Cortical Excitability and Post-Traumatic Stress Disorder

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Dear Editor,

We would like to add a contribution to the manuscript titled "Cortical Excitability and Agressive Behavior in Post-Traumatic Stress Disorder" that was published in the 52nd edition of your journal (1). To summarize this study, it was found the mean motor excitability threshold was lower in post-traumatic stress disorder (PTSD) patients than in control group patients; furthermore, a negative correlation was detected between the motor excitability threshold and the aggression scale and between aggression scores and the measurements of the cortical silent period that was presumed to be the findings of inhibition of the prefrontal cortex (1).

The activities of the amygdala and insula freed from the inhibition of the medial prefrontal cortex are mentioned in the biological hypothesis on PTSD that is accepted today (2). The disruption of anterior cingulate and orbitofrontal cortex functions in PTSD are consistent with this hypothesis (3,4,5).

Although increased amygdala activity and decreased frontal cortex inhibition are mentioned in the neurobiological etiology of PTSD, these findings are not applicable to all PTSD patients. For instance, increased frontal activity and the corresponding decreased amygdala excitability have been reported in PTSD patients dominated by dissociative symptoms such as depersonalization and derealization (6).

One of the most important changes in the The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition DSM-5 for PTSD was dissociative subtype identification (7). Following this change, studies related to the biological basis of the distinction between the dissociative and non-dissociative subtypes of PTSD have begun to be conducted (6). Neurobiological differences in the dissociative subtype of PTSD than rather than in the normal subtype may be seen.

More functional imaging studies and electrophysiological studies are needed to test whether there are neurobiological differences thought to exist between these subtypes. It should be considered that not making a distinction between the above-mentioned subtypes in studies on PTSD patients may impair the homogenization of the groups and affect biological data. In light of this information, making a distinction between dissociative and non-dissociative subtypes while determining patient groups in biological studies related to PTSD will enable us to more accurately interpret the obtained biological data.

Consequently, we believe that taking this issue into consideration in studies related to PTSD while designing studies will contribute to our understanding of the neurobiological etiology of PTSD.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - A.B., E.A., S.A.; Design A.B., E.A., S.A.; Supervision A.B., E.A., S.A.; Resource - A.B., E.A., S.A.; Materials - A.B., E.A., S.A.; Data Collection and/or Processing - A.B., E.A., S.A.; Analysis and/or Interpretation - A.B., E.A., S.A.; Literature Search - A.B., E.A., S.A.; Writing - A.B., E.A., S.A.; Critical Reviews - A.B.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

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Received: 26.08.2015 **Accepted:** 29.09.2015 **Available Online Date:** 19.01.2017

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