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Erratum

Diminished GABA_A Receptor Binding Capacity and a DNA Base Substitution in a Patient with Treatment-Resistant Depression and Anxiety

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Correction to: *Neuropsychopharmacology* (2004) **29**, 347–350. doi:10.1038/sj.npp.1300353

The abstract of this article was reproduced with typesetting errors. The correct version of the abstract is presented below.

In this report, we describe the case of a Caucasian male patient, aged 42, suffering from severe treatment-resistant generalized anxiety disorder with panic attacks and from severe major depression, for which he was treated with a course of electroconvulsive therapy. During electroconvulsive treatment, anesthesia was difficult to induce with etomidate and, once, propofol. Bispectral indices recordings (assessing the depth of anesthesia) revealed a much shorter duration of loss of responsiveness compared to a control patient receiving also a course of electroconvulsive therapy. Since GABA receptor-mediated regulation of cortical excitability is important with respect to general anesthesia,

we investigated the density of GABAA receptors with ¹²³Iiomazenil SPECT and found a clearly diminished binding of the radiotracer in the right frontal and orbitotemporal regions compared to the recordings in a 38-year-old healthy male control. Genetic analysis of exons 7 and 8 of the GABRB1-3 genes coding for the β 1-3 subunits of the GABA_A receptors revealed a silent G to A substitution in the third position of amino acid 257 of the β 1 subunit. To our knowledge, this is the first report of a link between insensitivity to anesthetic agents and altered GABAA receptor function in a clinical case. Whereas reduced GABA_A receptor-binding capacity has been investigated in anxiety disorders, this has not been the case in depressive disorders. This case illustrates how clinical observations in psychiatry can prompt investigation by modern techniques and potentially link clinics and basic sciences. No conclusions can, however, be made about the causal links in this single case.