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Involvement of Interleukin-1β, cyclooxygenase-2, and hypoxia-inducible factor -1α in idiopathic asthenozoospermia

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Asthenozoospermia may result from different biochemical and functional defects. In particular, it has been demonstrated that reactive oxygen species (ROS) have a role in its pathogenesis. These reactive molecules may mediate the biological effects of cytokines, acting as intracellular signals.

Human sperm cells are able to produce biologically active interleukin-1β (IL-1β), which stimulates cyclooxygenase-2 (COX-2) in various cells and tissues, including human reproductive tissues, even if its effects on male reproductive biology are largely unknown. COX-2 is the inducible isoform of the key enzyme in the biosynthesis of prostaglandins, and it could be also induced by hypoxia-inducible factor -1α (HIF -1α), which regulates many genes important for oxygen homeostasis. It has been recently proposed an involvement of COX-2 in male fertility disorders.

In this scenario, our aim was to evaluate sperm parameters, as well as the expression of IL-1β, COX-2, and HIF -1α in spermatozoa isolated from normospermic fertile donors and asthenozoospermic infertile patients, by means of immunohistochemical analysis and enzyme-linked immunosorbent assay (ELISA).

Sperm cells obtained from the asthenozoospermic group showed a higher expression of IL-1β, COX-2, and HIF-1α, compared with those from normospermic fertile subjects.

Our results can lead us to speculate that the increased expression of these substances may influence the kinetic characteristics of spermatozoa. Nevertheless, further studies are in progress in order to assess whether these bioactive mediators may represent targets for future therapeutic strategies.

Keywords

Asthenozoospermia, cyclooxygenase-2, hypoxia-inducible factor -1α, immunohistochemistry, interleukin-1 β, spermatozoa.