

Study suggests caution on a new anti-obesity drug in children

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A new class of anti-obesity drugs that suppresses appetite by blocking cannabinoid receptors in the brain could also suppress the adaptive rewiring of the brain necessary for neural development in children, studies with mice have indicated. One such drug, rimonabant (trade name Acomplia) has been developed by Sanofi-Aventis and is awaiting approval for use in the U.S., and other pharmaceutical companies are developing similar drugs.

Mark Bear and colleagues published their findings in the May 8, 2008, issue of the journal *Neuron*, published by Cell Press.

The principal aim of the researchers' experiments was to gain insight into regulation of the process called "experience-dependent cortical plasticity" in the brain. Such plasticity is the adaptive rewiring of the brain caused by experience that is central to neural development in children and young animals.

For their experimental model, the researchers used plasticity in the visual cortex of the mouse. The visual cortex is the brain region that processes visual signals from the eye, adapting to experience. To study visual cortex plasticity, the researchers used the long-known phenomenon that closing an eye in a young animal causes that eye to lose visual responsiveness—known as a shift in "ocular dominance (OD)—as the visual cortex rapidly adapts due to its plasticity. Specifically, the researchers wanted to understand the regulation of plasticity in two layers, or lamina, of the visual cortex called 2/3 and 4. Also, they knew

that activity of the cannabinoid receptor plays a role in plasticity by regulating the signaling connections among neurons.

In their experiments, the researchers closed one eye of an animal and measured the effect on plasticity using recording electrodes implanted in the layers of the visual cortex.

It had been previously believed that plasticity in layer 2/3 was required for plasticity in layer 4. However, the researchers found that when they used a drug called AM 251 to block the cannabinoid receptors in the animals' brains, plasticity in layer 2/3 was suppressed, but plasticity in layer 4 was unaffected.

“These findings simplify the mechanistic description of plasticity in layer 4, force a revision in the interpretation of previous studies in which laminar differences in OD plasticity mechanisms were unrecognized, and have important implications for the therapeutic use of cannabinoid receptor antagonists in humans,” concluded Bear and colleagues.

Citing the development of obesity drugs that block cannabinoid receptors, the researchers cautioned that “Our finding of a profound disruption of cortical plasticity in juvenile mice treated with AM 251 suggests caution is advised in the use of such compounds in children.”

Source: Cell Press

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