



Camp Delta Standard Operating Procedure

Description

Psychology played an important role in the development of the methodologies. Sensory deprivation is a pivotal field of study in this context (see attached references below).

The **Camp Delta Standard Operating Procedures** is a document that was written under the authority of [Geoffrey D. Miller](#) when he was the officer in charge of [Joint Task Force Guantanamo](#).^{[1][2]} This leaked document was published on [WikiLeaks](#) on Wednesday November 7, 2007.

According to [Wired](#) magazine, the 238-page document was dated March 28, 2003, and was signed by Miller.^[1] [Wired](#) reports that the [American Civil Liberties Union](#) had requested the release of the document under the [Freedom of Information Act](#) in October 2003.

[Wired](#) reports that Miller specified four levels of access the [International Committee of the Red Cross](#) (ICRC) would be allowed to captives:^[1]

- No Access
- Visual Access – ICRC can only look at a prisoner's physical condition.
- Restricted Access – ICRC representatives can only ask short questions about the prisoner's health.
- Unrestricted Access

[Wired](#) reports that spokesmen from the [Department of Defense](#) declined to comment on the leak.^[1] The [Associated Press](#) reports that [Army Lieutenant Colonel Ed Bush](#) called the manual out-of-date.^[2] According to Lieutenant Colonel Bush, AP reports, dogs are no longer used, and the Red Cross is no longer denied access to any of the captives.

en.wikipedia.org/wiki/Camp_Delta_Standard_Operating_Procedures

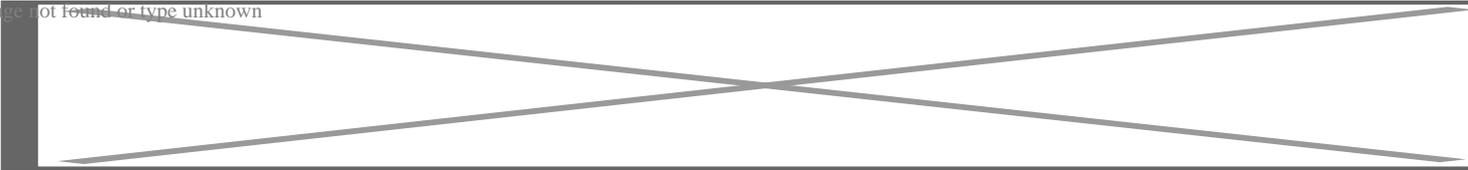
Joint Task Force Guantanamo (JTF-GTMO) standard operating procedures (SOP) for Camp Delta (Guantanamo Bay prison). This is the primary document for the operation of Guantanamo bay,

including the securing and treatment of detainees. The document is extensive and includes, in addition to text various forms, identity cards and even Muslim burial instructions. It is signed by Major General Miller, who Donald Rumsfeld later sent to Abu Ghraib to "Gitmoize it". The document is also the subject of an ongoing legal action between the ACLU, which has been trying to obtain it, and the Department of Defense, which has withheld it in full (see www.aclu.org/pdfs/safefree/20070110/dod_vaughn_r_denied_in_full_section_6_interim.pdf).

wikileaks.org/wiki/Camp_Delta_Standard_Operating_Procedure

hrlibrary.umn.edu/OathBetrayed/sop_2004.pdf

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Further References

Merabet, L. B., & Pascual-Leone, A.. (2010). Neural reorganization following sensory loss: The opportunity of change. *Nature Reviews Neuroscience*

Plain numerical DOI: 10.1038/nrn2758

[DOI URL](#)

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"There is growing evidence that sensory deprivation is associated with crossmodal neuroplastic changes in the brain. after visual or auditory deprivation, brain areas that are normally associated with the lost sense are recruited by spared sensory modalities. these changes underlie adaptive and compensatory behaviours in blind and deaf individuals. although there are differences between these populations owing to the nature of the deprived sensory modality, there seem to be common principles regarding how the brain copes with sensory loss and the factors that influence neuroplastic changes. here, we discuss crossmodal neuroplasticity with regards to behavioural adaptation after sensory deprivation and highlight the possibility of maladaptive consequences within the context of rehabilitation."

