



I'm not robot



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Child is sensitive to noise

Child is sensitive to loud noises. My child is overly sensitive to noise. My child is sensitive to loud noise. Why is my child sensitive to loud noises. What does it mean when a child is sensitive to loud noises. My child is very sensitive to noise. My child is sensitive to noise.

nature phase in the life of an organism for others, see the window of opportunity, in developmental psychology and development biology, a critical period is a maturing phase in the life of an organism during which the nervous system is particularly sensitive to certain environmental stimuli. If, for some reason, the organism does not receive the appropriate stimulus during this "critical period" to learn a certain skill or tract, it may be difficult, ultimately less successful, or even impossible, to develop certain associated functions later in life, the indispensable functions for the survival of an organism, such as vision, are particularly likely to develop during critical periods. The "critical period" also refers to the ability to acquire the first language, researchers found that people who have passed the "critical period" would not fluently acquire their first language. [1] some researchers differentiate between "strong critical periods" and "weak critical periods" (aka sensitive periods) â€¦ defining weak critical periods "I" sensitive periods "as longer periods, after which learning is still possible. [2] other researchers consider the same phenomenon. [3] for example, the critical period for the development of the binocular vision of the human child is considered between three and eight months, with sensitivity to damage that extends up to at least three years of age. further critical periods have been identified for the development of the hearing [4] and the vestibular system. [1] strong critical weak periods of critical periods of strong critical periods include monocular deprivation, filial imprint, monadal occlusion, [5] and prefrontal acquisition synthesis. [6] these traits cannot be acquired after the end of the critical period. Examples of weak critical periods include tuning of the phoneme, grammar processing, joint control, vocabulary acquisition, musical training, hearing processing, sports training and many other traits that can be greatly improved by training at any age. [7] [8] critical period mechanisms the opening of critical periods of plasticity occur in the prenatal brain and continue throughout childhood until adolescence and are very limited during adulthood, two main factors influence the opening of critical periods: cell events (i.e. changes in the molecular landscape) and sensory experience (i.e. audio, visual input, etc.) both must coincide for the critical period to open properly, on a cellular level, critical periods are characterized by the maturation of inhibitory circuits. [9] More precisely, factors such as neurotrophic factors derived from the brain (bDNF) and orthodentis homeobox 2 (otx2)At the maturation of a large class of neurons inhibitors: interneurons of Parvalbumin-positive (PV cells) [9]. Before the beginning of critical period, the modulation of this circuit is hampered by primitive factors such as polysial acid (PSA). [9] PSA acts, partly, preventing OTX2 OTX2 After opening the critical period, PSA levels decrease, allowing photovoltaic cells to maturity by activating Gabaab inhibitory receptors that facilitate remodeling of the inhibitory circuit. Artificial removal of the PSA, or the experimental manipulation of the inhibitory transmission can lead to the premature opening of the critical period. [10] [11] While the times of these molecular events seem to be partially explained by the genes of the clock, [12] the experience is crucial as the experiments of sensory deprivation were shown to interfere with the correct timing of the periods Critics. [13] [14] [15] The activity theory depend on Hebbian's theory guides the idea of competition dependent on the activity: if two neurons have both the potential to make a connection with a cell, the neuron that the most fires will do. Ocular domain This competition phenomenon dependent on the activity is particularly seen in the formation of ocular dominance columns within the visual system. At the beginning of development, most of the visual cortex is binocular, which means that it receives input approximately the same from both eyes. [16] Normally, while development progresses, the visual cortex will see to monocular columns that receive input from one eye. [16] However, if an eye is patched, or otherwise prevented to receive sensory inputs, the visual bark moves to the representation of the discovered eye. This demonstrates the competition dependent on the activity and the theory of Hebbian because the inputs from the discovered eye make and retain more connections than the patched eye. [17] Axon growth Further information: Axon axis training and growth are another fundamental part of the competition dependent on plasticity and activity. Axon's growth and branching was demonstrated to be inhibited when neuron electricity is suppressed under the level of a nearby neighbor. [18] This shows that asylum growth dynamics are not independent but rather depend on local circuits within which they are active (ie the activity