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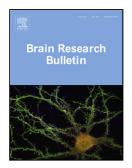
Title: Gastrin-Releasing Peptide attenuates fear memory reconsolidation

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Gastrin-Releasing Peptide attenuates fear memory reconsolidation

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Abstract

Background: Gastrin Releasing Peptide (GRP) may play a role in fear learning. The GRP Receptor is expressed in the basolateral amygdala and hippocampus, and central administration of GRP mediates fear learning. The effects of GRP on reconsolidation, however, have been minimally explored. Reconsolidation, the process by which formed memories are rendered labile following recall, provides a window of opportunity for pharmacological intervention. Although evidence suggests the window of opportunity to alter reactivated consolidation memory can be as long as 6 h, shorter intervals have not been extensively investigated.

Method: Male Sprague-Dawley rats received six 1.0 mA continuous footshocks. 24 h later, were re-exposed to the context (shock chamber). Immediately following memory retrieval rats received i.p. injection of GRP (10 nmol/kg), Flumazenil (1 mg/kg), GRP + Flumazenil (10 nmol/kg GRP with 1 mg/kg Flumazenil), or Vehicle. Other groups received GRP or Vehicle at 0, 10, 30, or 60 min post-reactivation. 24 h and 5 days later rats were assessed for fear expression upon re-exposure to the fearful stimulus.

Results: GRP significantly attenuated the reconsolidation of learned fear when administered immediately (but not 10 min or longer) following recall. Some of the variability in the impact of treatments aimed at disrupting fear memories may be governed, in part, by the time-frame of the reconsolidation window. Our results indicate that the effect of immediate administration persisted

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