expression of physical and mental symptoms; and 3) the role of post-disaster social support, and secondary stressors, in mediating the disaster effects.

Our findings will highlight the specific needs for mental health care associated with long term post-disaster psychopathology among high risk populations and will underscore the importance of developing evidence based post-disaster care, including screening and treatment capacities for individuals exposed to trauma in general medical practices.

P223

System of immunity in posttraumatic stress disorders

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90 male inpatients – participants of local combat actions on Caucasus with PTSD, aged 31,41 ± 0,88 years.

Analysis of structure of secondary immune deficiency (SID) in acute stress of combat actions has shown absence of SID in 31,7%, 68,3% - risk group for SID. Leading clinical syndrome - infectious (47,6%). More seldom allergic (3,7%) and autoimmune (2,4%) syndromes. 13,0% - combination of infectious and allergic syndromes.

In laboratory SID is confirmed in 37,5%. Study of the process of apoptosis has revealed a reliable as compared with control increase of content of CD95+ lymphocytes ($p<0,001$) in this group. It is possibly conditioned by formation of persistent ID with predominant decrease of T-helpers/inductors, modifying apoptogenic signal and predominating the development of apoptosis during activation through receptor complex CD3+-TCR. In combatants as compared with control total number of phagocyting neutrophiles ($p<0,001$) and number of particles absorbed by one phagocyte ($p<0,001$) is decreased. Background activity of oxidant systems of neutrophiles compatible with indices of stimulated variant of HCT-test of healthy persons ($p<0,05$) is decreased. Humoral link of immunity is activated - increase of level of circulating immune complexes ($p<0,001$), increase of concentration of serum immunoglobulines of classes M ($p<0,01$), G ($p<0,001$) and A ($p<0,05$).

In the process of treatment, number of leukocytes, lymphocytes of HLA-DR+ phenotype, concentration of IgG, phagocytar activity of neutrophiles is restored to level of control. Number of lymphocytes of CD3+, CD4+- phenotypes remains decreased.

P224

Social anxiety disorder and temperament: Excitatory and inhibitory mechanisms on primary motor cortex in patients and healthy controls

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Neurofunctional imaging studies comparing subjects with SAD and NC, reported a consistent increases in amygdala, changes in the lateral paralimbic regions and occipital cortices.

A current hypothesis underlying pathophysiology of social anxiety involves the dopaminergic system: SAD Subjects sow a reduction in D2 striatal binding (Schneier et al., 2000; Tihonen 1997)We hypothesized that subjects with SAD may have an altered cortical excitability, given previous imaging results showing changes in cortical activity. We also aimed to verify if SAD patients show at TMS a pattern Parkinson-like.

In order to highlight if there was a correlation between the temperamental dimensions and the measured parameters in our sample, we also explored the temperament of patients and HCs.

Method: We recruited n=15 SAD subjects and n=11 NC. We have utilized TMS on Primary Motor Cortex (M1) in order to study neuronal excitability and cortical inhibitory mechanisms. These has been achieved by examining EMG recording Motor Evoked Potentials (MEP). We measured MEP, Motor threshold, Cortical Silent Period (CSP), paired pulse inhibition both in patients and healthy controls. Clinical assessment was conducted with the MINI interview, Liebowitz Social Phobia Scale, TPQ

Results: neurophysiological variables are not significantly different between groups. Patients with SAD showed a significantly higher Harm Avoidance and lower Novelty Seeking. There was a positive correlation between CSP and Novelty seeking and a negative correlation between LICI and Novelty Seeking among patients but not among HCs

P225

Distinct patterns of premorbid social functioning in first-episode psychosis: Relationship to initial presentation

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Objective: To explore different longitudinal patterns of social functioning before onset of psychotic illness and how they relate to clinical presentation, substance use and acute treatment response.

Methods: Inclusion criteria: Drug-naïve first-episode psychosis, 18-50 yo, criteria for Schizophrenia or Other Psychotic Disorders (DSM-IV), excluding Psychotic Disorder due to a General Medical Condition and Substance-Induced Psychotic Disorder.

Exclusion criteria: Mental Retardation, neurological disease, brain injury or drug dependence.

Measures: Premorbid Adjustment Scale (PAS), Scale for the Assessment of Positive Symptoms (SAPS), Scale for the Assessment of Negative Symptoms (SANS).

Statistical analysis: Ward cluster analyses were carried out to differentiate three longitudinal patterns of social premorbid adjustment from childhood to late adolescence: stable good (N=75), stable bad (N=44) and deteriorating (N=35). Chi-square and ANOVA tests were used.

Results: 154 subjects (64.5% male, mean age 26.81, SD=6.98) participated in the study.

At baseline the socially stable good group had more positive symptoms, SAPS 13.85 (3.99), than the stable bad group, SAPS 11.82 (3.93) (p=0.023).

At six weeks post-treatment the socially deteriorating group had more negative symptoms, SANS 8 (4.89), than the stable good, SANS 3.85 (4.11), and the stable bad, SANS 5.23 (5.45) (p=0.000).

The stable good group had the highest rates of substance use, followed by the deteriorating group.

Conclusions: A good premorbid social life was related to higher substance use and more positive symptoms at presentation. A social deteriorating pattern was associated with more negative symptoms

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