of other neurons that compete for connections). Microglia Microglia microglia play a role in synaptic pruning during adolescence. As resident immune cells of the central nervous system, the main role of Microglia is phagocytosis and excitation. Studies have discovered that during critical periods in the visual cortex, neural synapses become the objective of microglial phagocytosis. [19] The neurons who received a less frequent entry from the cells of the ridge ganglion during the first postnatal periods were more inclined to be swallowed and free of Microglia, as for monocular deprivation experiments. [19] Similar results were found during manipulation G-Coupled purinergic receptors on microglial processes. Block these receptors or perform a significantly lowered elimination experiment microglial interactions and synaptic pruning during the first critical period of the visual cortex. [20] More recently, the expression of the component 4 gene of the complement has been found to contribute significantly to abnormally high levels of synaptic pruning during the early stages of development in schizophrenic and microglia neurons, suggesting a genomic connection between the immune system and critical periods. [21] The dendritic motility of the spine is the alteration of the dendritic morphology of a neuron, in particular the appearance and disappearance of the small protrusions known as thorns. In the first postnatal development, the motility of the spine was found at high levels. Due to its more pronounced occurrence during postnatal days from 11 to 15, the motility of the spine is thought to have a role in neurogenesis.[22] Motility levels decrease significantly before the beginning of the critical period of the visual cortex and monocular deprivation experiments show that motility levels decrease constantly until the critical period is over, suggesting that motility may not be explicitly involved in this process. [23] However, binocular deprivation before the eye opening led to a significant increase in the motility of the spine until the culmination of the critical period.[24] which led to controversial results regarding the role of motility of the dendritic spine. Another critical component of neuronal plasticity is the balance of exciting and inhibitor inputs. At the beginning of development, GABA, the leading inhibitory neurotransmitter in the adult brain, shows an excitatory effect on its target neurons. [25] However, due to changes in internal chloride levels due to the up-regulation of potassium chloride pumps, GABA then switches to inhibitory synaptic transmission. [25] The maturation of the GABAergic inhibitory system helps to trigger the occurrence of critical periods. [11] Enhanced GABAergic systems can lead to an early critical period, while weaker GABAergic inputs can delay or even prevent plasticity.[26][27] Inhibition also guides plasticity once the critical period has begun. For example, lateral inhibition is particularly important in columnar formation driving in the visual cortex. [28] Ebbiana theory provides a vision of the importance of inhibition within neural networks: without inhibition, there would be more synchronous and therefore more connections, but with inhibition, less excitation signals pass through, allowing only the most salient connections to mature. [29] Periodic closures for European networks The closure of the critical period was demonstrated by the maturation of inhibitory circuits, mediated by the formation of periEuropean networks around inhibiting neurons. [11] Perineuronal networks (PNN) are structures in the extracellular matrix formed by proteoglycans chondroitine sulphate, hyaluron and link protein. [30] These structures wrap the soma of theinhibitors in the central nervous system, appearing with age to stabilize mature circuits. [30][31] The development of PNN coincides with the closure of critical periods, and both the formation of PNN and the critical timing of the period are delayed in dark learning. [31] For example, PNN digestion by ABC chondroitinase in rats leads to changeeey dominance over monocular deprivation, which is normally limited to its critical period long before in development. [32] Furthermore, PNNs are charged negatively, which is theorized to create a cation-rich environment around cells, potentially leading to an increase in the combustion rate of inhibiting neurons, thus allowing an increase in inhibition after the formation of PNNs and helping to close the critical period. [31] The role of the PNN in the closing of critical period is further supported by the finding that the parvalbumin-positive interneurons with rapid spiking are also surrounded by PNN.