Goel, N., Rao, H., Durmer, J. S., & Dinges, D. F.. (2009). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology*

Plain numerical DOI: 10.1055/s-0029-1237117

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"Sleep deprivation is associated with considerable social, financial, and health-related costs, in large

measure because it produces impaired cognitive performance due to increasing sleep propensity and instability of waking neurobehavioral functions. cognitive functions particularly affected by sleep loss include psychomotor and cognitive speed, vigilant and executive attention, working memory, and higher cognitive abilities. chronic sleep-restriction experiments—which model the kind of sleep loss experienced by many individuals with sleep fragmentation and premature sleep curtailment due to disorders and lifestyle—demonstrate that cognitive deficits accumulate to severe levels over time without full awareness by the affected individual. functional neuroimaging has revealed that frequent and progressively longer cognitive lapses, which are a hallmark of sleep deprivation, involve distributed changes in brain regions including frontal and parietal control areas, secondary sensory processing areas, and thalamic areas. there are robust differences among individuals in the degree of their cognitive vulnerability to sleep loss that may involve differences in prefrontal and parietal cortices, and that may have a basis in genes regulating sleep homeostasis and circadian rhythms. thus, cognitive deficits believed to be a function of the severity of clinical sleep disturbance may be a product of genetic alleles associated with differential cognitive vulnerability to sleep loss.”

Zheng, J. J., Li, S. J., Zhang, X. Di, Miao, W. Y., Zhang, D., Yao, H., & Yu, X.. (2014). Oxytocin mediates early experience-dependent cross-modal plasticity in the sensory cortices. *Nature Neuroscience*

Plain numerical DOI: 10.1038/nn.3634

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“Sensory experience is critical to development and plasticity of neural circuits. here we report a new form of plasticity in neonatal mice, where early sensory experience cross-modally regulates development of all sensory cortices via oxytocin signaling. unimodal sensory deprivation from birth through whisker deprivation or dark rearing reduced excitatory synaptic transmission in the correspondent sensory cortex and cross-modally in other sensory cortices. sensory experience regulated synthesis and secretion of the neuropeptide oxytocin as well as its level in the cortex. both in vivo oxytocin injection and increased sensory experience elevated excitatory synaptic transmission in multiple sensory cortices and significantly rescued the effects of sensory deprivation. together, these results identify a new function for oxytocin in promoting cross-modal, experience-dependent cortical development. this link between sensory experience and oxytocin is particularly relevant to autism, where hypersensitivity or hyposensitivity to sensory inputs is prevalent and oxytocin is a hotly debated potential therapy.”

Hofer, S. B., Mrsic-Flogel, T. D., Bonhoeffer, T., & Hübener, M.. (2009). Experience leaves a lasting structural trace in cortical circuits. *Nature*

Plain numerical DOI: 10.1038/nature07487

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“Sensory experiences exert a powerful influence on the function and future performance of neuronal circuits in the mammalian neocortex. restructuring of synaptic connections is believed to be one

mechanism by which cortical circuits store information about the sensory world. excitatory synaptic structures, such as dendritic spines, are dynamic entities that remain sensitive to alteration of sensory input throughout life. it remains unclear, however, whether structural changes at the level of dendritic spines can outlast the original experience and thereby provide a morphological basis for long-term information storage. here we follow spine dynamics on apical dendrites of pyramidal neurons in functionally defined regions of adult mouse visual cortex during plasticity of eye-specific responses induced by repeated closure of one eye (monocular deprivation). the first monocular deprivation episode doubled the rate of spine formation, thereby increasing spine density. this effect was specific to layer-5 cells located in binocular cortex, where most neurons increase their responsiveness to the non-deprived eye. restoring binocular vision returned spine dynamics to baseline levels, but absolute spine density remained elevated and many monocular deprivation-induced spines persisted during this period of functional recovery. however, spine addition did not increase again when the same eye was closed for a second time. this absence of structural plasticity stands out against the robust changes of eye-specific responses that occur even faster after repeated deprivation. thus, spines added during the first monocular deprivation experience may provide a structural basis for subsequent functional shifts. these results provide a strong link between functional plasticity and specific synaptic rearrangements, revealing a mechanism of how prior experiences could be stored in cortical circuits."

Lendvai, B., Stern, E. A., Chen, B., & Svoboda, K.. (2000). Experience-dependent plasticity of dendritic spines in the developing rat barrel cortex in vivo. *Nature*

Plain numerical DOI: 10.1038/35009107

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"Do changes in neuronal structure underlie cortical plasticity? here we used time-lapse two-photon microscopy of pyramidal neurons in layer 2/3 of developing rat barrel cortex to image the structural dynamics of dendritic spines and filopodia. we found that these protrusions were highly motile: spines and filopodia appeared, disappeared or changed shape over tens of minutes. to test whether sensory experience drives this motility we trimmed whiskers one to three days before imaging. sensory deprivation markedly (approximately 40%) reduced protrusive motility in deprived regions of the barrel cortex during a critical period around postnatal days (p)11-13, but had no effect in younger (p8-10) or older (p14-16) animals. unexpectedly, whisker trimming did not change the density, length or shape of spines and filopodia. however, sensory deprivation during the critical period degraded the tuning of layer 2/3 receptive fields. thus sensory experience drives structural plasticity in dendrites, which may underlie the reorganization of neural circuits."

