Journal of Andrology, Vol 13, Issue 6 551-559, Copyright © 1992 by The American Society of Andrology

JOURNAL ARTICLE

The human sperm acrosome reaction does not depend on arachidonic acid metabolism via the cyclooxygenase and lipoxygenase pathways

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The objective of this study was to determine whether the metabolism of arachidonic acid via the cyclooxygenase pathway, the lipoxygenase pathway, or both has a pivotal role in the human sperm acrosome reaction. To do so, the stimulatory effect of arachidonic acid and a number of its metabolites, as well as the inhibitory effect of cyclooxygenase and lipoxygenase inhibitors on the acrosome reaction, was evaluated. Arachidonic acid, prostaglandin E2, and prostacyclin (PGI2) induced the acrosome reaction when added to 3-hour preincubated (capacitated) spermatozoa. The arachidonic acid-induced acrosome reaction was dependent upon extracellular calcium. Leukotriene B4 and 15-HPETE only induced the acrosome reaction when present throughout the preincubation period, indicating that they may enhance the capacitation process rather than the acrosome reaction. Thromboxane did not affect the acrosome reaction under any of the conditions tested. Inhibitors of cyclooxygenase (indomethacin, phenylbutazone) and lipoxygenase (phenidone, nordihydroguiaretic acid) or FPL 55712 (a leukotriene antagonist) did not prevent the arachidonic acid-stimulated acrosome reaction. Furthermore, 5, 8, 11, 14-eicosatetraynoic acid (ETYA), the acetylenic analog of arachidonic acid that inhibits arachidonic acid metabolism, induced an acrosome reaction equivalent to that of arachidonic acid. These results strongly suggest that the acrosome reaction induced by exogenous arachidonic acid is not mediated via either the cyclooxygenase pathway or the lipoxygenase pathway.(ABSTRACT TRUNCATED AT 250 WORDS)

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