[33] Peri-European networks have also been found to control chemorepulsive factors such as semaphorin3A, which may limit the growth of the axits necessary for plasticity during critical periods. [34] In all these data suggest a role for PNNs in the maturation of CNS inhibition, in the prevention of plastic association growth, and subsequently the critical closure of the period. Myelin Myelin formation is also a key event in the closing of critical periods. Myelin sheaths are formed by oligodendrocytes in the CNS that wraps segments of axons to increase their cooking speed. [35] Myelin is formed in the early stages of development and progresses in the waves, with later phylogenetic development brain areas (i.e. those associated with higher brain functions such as frontal lobes) which have subsequently reappointed. [36] The maturation of myelination in intracortical layers coincides with the critical closure of the period in mice, which led to further research on the role of myelination on the critical duration of the period. [37] Myelin is known to bind many different assonic growth inhibitors that prevent plasticity seen in critical periods. [38] The Nogo Receptor is expressed in miline and binds to the assonic growth inhibitors Nogo and MAG (among others), preventing the growth of axone in mature and milinated neurons. [38] Instead of affecting the timing of the critical period, Nogo receptor mutations temporarily prolong the critical period. [37] A Nogo receptor mutation in mice was found to extend the critical period for monocular dominance from about 20 - 32 days to 45 to 120 days, suggesting a probable role of myelin Nogo receptor in the critical closure of the period. [37] Moreover, the effects of my elimination are limited temporally, since my elimination itself can have its own critical period and timing. [36] [39] Research has shown that the social isolation of mice leads to reduce the thickness of myelin and to a poor working memory, but only during a critical period of youth. [39] In primates, isolation is correlated with abnormal changes in white matter potentially due to a decrease in myelination. [40] In everything, myelin and its receptorsThey bind different important axial growth factors and help close critical period. [37] [38] The timing of this myelination process is also influenced by external factors such as the environment. [36] [39] Factors that influence the timing of myelination include genetic and ethical impermeability of replicating them make it difficult to draw conclusions on them. Children may have been disabled for childhood, or their inability to develop language could be derived from the deep intelligence and abuse they suffered [52] Many later researchers have further developed CPH, especially Elissa Newport and Rachel Mayberry. Studies conducted by these researchers have shown that deeply hearing individuals who are not exposed to a sign language since children never reach full competence, even after 30 years of daily use. [54] While the effect is deeper for people who do not receive sign language entry until after 12 years, even those deaf who started learning a sign language at the age of 5 were significantly less fluent than native deafers (whose exposure to a sign language began at birth). Early language exposure also affects the ability to learn a second language later in life: deaf individuals with early-language exposure reach comparable levels of competence in a second language to individuals with early-language exposure. Contrary to this, the unheard individuals without early-language exposure are much worse. [55] Other evidence comes from neuropsychology in which it is known that adults well beyond the critical period are more likely to suffer a permanent linguistic impairment from brain damage than children, considered due to the youth resilience of neural reorganization [52]. Steven Pinker discusses CPH in his book, the language instinct. According to Pinker, themust be seen as a concept rather than a specific language because sounds, grammar, meaning, vocabulary and social norms play an important role in language acquisition. [56] The physiological changes in the brain are also provocative causes for the terminus of the critical period for languageBecause the acquisition of language is crucial at this stage, similarly the attachment of child parents is crucial for the child's social development. A child learns to trust and feel safe with the parent, but there are cases in which the child could remain in an orphanage where he does not receive the same attachment with their caregiver. Research shows that children who have not been able to develop this attachment have more difficulties in maintaining relationships, and have had more behavioral problems during the period adopted. [11] The discussion of the linguistic critical period suffers from lack of a commonly accepted language definition. Some aspects of language, such as phonetic optimization, grammar processing, critical joint control and vocabulary acquisition, can be significantly improved by forming at any ages and therefore have weak critical periods. [7] [8] Other aspects of language, such as prefrontal synthesis, have strong critical periods and cannot be acquired after the end of the critical period. [6] Consequently, when the language is discussed in general, without dissection in the components, the arguments can be built both in favor and against the strong critical period of the acquisition L1. Acquisition of the second language The theory [58] has often been extended to a critical period for the acquisition of a second language (SLA), which influenced researchers on the field on both sides of the spectrum, support and non-support of CPH, to explore. [59] However, the nature of this phenomenon was one of the most fiercely discussed issues in psycholing and cognitive science in general for decades. Certainly, the older students of a second language rarely reach the native flu-like that younger students show, although they often progress more quickly