area.[90][91]

Eleanor Maguire documented changes in hippocampal structure associated with acquiring the knowledge of London's layout in local taxi drivers.[92][93][94] A redistribution of grey matter was indicated in London Taxi Drivers compared to controls. This work on hippocampal plasticity not only interested scientists, but also engaged the public and media worldwide.

Michael Merzenich is a neuroscientist who has been one of the pioneers of neuroplasticity for over three decades. He has made some of "the most ambitious claims for the field – that brain exercises may be as useful as drugs to treat diseases as severe as schizophrenia – that plasticity exists from cradle to the grave, and that radical improvements in cognitive functioning – how we learn, think, perceive, and remember are possible even in the elderly."[88] Merzenich's work was affected by a crucial discovery made by David Hubel and Torsten Wiesel in their work with kittens. The experiment involved sewing one eye shut and recording the cortical brain maps. Hubel and Wiesel saw that the portion of the kitten's brain associated with the shut eye was not idle, as expected. Instead, it processed visual information from the open eye. It was "...as though the brain didn't want to waste any 'cortical real estate' and had found a way to rewire itself."[88]

This implied neuroplasticity during the critical period. However, Merzenich argued that neuroplasticity could occur beyond the critical period. His first encounter with adult plasticity came when he was engaged in a postdoctoral study with Clinton Woosley. The experiment was based on observation of what occurred in the brain when one peripheral nerve was cut and subsequently regenerated. The two scientists micromapped the hand maps of monkey brains before and after cutting a peripheral nerve and sewing the ends together. Afterwards, the hand map in the brain that they expected to be jumbled was nearly normal. This was a substantial breakthrough. Merzenich asserted that, "If the brain map could normalize its structure in response to abnormal input, the prevailing view that we are born with a hardwired system had to be wrong. The brain had to be plastic."[88] Merzenich received the 2016 Kavli Prize in Neuroscience "for the discovery of mechanisms that allow experience and neural activity to remodel brain function."[95]

See also

- Activity-dependent plasticity
- Environmental enrichment (neural)
- Neural backpropagation
- Neuroplastic effects of pollution
- Kinesiology
- Lumosity

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