P.1.b.004 Neural basis of acquired psychomotor resistance deficiency syndrome against to stress induced by toxic levels of noise

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Purpose: Large pressure and low frequency impulse noise is an irresistible stress factor and it may induce various and serious neuropsychiatric diseases. Intense impulse noise (202 dB/2 h) may result in prominent neuronal injury in the hippocampus, amygdala and cerebral cortex [1]. Amygdala contains more neurons in aggressive animals and impulse noise causes aggressive and violent attacks via affecting the amygdala. Hyperresponsive amygdala plays a major role in violent attacks [2]. If insensitivities against to stress may be originated from the ineffectiveness of amygdala [3], amygdalohippocampal degeneration may result in an Acquired Psychomotor Resistance Deficiency Syndrome by described firstly in the presented study.

Method: We investigated experimentally if a neural basis of Acquired Psychomotor Resistance Deficiency Syndrome may be associated with amygdalohippocampal injury induced by impulse noise. Twenty male rats were exposed to 120 dB impulse noice at doses of 10×20 min/day for one month. All of them were followed up in their cages was containing of five rats. The animals adapted to impulse noise were accepted as APRDS group (GI, n = 6) and the others were named as stress resistant group (GII, n = 14). After one month, all animals were sacrified and amygdalohippocampal areas were examined histopathologically. Live and degenerated neurons were counted stereologically and results were analysed statistically.

Results: At the beginning, all animals expressed the fearful and bewildered reactions to the intense sounds. Timely, some animals showed aggressive and violent attacks to themselves or each others. Lately, some animals adapted in helplessness and indifference behaviours without reaction to noise (GI, n = 6). But the others showed psychomotor exhaustedness signs (GII, n = 14) and five of them died at the last week due to weight loss, intracerebral or subarachnoid hemorrhage and lung injury. Interestingly, degenerated neuron number in the amygdalohippocampal areas were more in the GI than in the GII ($p < 0.001$), and another regions of the brains were also nearly normal in GI. In contrary, neurodegenerative changes at the extra amygdalohippocampal regions and another organic damages were more prominent for the GII than those of the GI.

Conclusion: Impulse noise induced neurodegenerative changes at the amygdalohippocampal regions may result in Acquired Psychomotor Resistance Deficiency Syndrome. This syndrome may be considered as a result of planned activity of the irritated amygdalohippocampal neurons with either voluntary or obligatory apoptosis. The aim of apoptosis may be protect the subjects from the extreme complication of impulse noise. Because stress and defence informations are recorded by amygdalohippocampal neurons [3], their apoptotic death can result in destruction of stressor agents recalling and defence informations generating neuronal units. As a result, stress creating impulses can not be perceived and defence generating impulses can not be produced by the brain. Consequently, we concluded that Acquired Psychomotor Resistance Deficiency Syndrome may be created systematically by the brain to protect the subjects from the more hazardous effects of the stress.

References

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P.1.c Basic neuroscience – Neuropharmacology

P.1.c.001 The effects of two different types of chronic morphine pretreatment on anxiety and nociception in adolescence offspring

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As is well known treatment of female rats with morphine may have long-term effects on their offspring. The purpose of this study was to analyze the potential effects of two doses of pre-mating morphine-chloride exposure on the neurobehavioral changes in offspring.

Female Wistar rats weighing 200–250 g were treated during 7 days before mating with drug-naive males with 10 mg/kg of morphine-chloride daily (M1-group) or with the accelerated doses from 10 to 60 mg/kg at 12 hours intervals (M2-group). At the time of weaning pups were tested for anxiety (elevated plus-maze, postnatal day 31) and locomotor activity in the novel environment (postnatal day 40). At postnatal days 41–47 we studied acute and chronic effects of morphine using the "tail flick" test. For statistical analysis we used criteria for independent samples and ANOVA methods. A number of changes were found in the offspring of female rats, exposed to morphine. Measuring the pup's weights during the first weeks of postnatal development revealed that both M1 and M2 pups had a significant weight loss at age of 1–1.5 month. At postnatal day 40 the mean weight of M1 pups was 147.1 g (168.2 in controls, $p < 0.05$), the weight of M2 pups – 127.5 (144.5 in controls, $p < 0.01$).

Behavioral studies have shown that the risk assessment behavior in the elevated plus-maze in the M1-group was significantly reduced. The number of head-dippings in this group (1.6+1.5) was lower than in the control (2.9 ± 2.6, $p < 0.05$). We have also observed a tendency for decrease in the time spent on the open arms of the maze (23.6 ± 18.7 sec in M1; 30.9 ± 26.7 in control; $p = 0.13$). M1 pups had reduced vertical exploration in the plus-maze (4.1 ± 2.4 compare to 5.9 ± 3.3 in the control, $p < 0.05$).

M2-offsprings demonstrated similar changes in the elevated plus-maze. The time spent on the open arms was lower than in control (15.7 ± 12.3 sec in M2-group; 22.4 ± 16.6 sec in control group) and the risk assessment behavior was reduced ($p < 0.05$). Finally, both M1 and M2 pups have demonstrated 2–4 fold lower locomotor activity in the novel environment at age of 40 days ($p = 0.001$–0.05 compared to controls). These data allow to sug-