than children in the early stages. This is generally accepted as proof that supports the CPH. Incorporating the idea, "the same equal younger" by Penfield, David Singleton (1995) states that in learning a second language there are many exceptions, noticing that five percent of adult bilingual master of a second language even if they start to Learn when they are well in adulthood â€¦ "very after any critical period has presumably reached a conclusion. The hypothesis of the critical period argues that the acquisition of the first language must take place before the cerebral lateralization complete, at about the ages of puberty. A forecast of this hypothesis is that the acquisition of the second language is relatively fast, of successful and qualitatively similar to the first language only if it occurs before the EÂ de la PubertÃ . [60] To better understand the SLA, it is essential to consider linguistic, cognitive and social factors rather than etA alone, as they are all for the acquisition of the instructor's language. [59] Over the years, many experimenters have tried to find evidence in support or against critical periods for the acquisition of the second language. Many have found evidence that small children acquire the language more easily than adults, but there are special cases of adults who acquire a second language with native-similar competence. So it was difficult for researchers to separate the correlation from causality. [61] In 1989, Jacqueline S. Johnson and Elissa L. Newport found support for the claim that second languages are more easily acquired before puberty, or more specifically before the age of seven. [62] They tested students of the second language of English, who arrived in the United States at various ages from three to thirty-nine and found that there was a decline in grammatical correctness after the age of seven. Johnson and Newport have attributed this statement of a decrease in language learning ability with age. Opponents of the critical period argue that the difference in language skills found by Johnson and Newport could be due to the different types of input that children and adults receive; Children received a reduced entrance while adults receive more complicated facilities. Further evidence against a severe critical period is also found in the work of Pallier et al. (2003) Those who discovered that children adopted in France, Korea, were able to become natives in their French performance even after the critical period for phonology. [63] Their experiment can be a special case in which subjects must lose their first language to acquire their second one more perfectly. There is also a little debate on how you can judge the native quality of the participants in the produced speakers and what exactly it means to be a native native speaker of a second language. [64] White et al. Found that it is possible that non-native speakers of a language become native in some aspects, but such aspects are influenced by their first language. Recently, a link model has been developed to explain the changes that take place in learning the second language assuming that the sensitive period affects the lexical learning and the syntactic learning parts of the system differently, which remains a further light on how the first and second acquisition of the language changes the course of student development [65] Vision in mammals, neurons in the brain that the vision process actually develops after the eyes. An experiment of a reference point of David H. Hubel and Torsten Wiesel (1963) geniculocortical axonal arbori in monocularly long-term private animals (4-weeks) and short-term (6-7 days) during the critical period established by Hubel and Wiesel (1963). They have discovered that in the long term, monocular deprivation causes reduced branching at the end of neurons, while the quantity of afferent axons to the non-private eye has increased. Even in the short term, Antonini and Stryker (1993) discovered that geniculocortical neurons were equally affected. This supports the concept of a critical period for a correct neural development for vision in the cortex. [68] The studies of persons whose sight was restored after a long ceceta (both from the birth that from a subsequent point of life) reveal that they cannot necessarily recognize objects and faces (as opposed to color, movement and simple geometric shapes). Some hypothetical that being blind during childhood prevent a part of the visual system necessary for these higher level tasks to be developed correctly. [69] The general conviction that a critical period lasts up to 5 or 6 years is Contested by a 2007 study that found that elderly patients could improve these skills with years of exposure. [70] The expression of the LYNXI protein has been associated with the normal end of the critical period for syntactic plasticity in the visual system. [71] [71] Konrad Lorenz in Psychology, imprinting is any type of rapid learning that occurs in a particular phase of life. While this rapidly learning is independent of the behavioral result, it also establishes it and can influence behavioral responses to different stimuli. Konrad Lorenz is well known for his classical studies of affiliate imprinting in Graylag geese. From 1935 to 1938, he presented himself to a new-born ban group and took note of how he was immediately accepted, followed, and