Propranolol Shows Early Promise for PTSD

BY KATE JOHNSON

FROM THE ANNUAL MEETING OF THE INTERNATIONAL SOCIETY FOR TRAUMATIC STRESS STUDIES

MONTREAL – Treatment of posttraumatic stress disorder with the beta-blocker propranolol might interrupt memory reconsolidation by inhibiting protein synthesis in the brain, reported researchers at the International Society for Traumatic Stress Studies.

"It does not erase memories; this is a misnomer," clarified Alain Brunet, Ph.D., of the department of psychiatry at McGill University and a researcher at the Douglas Mental Health Institute, both in Montreal.

Dr. Brunet presented several studies conducted by his group that suggest this pharmacologic interruption of memory might dampen emotional response to the information, presenting a promising treatment opportunity for posttraumatic stress disorder (PTSD).

Current treatment for PTSD is centered on psychotherapy that focuses on exposure to the traumatic memory and learning new responses to it, Dr. Brunet said. But a recent analysis found that only about one-third of patients treated this way experience a lasting, clinically meaningful improvement, he said.

Propranolol treatment takes a different approach. It is based on the notion that memories, once they are consolidated, can be retrieved, and they exist in a labile state during which they are susceptible to modification until they are reconsolidated. During the labile window of opportunity, which is believed to be several hours, administration of propranolol can strip the memory of its emotional meaning, making it less stressful, he said.

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Major Finding: Propranolol administered at traumatic memory recall appears to block reconsolidation of the memory.

Data Source: A randomized, controlled trial of 19 patients and two open-label studies totaling 42 patients, by the same group, showed a reduction in memory-induced signs and symptoms of traumatic stress.

Disclosures: Dr. Brunet had no disclosures to report.

In a randomized, controlled trial involving 19 chronic PTSD patients with an average symptom of duration 10 years (J. Psychiatr. Res. 2008;42:503-6), Dr. Brunet and his colleagues asked the patients to recall their memory by writing a trauma script and outlining the details of their traumatic experience and the emotions they felt.

Nine patients were then given a two-dose regimen of fast-acting propranolol (40 mg) immediately after memory recall, followed by an extended-release propanolol dose (60 mg) 75 minutes later, and the other 10 patients received placebo. One week later, after reviewing their trauma script, patients' physiologic responses to the memories were compared, using heart rate, skin conductivity, and corrugator electromyography (EMG) measurements.

Dr. Brunet reported significant differences between the placebo and treatment groups on heart rate and skin conductivity tests but not on EMG. There was a trend toward decreased symptoms, measured on the self-report Impact of Event Scale-Revised (IES-R).

The field of memory reconsolidation blockade is young when it comes to human studies, but there is substantial animal research to support it, commented Dr. Charles Marmar, professor and chair in the department of psychiatry at New York University Medical Center, who chaired a panel discussion after the session.

"The notion of building a pipeline from basic science, to translational studies in humans, to new treatments is very, very important in psychiatry," he said in an interview. "This work is pioneering. We should have some patience about this and appreciate that this is a new paradigm in mental health research." ■