Atagün and Tunç, Bipolar Disord 2015, 1:1
DOI: 10.4172/2472-1077.1000e101

Bipolar Disorder: Open Access

Editorial Open Access

The Importance of Auditory Cortex Abnormalities in Type I Bipolar Disorder

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Functional and structural abnormalities of auditory cortices are associated with the neuropathology of schizophrenia [1-3]. Superior temporal lobe (STL) host primary and secondary auditory cortices and STLs have been associated with auditory hallucinations [2,4] and thought disorder [1] in schizophrenia. On the contrary, there are no significant differences in volumetric measurements of the STL regions in euthymic patients with bipolar disorder [5]. However, in the absence of auditory hallucinations or thought disorder in euthymia, functional studies with auditory paradigms have reported significant abnormalities in bipolar disorder [6-8]. In a recent *in vivo* neurochemical investigation with magnetic resonance spectroscopy study, we reported metabolic abnormalities in the left hemispheric STL in euthymic patients with bipolar disorder [9]. These findings are suggesting a specific neuropathology for bipolar disorder, which is independent of the clinical course of the disorder. STLs are among the most prominent brain regions that are explicitly influenced by the certain neuropathological processes.

Such state of the art linguistic functions are unique to human in nature and the involved brain regions are highly sophisticated in comparison to other species. Development of auditory cortices is long and maturation of the auditory network prolongs until adolescence [10]. For example, myelin sheath in the thalamocortical projections to auditory cortices begins to improve at the first year of life and continues until the age of four. Synthesis of mature neurofilaments that stand in the axonal skeleton prolongs until the age of ten. Consistently, prolonged developmental processes neurophysiological maturation follows the abovementioned developments. Particularly, in event-related potentials P1 and N1 are thought to be related with the primary auditory cortices and P1, N1 and P2 appear around the ages of fifteen [11]. Because of this slow and delicate developmental trajectory, auditory networks are vulnerable to neuropathological processes more than rapidly developing networks.

While the elicitation of several disturbances are attributable to post-onset developmental lag in bipolar disorder [12], large cohorts have also showed cognitive dysfunction and decrease of scholastic performance [13] and visuospatial dysfunction [14] that emerge before the onset of bipolar disorder. Accordingly, the current literature is suggestive of a slightly disruptive pathological process that interferes in auditory networks becomes active before the onset of the disorder and remains active during euthymia. Taken together auditory networks and STLs are among the most susceptible brain regions for disruptive neuropathological processes in bipolar disorder as well as schizophrenia. Further clarification of the differences between bipolar disorder type I and type II, recurrent depression and first degree relatives would be informative about the neuropathology of mood disorders in future studies. Geographically, STLs are located very close to the brain surface or cranium and such short distance makes it easier to assess this region with neuroimaging modalities.

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Received September 07, 2015; **Accepted** September 09, 2015; **Published** September 16, 2015

Citation: Atagün Mİ, Tunç S (2015) The Importance of Auditory Cortex Abnormalities in Type I Bipolar Disorder. Bipolar Disord 1: e101. doi:10.4172/2472-1077.1000e101