called as if he were himself. As the first moving object encountered, Lorenz studied the phenomenon as quickly geese were able to form such an irreversible bond. Through his work he showed that this developed only during a short period "Critical period", which was about a few hours after hatching, suggesting a strong critical period. [72] Lorenz also discovered a lasting effect of his studies, and this was a shift in the sexual imprinting of the species as a result of imprinting on an adoptive mother of a second species. For certain species, if raised by a second, develop and maintain imprinted preferences, and approach the second species that have been raised rather than choose their own, if given a choice [73]. The footprint serves as the distinctive factor for humans. The mother and her mother's figures. The mother and the mother's figures. This is a strong moment of bonding for humans. It provides a kind of model or guide to adult behavior as well as other factors such as nutrition, child protection, guidance and nutrition. Also the process of imprint, Lorenz also found, brought a sense of familiarity for young animals. When such a strong bond is formed at such an early stage, it creates a sense of safety and comfort for the subject and in reality encourages the behavior of imprinting. Pheromones play a key role in the fingerprint process, activate a biochemical response in the recipient, leading to a confirmed identification in the other individual. If direct contact between mother and child is not maintained during the period of critical imprint, then the goose mother can refuse the child because it is unfamiliar with the perfume of her newborn. If this happens, then the life of the child would be in danger unless it was claimed by a substitute mother, possibly leading to embarrassing social behaviors in the next life. [74] In relation to human beings, a newborn during the critical period identifies with the perfumes of his mother and other peoples since his perfume is one of the most developed senses at that point of life. The newborn uses this identification of pheromones to search for the people with whom he identifies, when in times of anguish, hunger and discomfort as a skill of[75] Inferences could be made for infants based on Lorenz's studies. When the imprint of their mothers, newborns look at them for nourishment, a sense of safety and comfort. Human infants are among the most targeted kinds withRanking of second newborns. The newborns of these species have a very limited series of survival innate. Their most important and functional ability is to form links with neighboring individuals who are able to keep them alive. Imprinting is a crucial factor of the critical period because it influences the ability of the newborn to form links with other individuals from childhood to adulthood. Holding processing Many studies have supported a correlation between the type of hearing stimuli present in the first postnatal environment and in the development of topographic and structural development of the auditory system. [4] The first reports on critical periods came from children and deaf animals that received a cochlear plant to restore hearing. Approximately at the same time, both an electroencephalographic study of Sharma, Dorman and Spahr [76] and in a vivo survey of cortical plasticity in the non-hearing cats of Kral and colleagues [77] has shown that adaptation to the cochlear implant is it is subject to an early and developmental sensitive period. The closure of sensitive periods probably involves a multitude of processes that in their combination make it difficult to reopen these behind. [4] The understanding of the mechanisms behind critical periods has consequences for the medical therapy of hearing loss. [78] M. Merzenich and colleagues showed that during an early critical period, noise exposure can affect the organization of the auditory cortex frequency [79]. Recent studies have examined the possibility of a critical period for the thalamocortical connectivity in the auditory system. For example, Zhou and Merzenich (2008) studied the effects of noise on development in the primary hearing cortex in rats. In their study, rats were exposed to impulse noise during the critical period and the effect on cortical processing was measured. The rats that were exposed to pulsed noise during the critical period had cortical neurons that were less able to respond to repeated stimuli; The earlier auditory environment has interrupted the normal structural organization during development. In a related study, Barkat, Polley and Hensch (2011) examined how exposure to different audio frequencies influences the development of the tonotopic map in the primary auditory cortex and the ventral gene body. In this experiment, the mice were bred in normal environments or in the presence of 7 KHz tones during their first postnatal days. They found that mice that were exposed to an abnormal hearing environment during a critical period P11- P15 had an atypical tonotopic map in the primary hearing cortex. [80] These studies support the notion that exposure to certain sounds within the critical period can influence the tonotopic map in the primary hearing cortex. [80] The effects of noise on development in the primary hearing cortex in rats. 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