Prenatal priming with lipopolysaccharide enhances the vulnerability to the infections in the adulthood – the study in the neurodevelopmental model of schizophrenia

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Agnieszka Basta-Kaim, Katarzyna Chamera, Magdalena Szuster, Ewa Trojan, Natalia Bryniarska

Department of Experimental Neuroendocrinology, Maj Institute of Pharmacology, Polish Academy of Sciences, Smętna 12 Street, 31-343 Cracow, Poland

OBJECTIVES

The prenatal priming may lead to schizophrenia-related behavior and modulate the vulnerability to the immune-stimulation in the adult offspring rats. The risk of developing schizophrenia appears to be influenced by a combination of various factors (Fig. 1).

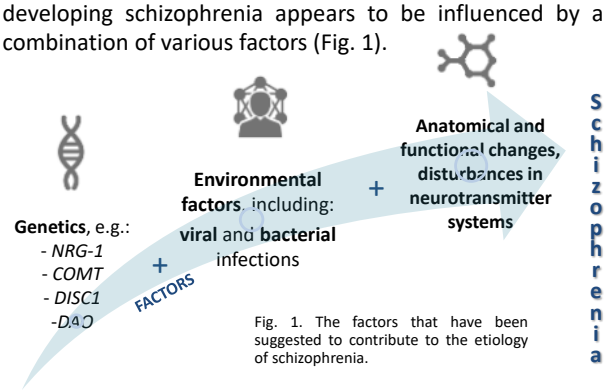

Fig. 1. The factors that have been suggested to contribute to the etiology of schizophrenia.

BACKGROUND AND AIMS

The present study was designed to examine if the maternal treatment with lipopolysaccharide (LPS) leads to behavioral and neuron-microglia proteins axis malfunction in the frontal cortex and the hippocampus of adult offspring rats. Moreover, we tested the impact of prenatal priming on the vulnerability to an additional stimulus in adulthood.

RESULTS

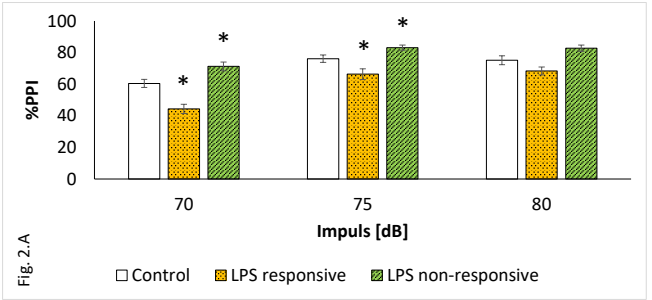

Fig. 2.A

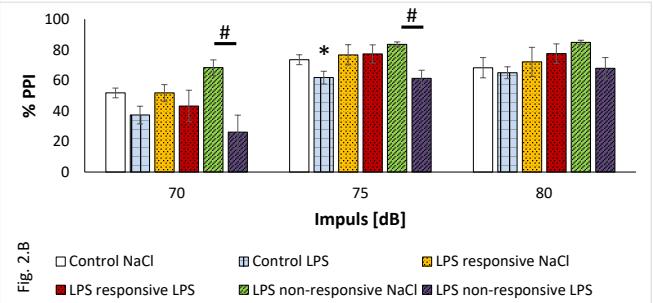

Fig. 2.B

Fig. 2. The effect of the prenatal administration of LPS (A) and a single injection of LPS (B) on the behavioral outcome of the offspring rats in the PPI test. The data are presented as the means ± SEMs, with n ≥ 9 for each group, contrast analysis. *p ≤ 0.05 compared with the Control group. # p ≤ 0.05 compared with the LPS non-responsive NaCl group.

MATERIALS AND METHODS

Pregnant Sprague-Dawley rats were injected with LPS from the 7th day of pregnancy every second day. 3-month-old male offspring animals were subjected to the behavioral examination. Afterwards, animals received a single injection of LPS. Next the behavioral tests were performed again and rats were sacrificed to dissect brain structures. The protein levels of CX3CL1, CX3CR1, CD200 and CD200R were measured using ELISA assays.

RESULTS

Fig. 3.

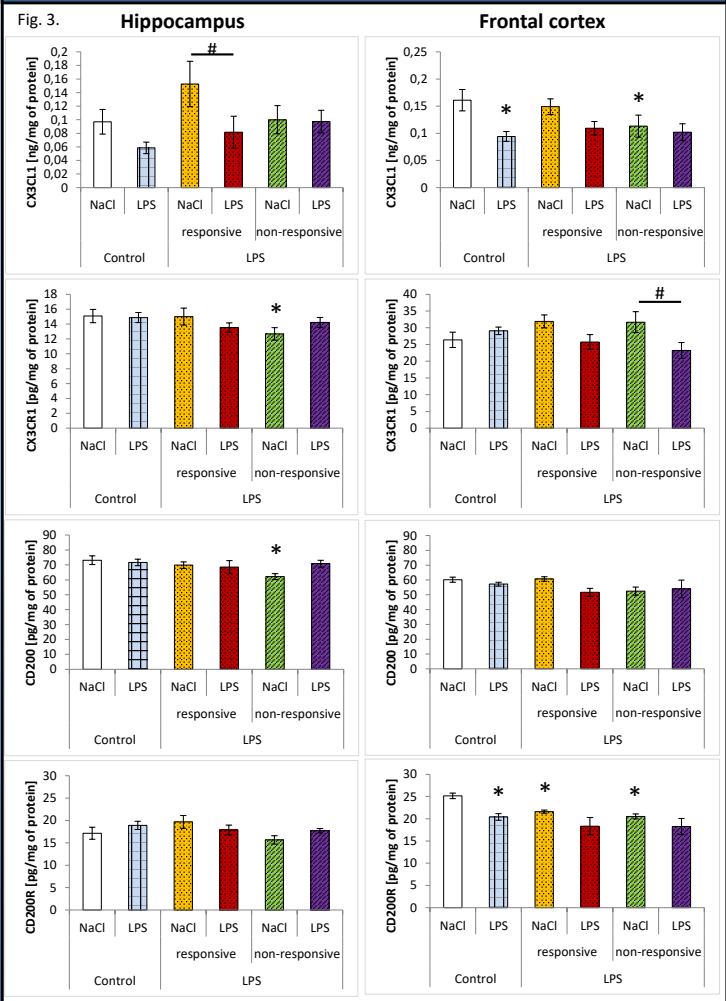

Fig. 3. The effect of the prenatal administration of LPS and a single injection of LPS on the protein levels of CX3CL1, CX3CR1, CD200, CD200R in the hippocampus and the frontal cortex of the offspring rats. The data are presented as the means ± SEMs, with n ≥ 4 for each group, contrast analysis. *p ≤ 0.05 compared with the Control group. # p ≤ 0.05 compared with the LPS non-responsive NaCl group.

CONCLUSIONS

Our results show that prenatal exposure to lipopolysaccharide causes not only behavioral schizophrenia-related change in offspring, but also sensitize them to immune-stimulation encountered in adulthood. Importantly, the observed behavioral alternations are followed by disturbances in the neuron-microglia protein systems.

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