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**ROLE OF EXCITATORY AND INHIBITORY
NEUROTRANSMISSION IN THE BASOLATERAL AMYGDALA OF
RATS IN REGULATING ANXIETY RESPONSES**

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Submitted to the faculty of the University Graduate School

in partial fulfillment of the requirements

for the degree

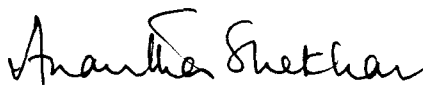
Doctor of Philosophy

in the Program in Medical Neurobiology,


Indiana University

August, 1997

Accepted by the Graduate Faculty, Indiana University, in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

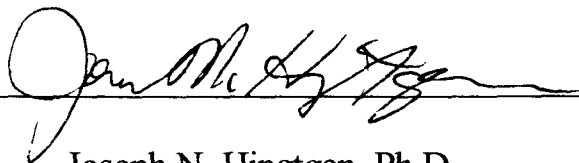


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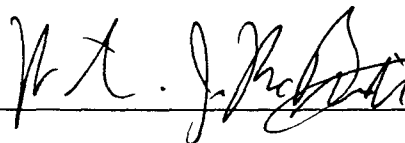


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ABSTRACT

Blocking GABA_A receptors in the anterior basolateral amygdala (BLA) with bicuculline methiodide (BMI) elicits increases in heart rate (HR), blood pressure (BP) and 'anxiety' in rats, as measured in the social interaction test. Glutamate receptors in the BLA are also reported to be involved in generating anxiety responses. Therefore, the purpose of this project was to: (1) determine if a balance exists between excitatory amino acid (EAA) excitation and GABAergic inhibition in the BLA that regulates the anxiety response; (2) show that repeated activation of the BLA (by decreasing GABA inhibition and thus increasing glutamate excitation) will result in a long term synaptic plasticity (priming) causing a facilitation of the anxiety response; and (3) reveal that the anxiety response generated by priming leads to a subtype of anxiety similar to human panic disorder. Male rats were fitted with catheters into their femoral artery, and vein if necessary, then implanted with microinjection cannulae bilaterally into the BLA. After recovery, the following experiments were conducted. First, BMI was administered with or without the presence of different EAA antagonists, which revealed that blockade of both the NMDA and non-NMDA receptors could significantly block the increases in HR, BP and

experimental anxiety induced by BMI. Next, EAA antagonists were injected alone into the BLA, and the results showed that only anxiety was significantly decreased as compared to baseline, with no effect on either HR or BP. EAA antagonists were then administered either during or following the priming of anxiety in the BLA. It was found that only the NMDA antagonist AP5 could completely reverse the increases in HR, BP and experimental anxiety elicited by priming. Finally, a group of primed and sham-primed animals were infused with sodium lactate, an agent known to induce panic attacks in individuals with panic disorder. The results showed that primed and not sham-primed animals became sensitive to lactate infusions, which elicited significant increases in HR, BP and experimental anxiety. These results suggest that there exists a balance between EAA excitation and GABAergic inhibition within the BLA which regulates anxiety. Furthermore, it appears that disturbing this balance leads to a priming of anxiety which is similar to that of human panic disorder.

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