Maffei, A., Nataraj, K., Nelson, S. B., & Turrigiano, G. G.. (2006). Potentiation of cortical inhibition by visual deprivation. *Nature*

Plain numerical DOI: 10.1038/nature05079

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"The fine-tuning of circuits in sensory cortex requires sensory experience during an early critical period."

visual deprivation during the critical period has catastrophic effects on visual function, including loss of visual responsiveness to the deprived eye, reduced visual acuity, and loss of tuning to many stimulus characteristics. these changes occur faster than the remodelling of thalamocortical axons, but the intracortical plasticity mechanisms that underlie them are incompletely understood. long-term depression of excitatory intracortical synapses has been proposed as a general candidate mechanism for the loss of cortical responsiveness after visual deprivation. alternatively (or in addition), the decreased ability of the deprived eye to activate cortical neurons could be due to enhanced intracortical inhibition. here we show that visual deprivation leaves excitatory connections in layer 4 (the primary input layer to cortex) unaffected, but markedly potentiates inhibitory feedback between fast-spiking basket cells (fs cells) and star pyramidal neurons (star pyramids). further, a previously undescribed form of long-term potentiation of inhibition (ltpi) could be induced at synapses from fs cells to star pyramids, and was occluded by previous visual deprivation. these data suggest that potentiation of inhibition is a major cellular mechanism underlying the deprivation-induced degradation of visual function, and that this form of ltpi is important in fine-tuning cortical circuitry in response to visual experience."

Margolis, D. J., Lütcke, H., Schulz, K., Haiss, F., Weber, B., Kügler, S., ... Helmchen, F.. (2012). Reorganization of cortical population activity imaged throughout long-term sensory deprivation. *Nature Neuroscience*

Plain numerical DOI: 10.1038/nn.3240

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"Sensory maps are reshaped by experience. it is unknown how map plasticity occurs in vivo in functionally diverse neuronal populations because activity of the same cells has not been tracked over long time periods. here we used repeated two-photon imaging of a genetic calcium indicator to measure whisker-evoked responsiveness of the same layer 2/3 neurons in adult mouse barrel cortex over weeks, first with whiskers intact, then during continued trimming of all but one whisker. across the baseline period, neurons displayed heterogeneous yet stable responsiveness. during sensory deprivation, responses to trimmed whisker stimulation globally decreased, whereas responses to spared whisker stimulation increased for the least active neurons and decreased for the most active neurons. these findings suggest that recruitment of inactive, 'silent' neurons is part of a convergent redistribution of population activity underlying sensory map plasticity. sensory-driven responsiveness is a key property controlling experience-dependent activity changes in individual neurons."

Celikel, T., Szostak, V. A., & Feldman, D. E.. (2004). Modulation of spike timing by sensory deprivation during induction of cortical map plasticity. *Nature Neuroscience*

Plain numerical DOI: 10.1038/nn1222

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"Deprivation-induced plasticity of sensory cortical maps involves long-term potentiation (ltp) and depression (ltd) of cortical synapses, but how sensory deprivation triggers ltp and ltd in vivo is

unknown. here we tested whether spike timing-dependent forms of ltp and ltd are involved in this process. we measured spike trains from neurons in layer 4 (l4) and layers 2 and 3 (l2/3) of rat somatosensory cortex before and after acute whisker deprivation, a manipulation that induces whisker map plasticity involving ltd at l4-to-l2/3 (l4-l2/3) synapses. whisker deprivation caused an immediate reversal of firing order for most l4 and l2/3 neurons and a substantial decorrelation of spike trains, changes known to drive timing-dependent ltd at l4-l2/3 synapses in vitro. in contrast, spike rate changed only modestly. thus, whisker deprivation is likely to drive map plasticity by spike timing-dependent mechanisms."

