T113. Mindfulness-Based Cognitive Therapy Modulates Functional Brain Activation During Affective Distraction in Treatment-Resistant Depression: A Randomized Controlled Study

To see this abstract, please see Oral Abstract #O17.

T114. Behavioral Changes and Glial Staining in Mice Subjected to the Chronic Social Defeat Stress Followed by Electroconvulsive Stimulation

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Background: Chronic social defeat stress (cSDS) is a means of modeling depression- and anxiety-like phenotypes in mice. There has been little study of the effects of electroconvulsive stimulation (ECS) on mouse behavior after chronic social defeat.
Methods: We applied cSDS to C57BL/6J mice then assayed their behavior with social interaction test, open field test, sucrose preference test, and forced swim test to evaluate the phenotype. We then exposed defeated and control mice to 10 days of either active (isoflurane 2%, current 50mA, 100 pulses/sec, 0.5msec pulse width, one second duration) or sham (same handling and electrode placement) ECS stimulation and evaluated post-ECS behaviors (pre-ECS tests plus elevated zero maze, acoustic hyperarousal, tail flick, hot plate and the novel object recognition test) for changes in their behavioral phenotype. We used immunofluorescence to investigate the distribution of glial markers (Iba1, GFAP and S100b) across mouse brain regions, including cortical (cingulate, prelimbic, infralimbic, insula), limbic (nucleus accumbens, amygdala, hippocampus) as well as corpus callosum.
Results: After cSDS, defeated mice displayed an anxiety-like phenotype ($p<0.05$ for open field test) with a milder depression-like phenotype ($p<0.05$ forced swim) compared to controls. We observed an ECS-effect on both control and defeated mice, although there was little behavioral differences between sham mice post treatment. Similarly, there was an ECS effect on the amount ($p<0.01$) and branching ($p<0.01$) of GFAP staining in the dorsal and ventral hippocampus as well as the amygdala ($p<0.05$).
Conclusions: These findings will drive further investigation into how these cellular changes regulate neurocircuitry changes in response to ECS.
Supported By: R01
Keywords: Electroconvulsive Therapy, Chronic Social Defeat Stress, Glial Cells

T115. Necroptosis Might be a Time Dependent Neuronal Cell Death Mechanism on Hippocampus After Chronic Restraint Stress

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Background: Chronic stress causes neuronal cell death on the hippocampus and shrinking of the hippocampus is associated with depression. The connection between inflammation-mediated necroptosis and neuronal damage has been suggested by studies demonstrating a protective effect of necroptosis inhibitor on the brain injury. Therefore, we aimed to understand the molecular background of cell death mechanisms in hippocampal tissue isolated from chronic restraint-stressed rats.
Methods: Male Wistar rats received chronic restraint stress (CRS) for 7 and 28 consecutive days ($n=7$ for each group and controls). Tail suspension test was used to determine the depressive-like behaviour, the plasma levels of cytokines (TNF-α, IL1-α, IL1-β, IFN-γ) were measured by Luminex. The expressions of proteins related necroptosis (RIP, RIP3, MLKL), apoptosis (PARP, Bax, Bcl-2) and GR, BDNF, nNOS were measured by Western Blotting.
Results: There was a significant decrease in the level of depressive-like behaviour at the 28-day CRS compared to 7-day CRS. There was an increase in the level of IL1-β ($p\leq0.001$) and TNFα ($p=0.04$) at the 7-day CRS than the 28-day CRS. Concordantly, in the expression of RIP3 ($p\leq0.05$) and GR ($p\leq0.05$) were found increased while in the expression of PARP, BDNF and nNOS were significantly decreased ($p\leq0.05$) at the 7-day CRS than 28-day CRS.
Conclusions: Our data suggested that the duration of chronic stress might differ the pathway of neuronal cell death and the early period may be under the control of inflammatory mechanisms. Targeted prevention of necroptosis may provide a novel therapeutic approach for the treatment of inflammatory diseases including depression.
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Keywords: Hippocampus, Necroptosis, Apoptosis, Chronic Restraint Stress Model, Depression

T116. Anti-Inflammatory Parp Inhibitor Demonstrates Antidepressant Activity in Animal Model of Treatment-Resistant Depression

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Background: Major depressive disorder is associated with elevated levels of DNA oxidation, DNA damage, and gene expression of DNA repair enzymes including poly (ADP-ribose) polymerase-1 (PARP1). Elevated PARP1 activity is directly linked to neuroinflammation and PARP inhibitors are anti-inflammatory and neuroprotective. We previously showed that PARP inhibitors produce antidepressant-like effects equivalent to fluoxetine in rodent models. Here, we examined whether the PARP inhibitor 3-aminobenzamide (3AB) is effective in a rat model of treatment-resistant depression.
Methods: Treatment-resistant depression was modeled with injections of lipopolysaccharide (LPS; 0.1 ug/kg/day) and daily chronic unpredictable stress (CUS) for 28 days. Anhedonia and