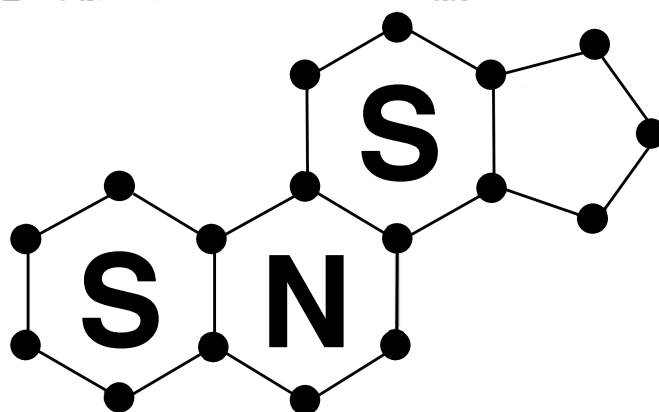


12<sup>th</sup> International Meeting

# STERIODS AND NERVOUS SYSTEM



Torino, Italy  
24-27 February 2024

**ABSTRACT BOOK**

**TORINO – 2024**

ABSTRACTS OF INVITED LECTURES  
AND FREE CONTRIBUTIONS

S. Gotti, editor

*Conference organized with the support of*

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**Fondazione Cavalieri Ottolenghi, Torino**

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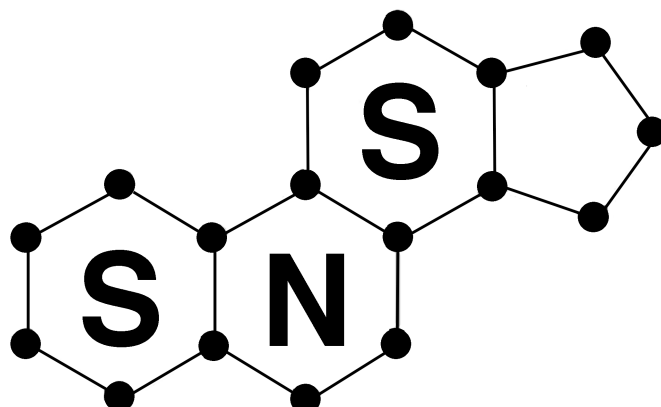
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*We are very grateful to Susanna Monteleone  
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## THE HYPOTHALAMIC-PITUITARY-GONADAL AXIS IS SUPPRESSED DURING EXPERIMENTAL AUTOIMMUNE ENCEPHALOMYELITIS IN MALE RATS

Milosevic A., Savic D., Lavrnja I., Milosevic K., Bjelobaba I., Janjic M.M.

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Multiple sclerosis (MS) is an autoimmune disease that usually occurs in both sexes during the reproductive years. Various neuroendocrine changes have been described in this inflammatory, demyelinating and debilitating disease, and many male MS patients have lower blood testosterone levels. Our aim was to determine the extent of alterations in the hypothalamic-pituitary-gonadal axis in the male rat model of MS, experimental autoimmune encephalomyelitis (EAE). During the course of the disease, hypothalamic tissue showed a transient upregulation of the inflammatory marker genes *Gfap*, *Cd68*, *Ccl2* and *Il1b*, accompanied by a downregulation of *Gnrh1* expression and pituitary *Gnrhr* expression. Serum levels of luteinizing hormone and testosterone were also reduced during the disease. To better understand the causes of decreased testosterone production during EAE, we examined the expression status of genes and proteins associated with steroidogenesis in the testes. No changes in the number of interstitial cells were detected in the EAE animals, but the expression of the gene insulin-like 3 was reduced at the peak of the disease, suggesting that the functional capacity of Leydig cells was impaired. Consistent with this finding, the expression of most steroidogenic enzyme genes and proteins was reduced during EAE, including StAR, CYP11A1, CYP17A1 and HSD3B. No signs of testicular inflammation were observed. Steroidogenesis recovered after the injection of hCG, a placental gonadotropin or busserelin acetate, an analogue of gonadotropin-releasing hormone, at the peak of EAE. Overall, our results are consistent with the hypothesis that impaired testicular steroidogenesis originates upstream of the testes and that low serum LH levels are the main cause of decreased testosterone levels during EAE.

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