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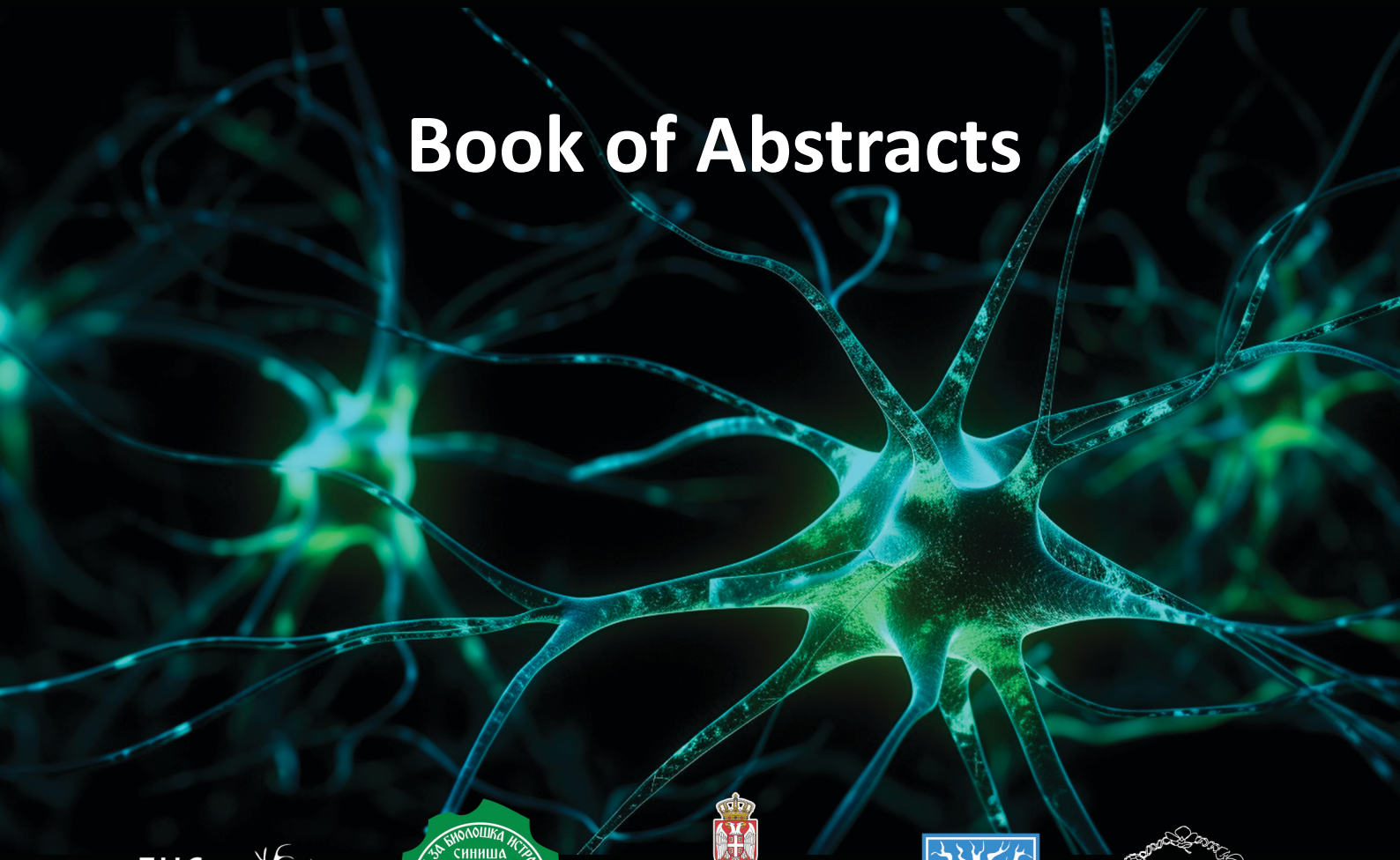

Book of Abstracts

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Protein tyrosine phosphatase receptors N and N2 regulate gonadotropin-releasing hormone neuron function

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Simultaneous knockout of the neuroendocrine marker genes *Ptprn* and *Ptprn2*, which encode the protein tyrosine phosphatase receptors N and N2, respectively, causes infertility of female mice while males are fertile. To clarify the mechanism of sex-specific roles of *Ptprn* and *Ptprn2* in mice reproduction, we analyzed the effects of their double knockout (DKO) on the hypothalamic-pituitary-gonadal axis. In DKO females, a delay in puberty and lack of ovulation were observed, supplemented by changes in ovarian gene expression and steroidogenesis. In DKO males, the testicular gene expression, steroidogenesis, and development of reproductive organs were not affected. However, in both sexes, pituitary luteinizing hormone (LH) beta gene expression and LH levels were reduced, while the calcium-mobilizing and LH secretory actions of gonadotropin-releasing hormone (GnRH) receptors were preserved. The expression of hypothalamic *Gnrh1* and *Kiss1* genes were also reduced in DKO females and males. The density of immunoreactive GnRH fibers was decreased in the median eminence in DKO females and males. The density of immunoreactive kisspeptin fibers was also decreased in the rostral periventricular region of the third ventricle of females and in the arcuate nucleus of females and males. Therefore, infertility in DKO females cannot be explained only by sex-specific gonadotroph impairment. Instead, changes in hypothalamic gene expression, specifically *Kiss1* in the rostral periventricular region of the third ventricle, might provide an alternative hypothesis due to its sexual dimorphism and involvement in puberty onset and ovulation.