Yashiro, K., Riday, T. T., Condon, K. H., Roberts, A. C., Bernardo, D. R., Prakash, R., ... Philpot, B. D.. (2009). Ube3a is required for experience-dependent maturation of the neocortex. *Nature Neuroscience*

Plain numerical DOI: 10.1038/nn.2327

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"Experience-dependent maturation of neocortical circuits is required for normal sensory and cognitive abilities, which are distorted in neurodevelopmental disorders. we tested whether experience-dependent neocortical modifications require ube3a, an e3 ubiquitin ligase whose dysregulation has been implicated in autism and angelman syndrome. using visual cortex as a model, we found that experience-dependent maturation of excitatory cortical circuits was severely impaired in angelman syndrome model mice deficient in ube3a. this developmental defect was associated with profound impairments in neocortical plasticity. normal plasticity was preserved under conditions of sensory deprivation, but was rapidly lost by sensory experiences. the loss of neocortical plasticity is reversible, as late-onset visual deprivation restored normal synaptic plasticity. furthermore, ube3a-deficient mice lacked ocular dominance plasticity in vivo when challenged with monocular deprivation. we conclude that ube3a is necessary for maintaining plasticity during experience-dependent neocortical development and suggest that the loss of neocortical plasticity contributes to deficits associated with angelman syndrome."

Kral, A., & Sharma, A.. (2012). Developmental neuroplasticity after cochlear implantation. *Trends in Neurosciences*

Plain numerical DOI: 10.1016/j.tins.2011.09.004

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"Cortical development is dependent on stimulus-driven learning. the absence of sensory input from birth, as occurs in congenital deafness, affects normal growth and connectivity needed to form a functional sensory system, resulting in deficits in oral language learning. cochlear implants bypass cochlear damage by directly stimulating the auditory nerve and brain, making it possible to avoid many of the deleterious effects of sensory deprivation. congenitally deaf animals and children who receive implants provide a platform to examine the characteristics of cortical plasticity in the auditory system. in this review, we discuss the existence of time limits for, and mechanistic constraints on, sensitive periods for cochlear implantation and describe the effects of multimodal and cognitive reorganization

that result from long-term auditory deprivation. © 2011."

Hooks, B. M., & Chen, C.. (2006). Distinct Roles for Spontaneous and Visual Activity in Remodeling of the Retinogeniculate Synapse. *Neuron*

Plain numerical DOI: 10.1021/acssensors.5b00047

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"Sensory experience and spontaneous activity play important roles in development of sensory circuits; however, their relative contributions are unclear. here, we test the role of different forms of activity on remodeling of the mouse retinogeniculate synapse. we found that the bulk of maturation occurs without patterned sensory activity over 4 days spanning eye opening. during this early developmental period, blockade of spontaneous retinal activity by tetrodotoxin, but not visual deprivation, retarded synaptic strengthening and inhibited pruning of excess retinal afferents. later in development, synaptic remodeling becomes sensitive to changes in visually evoked activity, but only if there has been previous visual experience. synaptic strengthening and pruning were disrupted by visual deprivation following 1 week of vision, but not by chronic deprivation from birth. thus, spontaneous activity is necessary to drive the bulk of synaptic refinement around the time of eye opening, while sensory experience is important for the subsequent maintenance of connections. © 2006 elsevier inc. all rights reserved."

Zuo, Y., Yang, G., Kwon, E., & Gan, W. B.. (2005). Long-term sensory deprivation prevents dendritic spine loss in primary somatosensory cortex. *Nature*

Plain numerical DOI: 10.1038/nature03715

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"A substantial decrease in the number of synapses occurs in the mammalian brain from the late postnatal period until the end of life. although experience plays an important role in modifying synaptic connectivity, its effect on this nearly lifelong synapse loss remains unknown. here we used transcranial two-photon microscopy to visualize postsynaptic dendritic spines in layer i of the barrel cortex in transgenic mice expressing yellow fluorescent protein. we show that in young adolescent mice, long-term sensory deprivation through whisker trimming prevents net spine loss by preferentially reducing the rate of ongoing spine elimination, not by increasing the rate of spine formation. this effect of deprivation diminishes as animals mature but still persists in adulthood. restoring sensory experience after adolescent deprivation accelerates spine elimination. similar to sensory manipulation, the rate of spine elimination decreases after chronic blockade of nmda (n-methyl-d-aspartate) receptors with the antagonist mk801, and accelerates after drug withdrawal. these studies of spine dynamics in the primary somatosensory cortex suggest that experience plays an important role in the net loss of synapses over most of an animal's lifespan, particularly during adolescence."

Hofer, S. B., Mrsic-Flogel, T. D., Bonhoeffer, T., & Hübener, M.. (2006). Prior experience enhances plasticity in adult visual cortex.

