

MIT Science News In Review

[Brain and Cognitive Sciences]

Children Think Like Scientists

MIT assistant professor Laura E. Schulz and colleague Jessica Sommerville of the University of Washington have recently discovered a higher level of sophistication in young children's thinking than previously believed. The researchers' findings, delineated in their paper "God Does Not Play Dice: Causal Determination and Preschoolers' Causal Interferences," show how preschoolers possess a reasoning process similar to that of scientists. Children, like scientists, are more likely to attribute an event to an unobserved cause than to random chance.

Schulz and Sommerville tested this theory on 144 children by teaching them how to flip a switch and subsequently light up a toy with a metal ring. They also showed the preschoolers how removing the ring would prevent the toy from lighting up. The experimenters then instructed the children to prevent the toy from lighting up. Those children whose toys consistently lit up when the switch was flipped, removed the ring as expected. However, subjects whose toys lit up only half of those times when the switch was flipped realized that some other, unobserved cause must be responsible for the toy's ability to illuminate and darken itself. These children responded by picking up an additional object that had been in the experimenter's hand and using that to try to prevent the toy from lighting up. Like scientists, the children were hesitant to believe that any event, in this case the illumination of the toy, could occur simply at random. —G. Denman

Source: "Scientists show that children think like scientists"
<http://web.mit.edu/newsoffice/2006/children.html>

Visual Role for Growth Factor

At MIT's Picower Institute for Learning and Memory, a team of scientists led by Mriganka Sur, the Sherman Fairchild Professor of Neuroscience and Department Head, postdoctoral fellows Daniela Tropea and Gabriel Kreiman, research affiliates Alvin Luckman and Sayan Mukherjee, postdoctoral associate Hongbo Yu and graduate student Sam Horng, has recently identified an insulin-like growth factor that prevents the usual effects of visual deprivation in the brain.

The molecule in question, insulin-derived growth factor-1 (IGF1) may one day provide a means for scientists to manipulate connections among neurons in the developing brain and possibly even in the adult brain. Researchers may use it to repair brain cell connections that have been lost or damaged through injury or diseases, such as Alzheimer's, or to treat conditions like autism.

IGF1 prevents the physiological effects that occur when visual input to one eye is shut down. In earlier research conducted in the 1960s, Harvard scientists David Hubel and Torsten Weisel, who later won the Nobel Prize, found that depriving neurons of stimulation from one eye led to a reassignment of neurons to the non-deprived eye.

Sur states that with the addition of IGF1, "there is no loss of deprived eye connections or growth of non-deprived eye connections. We hope that this growth factor will turn out to be equally important for other kinds of developmental and even adult plasticity."

—D. Zhang

Source: "Brain researchers see visual role for growth factor"
<http://web.mit.edu/newsoffice/2006/nature-nurture.html>

Brain Map Found in Rat Whiskers

Neuroscientists at MIT's McGovern Institute for Brain Research recently confirmed the existence of micro-maps of the brains of rats present in the animals' whiskers. Maps of mammals' brains had previously been known to exist only in the visual cortexes of the animals. However, due to rodents' reliance on their whiskers rather than on visual input to guide them, the presence of sensory maps in rats' whisker systems is not wholly unexpected.

The MIT research team, which includes principal investigator Christopher Moore and graduate student Mark Andermann, has found that rat whiskers form topographic maps of the brain, with each whisker corresponding to 4,000 closely packed neurons. These whiskers act as both visual and auditory maps, providing the brain with all necessary information on nearby objects and obstacles. The research team's discovery of brain maps in rat whiskers will render rodents even more useful in the study of sensory processing in mammals and the effects of mental illness, strokes, and epilepsy on this processing.

—G. Denman

Source: "Rat whiskers lead to brain map"
<http://web.mit.edu/newsoffice/2005/deuterium.html>



A rat's whiskers help the animal create a topographic map of its surroundings in its brain, MIT researchers report.

<http://www.abc.net.au/science/news/img/health/mouse24804.jpg>

Epilepsy Detection

MIT researchers and their collaborators at the Beth Israel Deaconess Medical Center are taking steps to improve the current methods of epilepsy treatment. Epilepsy is a neurological condition that causes frequent seizures and affects over 50 million people worldwide. Seizures are triggered by sudden surges in electrical activity in the brain and have traditionally been treated with anticonvulsant medications. However, about a third of patients do not respond to these treatments. An alternative treatment utilizes a vagus nerve stimulator (VNS), an implanted defibrillator that delivers mild electrical impulses to the left vagus nerve at regular intervals. Because the vagus nerve controls heart rate and airway dilation, stimulating it at the proper time can prevent a seizure from occurring.

John Gutttag, MIT professor of Electrical Engineering and Computer Science, and graduate student Ali Shoeb have improved the VNS by developing software that interprets brain activity patterns and predicts when a seizure will occur. Patients, using this device, will wear an electrode embedded

cap that is connected to a customized detector. This system will be able to sense an oncoming seizure, and activate the VNS to prevent it.

In addition to treating epilepsy, Guttag hopes that the same device can be used to shed light on other debilitating neurological disorders, such as depression, schizophrenia, and attention deficit disorder.

—M. Anahtar

Source: "Epilepsy breakthrough on horizon"
<http://web.mit.edu/newsoffice/2006/epilepsy.html>

New Alzheimer's Treatment

Research at MIT has led to the development of a cocktail of dietary supplements, now in human clinical trials, which could delay the cognitive decline seen in Alzheimer's disease that currently afflicts an estimated 4 to 5 million Americans. Currently in human clinical trials, it holds promise for treating Alzheimer's disease.

