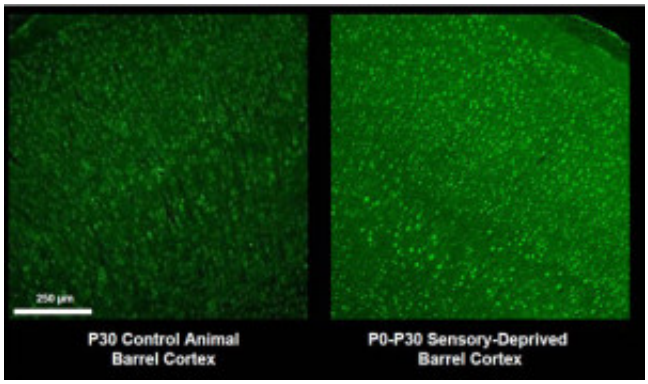
Sensory deprivation early in life has lasting impacts

What happens to a brain area when it is underutilized when a person or animal is growing up? Does this kind of deprivation of experiences lead to negative effects for brain development? For a long time, the research in this area has been rather unclear, and has been an area of debate in the scientific field.

Scientists from the Graduate Center and Queens College, City University of New York may have contributed to answering this question. In a research published in May of 2015, the researchers found an enzyme, termed tissue plasminogen activator (tPA), produced by neurons (nerve cells) and supporting cells (glia) in the brain, is regulated by sensory experience.



Staining for tPA (green) in mice brains reveal that tPA levels increase (right hand panel) following one month of sensory deprivation (whisker trimming every other day from birth) compared to animals who have intact facial whiskers

tPA may sound very familiar to physicians and patients alike, who has strong affinity to various types of strokes. Yes, it is the same chemical that emergency room doctors administer to stroke patients as an acute treatment, due to its powerful effects in dissolving blood clots. So what does tPA, a drug that is used to treat strokes, have to do with brain maturation? It turns out that for a long time, endogenous (meaning produced inside of the body) tPA was thought to be only produced by the endothelial cells, the cells that line the blood vessels, and the release of tPA into the blood stream would work to prevent the formation of blood clots. Its roles outside of ensuring the smooth flowing of blood has been unclear until recently. Neuroscience researches started to show that tPA is also produced by neurons, but what its functions are in the brain has not been clear. Until now.

When we are young, the brain is constantly shaping and remodeling itself, and making new synaptic connections based on significant life experiences. This process is known as neuroplasticity. The brain loses some of its abilities to this youthful plasticity as we grow up, and we

become less capable of making new connections in our brain. It turns out the same process occur in animals, thus it is much harder to make old dogs learning new tricks. Interestingly, tPA levels decrease as we age, another possibility to why our mental sharpness declines with age. But that is not all it does, it turns out that tPA also plays a very important role in this experience-dependent neuroplasticity. When the researchers at the Graduate Center and Queens College trimmed off the whiskers on mice, a sensory organ that these rodents relies heavily on in order to navigate and interact with their environment, the tPA levels in the corresponding areas in the brain went through the roof, and the high tPA levels persisted well into adulthood, even after the whiskers had regrown. This may be a way that Mother Nature tries to preserve the permissibility of the cellular environments, in case the sensations do return later in the brain.

The findings in this study provides hints in how we may extend the period of a critical developmental phase. It also suggested the importance of tPA in regulating the neuroplasticity in general.

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Publication

Experience-dependent regulation of tissue-type plasminogen activator in the mouse barrel cortex.
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