Nature Neuroscience

Plain numerical DOI: 10.1038/nn1610

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“The brain has a remarkable capacity to adapt to alterations in its sensory environment, which is normally much more pronounced in juvenile animals. here we show that in adult mice, the ability to adapt to changes can be improved profoundly if the mouse has already experienced a similar change in its sensory environment earlier in life. using the standard model for sensory plasticity in mouse visual cortex-ocular dominance (od) plasticity-we found that a transient shift in od, induced by monocular deprivation (md) earlier in life, renders the adult visual cortex highly susceptible to subsequent md many weeks later. irrespective of whether the first md was experienced during the critical period (around postnatal day 28) or in adulthood, od shifts induced by a second md were faster, more persistent and specific to repeated deprivation of the same eye. the capacity for plasticity in the mammalian cortex can therefore be conditioned by past experience.”

Keck, T., Keller, G. B., Jacobsen, R. I., Eysel, U. T., Bonhoeffer, T., & Hübener, M.. (2013). Synaptic scaling and homeostatic plasticity in the mouse visual cortex in vivo. *Neuron*

Plain numerical DOI: 10.1016/j.neuron.2013.08.018

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“Homeostatic plasticity is important to maintain a set level of activity in neuronal circuits and has been most extensively studied in cell cultures following activity blockade. it is still unclear, however, whether activity changes associated with mechanisms of homeostatic plasticity occur invivo, for example after changes in sensory input. here, we show that activity levels in the visual cortex are significantly decreased after sensory deprivation by retinal lesions, followed by a gradual increase in activity levels in the 48hr after deprivation. these activity changes are associated with synaptic scaling, manifested invitro by an increase in mepsc amplitude and invivo by an increase in spine size. together, these data show that homeostatic activity changes occur invivo in parallel with synaptic scaling”

Petrus, E., Isaiah, A., Jones, A. P., Li, D., Wang, H., Lee, H. K., & Kanold, P. O.. (2014).

Crossmodal Induction of Thalamocortical Potentiation Leads to Enhanced Information Processing in the Auditory Cortex. *Neuron*

Plain numerical DOI: 10.1016/j.neuron.2013.11.023

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“Sensory systems do not work in isolation; instead, they show interactions that are specifically

uncovered during sensory loss. to identify and characterize these interactions, we investigated whether visual deprivation leads to functional enhancement in primary auditory cortex (a1). we compared sound-evoked responses of a1 neurons in visually deprived animals to those from normally reared animals. here, we show that visual deprivation leads to improved frequency selectivity as well as increased frequency and intensity discrimination performance of a1 neurons. furthermore, we demonstrate invitro that in adults visual deprivation strengthens thalamocortical (tc) synapses in a1, but not in primary visual cortex (v1). because deafening potentiated tc synapses in v1, but not a1, crossmodal tc potentiation seems to be a general property of adult cortex. our results suggest that adults retain the capability for crossmodal changes whereas such capability is absent within a sensory modality. thus, multimodal training paradigms might be beneficial in sensory-processing disorders. © 2014 elsevier inc."

Makino, H., & Malinow, R.. (2011). Compartmentalized versus global synaptic plasticity on dendrites controlled by experience. *Neuron*

Plain numerical DOI: 10.1016/j.neuron.2011.09.036

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"Synapses in the brain are continuously modified by experience, but the mechanisms are poorly understood. in vitro and theoretical studies suggest threshold-lowering interactions between nearby synapses that favor clustering of synaptic plasticity within a dendritic branch. here, a fluorescently tagged ampa receptor-based optical approach was developed permitting detection of single-synapse plasticity in mouse cortex. sensory experience preferentially produced synaptic potentiation onto nearby dendritic synapses. such clustering was significantly reduced by expression of a phospho-mutant ampa receptor that is insensitive to threshold-lowering modulation for plasticity-driven synaptic incorporation. in contrast to experience, sensory deprivation caused homeostatic synaptic enhancement globally on dendrites. clustered synaptic potentiation produced by experience could bind behaviorally relevant information onto dendritic subcompartments; global synaptic upscaling by deprivation could equally sensitize all dendritic regions for future synaptic input. © 2011 elsevier inc."

