

Letter to the Editor

Antidepressants and Emotional Processing

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Sir

Dr Harmer and colleagues reported that acute administration of the selective serotonin reuptake inhibitor (SSRI) citalopram (10 mg i.v.) facilitated recognition of fear and happy facial expressions as evidenced by greater accuracy and reduced response times in the citalopram treated group relative to the placebo group (Harmer *et al*, 2002a). The authors also report their findings of a second study in which they found reduced recognition of negative emotions such as fear and disgust with repeated administration of citalopram (Harmer *et al*, 2002b). Both findings provide important neuropsychological evidence of a possible mechanism of action of antidepressant drugs. The findings are consistent with our own study that examined the neurophysiological mechanisms underlying antidepressant effects on emotional processing. Our findings suggest that acute (oral) administration of citalopram inhibited the electrophysiological responses to unpleasant or negative visual stimuli (ie steady-state visual evoked potential latency reductions) (Kemp *et al*, 2003), while enhancing responses to pleasant or positive stimuli (Kemp *et al*, submitted). Taken together, these studies suggest that antidepressant-induced reductions in clinical symptoms of depression may in part be related to neurophysiological and neuropsychological changes to the processing of emotional stimuli, such that processing of positive emotions is enhanced, while processing of negative emotions is suppressed.

An interesting observation by the authors was that the processing of emotion was modulated independent of changes in subjective mood. Our findings similarly showed that neurophysiological processing of affective stimuli (which may occur earlier) was modulated independent of changes in subjective mood. It is likely that processing of emotion is more sensitive to pharmacological manipulation and thus modulated with acute treatments while mood changes require more chronic changes to the serotonergic system.

An important consideration for future research is how individual rather than a group of emotions (positive or

negative) respond to serotonergic or other neurochemical manipulation. Contrary to the author's hypothesis (ie reduced recognition of 'negative emotions') an enhancement of recognition of fear (a 'negative emotion') was observed following serotonergic augmentation. A possible reason for this observed finding could be that while basic emotions such as sadness, fear, disgust and anger can be classified as negative or unpleasant emotions, they all have different neuroanatomical and neurophysiological substrates (Phan *et al*, 2002), and thus may be differentially regulated by serotonin or other neurotransmitter systems. Hence it is possible that some basic emotions within the classification of 'negative emotions' may be selectively modulated by antidepressants while others are enhanced or unaffected. In addition, it is possible that differences may exist between acute vs chronic neurochemical manipulations on various basic emotions as indicated by the authors.

While the study by Harmer and colleagues, and our own observations, provide neuropsychological and neurophysiological evidence for possible mechanisms related to antidepressant efficacy, further studies of different classes of antidepressants and longer treatment durations are needed to confirm original observations.

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