Cortical Plasticity

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The phenomenon of cortical plasticity demonstrates that the organization of the brain is not fixed, but rather, changes in the course of development, learning and in response to injury. The present document focuses primarily on cortical plasticity in cats that have lost a sensory modality.

Cortical plasticity in development. The organization of the brain changes throughout development. In cats and monkeys (and in humans, as well), adult primary motor cortex (a.k.a. "striate" cortex) is separated into ocular dominance columns. Striate neurons representing visual input from the left and right eyes are organized in alternating columns of brain tissue, such that a column representing the right eye is located between columns representing the left eye.

In juveniles, this neat organization isn't readily observed. Instead, a given region of striate haphazardly represents the left and right eyes with no clear organization. This suggests that mammals (like cats, monkeys and humans) aren't born with elegant ocular dominance columns in striate cortex. Instead, these columns must develop over time. This is an example of cortical plasticity.

Monocular deprivation studies, in which only one eye is permitted to remain open during development, show that this particular type of cortical plasticity is sensitive to the sensory experience of the developing animal. In these studies, the ocular dominance columns are changed by monocular deprivation. In particular, the columns pertaining to the deprived eye disappear. Interestingly, the neurons that normally would have been devoted to the deprived eye are recruited for the functioning eye. The result of monocular deprivation is that the alternating left and right ocular dominance columns give way to a collection of columns that represent only one eye, namely, the functioning eye.

Cortical plasticity following learning. The above example demonstrates that the brain can change in response to variations in sensory experience. It also shows that poverty of sensory input results in reallocation of the corresponding neural tissue. These general principles are also illustrated in the case of monkeys learning to perform a tactile discrimination task. When monkeys practice using the third digit of one hand to discriminate between textures, the representation of the digit in somatosensory cortex grows. This growth, however, is at the expense of cortical representation of the remaining digits, which shrinks accordingly and accommodates the increased amount of neural tissue devoted to the third digit.

Surgical removal of the third digit has a complementary effect. In this case, the representation of the lost digit in somatosensory cortex disappears and is subsequently occupied by cortical representations of the remaining digits. This mirrors the effects of monocular deprivation.

Cortical plasticity following loss of sensation. The example of the monkey with a missing finger certainly demonstrates changes in the brain that follow injury, but there are more colourful examples to consider. In cases where sensory input is chronically absent, the redistribution of neural resources produces interesting results.

Bavelier et al. (2000) showed that deaf individuals more easily detect changes in visual stimuli appearing in the periphery than inviduals with normal hearing. These results are reminiscent of anecdotal reports that people with deficiencies in one sensory modality are more sensitive when relying on their intact senses. The same appears to be true when comparing deaf cats with hearing cats. Congenitally deaf cats have visual abilities that are superior than hearing cats. The deaf cats outperform their non-deaf counterparts in tasks that entail detection of movement and detection of visual stimuli in the periphery.

Recordings of neural activity in the FAES of deaf cats suggests that the neural resources have been reallocated. The FAES, which normally responds to auditory input, responds to visual input in deaf cats. Primary auditory cortex (A1), however, is unresponsive to visual input and so reallocation of neural resources appears to take place in higher order auditory brain areas.

Taken together, the evidence suggests that the brains of congenitally blind cats are organized in a way that does not quite resemble the brains of regular cats. In the deaf cat, the anterior ectosylvian visual

area (AEV), a cortical region normally devoted to vision, is unusually large and occupies the auditory field of the anterior ectosylvian sulcus (FAES), which is normally devoted to audition. This change in cortical organization echoes that seen in the somatosensory cortices of monkeys missing a finger. Tracing studies in these cats show that projections from the cortex to the thalamus (but not vice versa) also change, with new projections to non-geniculate, previously auditory areas.

Cortical plasticity in congenitally deaf cats is not without behavioural consequences. Indeed, in deaf cats only, deactivation of the FAES results in a detriment in performance during tasks that involve detection of visual stimuli. This result is consistent with the idea that congenital deafness results in a reallocation of neural resources, whereby auditory areas are recruited for visual processing. These effects can be somewhat reversed by restoring function to the lost sensory modality (audition), although the time during which sensation is restored is a critical factor.

Major Themes/Questions

- What are the commonalities between different cases of cortical plasticity, such as those observed in monocular deprivation, surgical removal of a digit, learning, and congenital deafness in cats?
- What are the general changes associated with "cortical plasticity"?
- Does cortical plasticity confer an adaptive advantage? If so, what evidence can be cited to support this conclusion?