Keck, T., Scheuss, V., Jacobsen, R. I., Wierenga, C. J., Eysel, U. T., Bonhoeffer, T., & Hübener, M.. (2011). Loss of sensory input causes rapid structural changes of inhibitory neurons in adult mouse visual cortex. *Neuron*

Plain numerical DOI: 10.1016/j.neuron.2011.06.034

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"A fundamental property of neuronal circuits is the ability to adapt to altered sensory inputs. it is well established that the functional synaptic changes underlying this adaptation are reflected by structural modifications in excitatory neurons. in contrast, the degree to which structural plasticity in inhibitory neurons accompanies functional changes is less clear. here, we use two-photon imaging to monitor the fine structure of inhibitory neurons in mouse visual cortex after deprivation induced by retinal lesions. we find that a subset of inhibitory neurons carry dendritic spines, which form glutamatergic synapses.

removal of visual input correlates with a rapid and lasting reduction in the number of inhibitory cell spines. similar to the effects seen for dendritic spines, the number of inhibitory neuron boutons dropped sharply after retinal lesions. together, these data suggest that structural changes in inhibitory neurons may precede structural changes in excitatory circuitry, which ultimately result in functional adaptation following sensory deprivation. © 2011 elsevier inc."

Villanueva, M., García, B., Valle, J., Rapún, B., Ruiz De Los Mozos, I., Solano, C., ... Lasa, I.. (2018). Sensory deprivation in *Staphylococcus aureus*. *Nature Communications*

Plain numerical DOI: 10.1038/s41467-018-02949-y

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"Bacteria use two-component systems (tcss) to sense and respond to environmental changes. the core genome of the major human pathogen *staphylococcus aureus* encodes 16 tcss, one of which (walk) is essential. here we show that *s. aureus* can be deprived of its complete sensorial tcs network and still survive under growth arrest conditions similarly to wild-type bacteria. under replicating conditions, however, the walk system is necessary and sufficient to maintain bacterial growth, indicating that sensing through tcss is mostly dispensable for living under constant environmental conditions.

characterization of *s. aureus* derivatives containing individual tcss reveals that each tcs appears to be autonomous and self-sufficient to sense and respond to specific environmental cues, although some level of cross-regulation between non-cognate sensor-response regulator pairs occurs in vivo. this organization, if confirmed in other bacterial species, may provide a general evolutionarily mechanism for flexible bacterial adaptation to life in new niches."

Fox, K., & Wong, R. O. L.. (2005). A comparison of experience-dependent plasticity in the visual and somatosensory systems. *Neuron*

Plain numerical DOI: 10.1016/j.neuron.2005.10.013

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"In the visual and somatosensory systems, maturation of neuronal circuits continues for days to weeks after sensory stimulation occurs. deprivation of sensory input at various stages of development can induce physiological, and often structural, changes that modify the circuitry of these sensory systems. recent studies also reveal a surprising degree of plasticity in the mature visual and somatosensory pathways. here, we compare and contrast the effects of sensory experience on the connectivity and function of these pathways and discuss what is known to date concerning the structural, physiological, and molecular mechanisms underlying their plasticity. copyright ©2005 by elsevier inc."

Cranin, A. N., & Sher, J.. (1979). Sensory deprivation. *Oral Surgery, Oral Medicine, Oral Pathology*

Plain numerical DOI: 10.1016/0030-4220(79)90120-8

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"The loss of vision, hearing, and speech, even on a temporary basis, may be responsible for strange, unpredictable, or bizarre behavior. the placement of obtundent surgical dressings may be responsible for the symptoms of sensory deprivation. © 1979."

McCurry, C. L., Shepherd, J. D., Tropea, D., Wang, K. H., Bear, M. F., & Sur, M.. (2010). Loss of Arc renders the visual cortex impervious to the effects of sensory experience or deprivation. Nature Neuroscience

Plain numerical DOI: 10.1038/nn.2508

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"A myriad of mechanisms have been suggested to account for the full richness of visual cortical plasticity. we found that visual cortex lacking arc is impervious to the effects of deprivation or experience. using intrinsic signal imaging and chronic visually evoked potential recordings, we found that arc(-/-) mice did not exhibit depression of deprived-eye responses or a shift in ocular dominance after brief monocular deprivation. extended deprivation also failed to elicit a shift in ocular dominance or open-eye potentiation. moreover, arc(-/-) mice lacked stimulus-selective response potentiation. although arc(-/-) mice exhibited normal visual acuity, baseline ocular dominance was abnormal and resembled that observed after dark-rearing. these data suggest that arc is required for the experience-dependent processes that normally establish and modify synaptic connections in visual cortex."

Category

1. Psychopolitics
2. Social psychology
3. Sociology

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