

Molecule found that inhibits recovery from stroke

July 27 2012, By Mark Wheeler

(Medical Xpress) -- Researchers at UCLA have identified a novel molecule in the brain that, after stroke, blocks the formation of new connections between neurons. As a result, it limits the brain's recovery. In a mouse model, the researchers showed that blocking this molecule—called ephrin-A5--induces axonal sprouting, that is, the growth of new connections between the brain's neurons, or cells, and as a result promotes functional recovery.

If duplicated in humans, the identification of this molecule could pave the way for a more rapid [recovery](#) from [stroke](#) and may allow a synergy with existing treatments, such as physical therapy.

Stroke is the leading cause of adult disability because of the [brain's](#) limited capacity for repair. An important process in recovery after stroke may be in the formation of new connections, termed axonal sprouting. The adult brain inhibits axonal sprouting and the formation of these connections. In previous work the researchers found, paradoxically, that the brain sends mixed signals after a stroke—activating molecules that both stimulate and inhibit axonal sprouting. In this present work, the researchers have identified the effect of one molecule that inhibits axonal sprouting and determined the new connections in the brain that are necessary to form for recovery.

The researchers also developed a new tissue bioengineering approach for delivering drugs to the brain after stroke. This approach uses a biopolymer hydrogel, or a gel of naturally occurring brain proteins, to

release neural repair [molecules](#) directly to the target region for recovery in stroke—the tissue adjacent to the center of the stroke.

Last, the paper also shows that the more behavioral activity after stroke, such as the amount an impaired limb is used, the more new connections are directly stimulated to form in the injured brain. This direct link between movement patterns, like those that occur in neurorehabilitation, and the formation of new brain connections, provides a biological mechanism for the effects of some forms of physical therapy after stroke.

The research appears online this week in the journal *PNAS*.

Provided by University of California, Los Angeles

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