Richard Wurtman, the Cecil H. Green Distinguished Professor of Neuropharmacology, was the leader of this research study. The study appears in the May 9th issue of *Brain Research* and has been presented by Wurtman at the International Academy of Nutrition and Aging 2006 Symposium on Nutrition and Alzheimer's Disease/Cognitive Decline.

The cocktail treatment consists of omega-3 fatty acids, uridine, and choline, which are needed by brain neurons to make phospholipids, the primary component of cell membranes. Wurtman's research shows an observed increase in the number of membranes, which form brain cell synapses, after adding these supplements to the diets of gerbils. Damage in brain synapses is believed to be the cause of dementia in Alzheimer patients. If similar results can be duplicated in human trials, the new treatment could perhaps offer not only a cure, but also a treatment for Alzheimer's.

Instead of the traditional tactic of targeting the amyloid plaques and tangles that develop in the brains of Alzheimer's patients, the new research focuses on brain synapses where neurotransmitters such as dopamine, acetylcholine, serotonin and glutamate carry messages from presynaptic neurons to receptors in postsynaptic neuron membranes. Omega-3 fatty acids, uridine and choline work to increase synaptic membrane proteins so that damage in brain synapses caused by Alzheimer's disease can be repaired. Further studies by researchers at MIT and scientists at Cambridge University have showed that either uridine or omega-3 fatty acids can also promote the growth of neurites, which are small outgrowths of neuronal cell membranes, which further supports the hypothesis that omega-3 fatty acids increase synaptic membrane formation.

Alzheimer's patients in clinical trials at multiple medical centers are now being given a drink that contains the compounds under study or a taste-matched placebo. "If it works as well on the brains of people with Alzheimer's disease as it does in laboratory animals, I think there will be a lot of interest," Wurtman said.

—D. Zhang

Source: "MIT research offers new hope for Alzheimer's patients"
<http://web.mit.edu/newsoffice/2006/alzheimers.html>

New Lazy Eye Theory

Researchers at MIT's Picower Institute for Learning and Memory recently reformulated a new explanation for the development of amblyopia. Commonly referred to as lazy eye, amblyopia is a developmental disorder in which a structurally normal eye gives rise to poor or blurry vision. Previously, physicians believed that lazy eye developed as a result of a lack of visual stimuli, thereby weakening the responsiveness of the eye's cortical neurons.



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However, the MIT research team has determined that poor image quality can also prompt development of the disease. Thus lazy eye is just as likely to develop through the receipt of blurry images as from outright deprivation of visual stimuli.

Study co-author, Professor Mark Bear reports that his team reached their conclusions by studying the traditional mode of treatment for lazy eye. This method requires each patient to wear an eye patch over the healthy eye in an effort to increase the number of images projected onto the weak eye and subsequently strengthen the responsiveness of that eye's cortical neurons. Bear and his colleagues concluded that the healthy eye was more likely to lose function when blurry images were projected through the patch for an extended period of time than when no images were projected at all.

—G. Denman

Source: "Research gives lazy eye theory a workout"
<http://web.mit.edu/newsoffice/2006/lazyeye.html>

New Compound Treats Huntington's Disease

Researchers at MIT and Harvard Medical School have identified a compound that interferes with the pathogenic effects of Huntington's, possibly resulting in the development of a new treatment for this disease.

A compound known as B2, developed by Ruth Bodner and others in the laboratories of MIT Professor of Biology David Housman, Harvard Medical School Assistant Professor Aleksey Kazantsev and Harvard Medical School Professor Bradley Human could possibly lead to a drug that will stop the course of Huntington's disease.

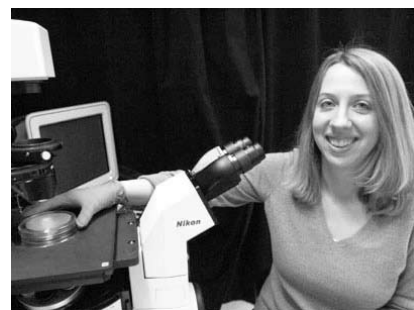
Huntington's disease is caused by misfolded proteins that aggregate and form large clump-like "inclusions" in the brain. It is characterized by

degeneration in the cortex and the striatum, an area associated with motor and learning functions. These misfolded proteins ultimately disrupt proteasomes necessary for disposing of misfolded or unneeded cellular proteins. Without a functional proteasome, cellular proteins accumulate, poisoning brain cells and impairing patients' motor and cognitive function.

Most Huntington's treatments have focused on compounds that prevent or reverse the aggregation of inclusions. Contrastingly, Bodner believes that the largest aggregation of inclusions may not necessarily be harmful and could even be protective. This theory led MIT and Harvard scientists to look for compounds that actually promote the formation of large inclusions. The highest concentration of protein inclusions occurred when researchers applied B2 to cells cultivated in the laboratory. B2 has a strong protective effect against proteasome disruption, thus blocking one of the toxic effects of the Huntington protein. Furthermore, the B2 compound has promoted large inclusions that showed a protective effect in a cellular model of Parkinson's disease. Researchers are now working on finding a more potent version of the compound, which can be tested in mice.

—D. Zhang

Source: "Research holds promise for Huntington's treatment"
<http://web.mit.edu/newsoffice/2006/huntington.html>



Ruth Bodner has discovered a compound that could lead to the development of a new treatment for Huntington's disease, a fatal neurodegenerative disorder.



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