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Part VI. Estrogens and Neurodegenerative Disorders

Estrogen Action in Neuroprotection and Brain Inflammation

SILVIA POZZI^a, VALERIA BENEDUSI^a, ADRIANA MAGGI^a AND ELISABETTA VEGETO^a^a Center of Excellence on Neurodegenerative Diseases, Department of Pharmacological Sciences, University of Milan, Via Balzaretti, 9, 20133 Milan, Italy**Key Words:** estrogen receptors • inflammation • neurodegenerationAddress for correspondence: Elisabetta Vegeto, Ph.D., Center of Excellence on Neurodegenerative Diseases, Department of Pharmacological Sciences, University of Milan, Via Balzaretti, 9, 20133 Milan, Italy. Voice: 0039-0250318263; fax: 0039-0250318284. e-mail: elisabetta.vegeto@unimi.it

The fertile period of women's life compared to menopause is associated with a lower incidence of degenerative inflammatory diseases. In brain, estrogens ameliorate brain performance and have positive effects on selected neural pathologies characterized by a strong inflammatory component. We thus hypothesized that the inflammatory response is a target of estrogen action; several studies including ours provided strong evidence to support this prediction. Microglia, the brain's inflammatory cells, and circulating monocytes express the estrogen receptors ER- α and ER- β and their responsiveness *in vivo* and *in vitro* to pro-inflammatory agents, such as lipopolysaccharide (LPS), is controlled by 17 β -estradiol (E₂). Susceptibility of central nervous system (CNS) macrophage cells to E₂ is also preserved in animal models of neuroinflammatory diseases, in which ER- α seems to be specifically involved. At the molecular level, induction of inflammatory gene expression is blocked by E₂. We recently observed that, differently from conventional anti-inflammatory drugs, E₂ stimulates a nongenomic event that interferes with the LPS signal transduction from the plasma membrane to cytoskeleton and intracellular effectors, which results in the inhibition

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of the nuclear translocation of NF- κ B, a transcription factor of inflammatory genes. Interference with NF- κ B intracellular trafficking is selectively mediated by ER- α . In summary, evidence from basic research strongly indicates that the use of estrogenic drugs that can mimic the anti-inflammatory activity of E₂ might trigger beneficial effects against neurodegeneration in addition to carrying out their specific therapeutic function.

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7 World Trade Center, 250 Greenwich St, 40th Fl, New York, NY 10007-2157

info@nyas.org | 212.298.8600