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## JOURNAL ARTICLE

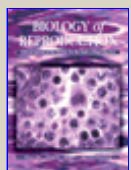
# Low levels of nitric oxide promote human sperm capacitation in vitro

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The influence of nitric oxide on human sperm hyperactivation and capacitation, as well as its mechanism of action and its possible origin from spermatozoa were studied. Percoll-washed spermatozoa from healthy volunteers were incubated in Ham's F-10 medium supplemented or not with the nitric oxide-releasing agents, diethylamine-NONOate or spermine-NONOate, in combination or not with superoxide dismutase or catalase (scavengers for the superoxide anion and for hydrogen peroxide, respectively), or with sodium nitrate, sodium nitrite, or preincubated NONOates. Sperm hyperactivation, capacitation, and nitric oxide synthase activity were determined. High concentrations (0.3 to 1 mM) of NONOates reduced sperm motility. However, a lower concentration (0.1 mM) of the two NONOates had no effect on the percentage of sperm motility or of hyperactivation but resulted in a significant increase in sperm capacitation (24% +/- 4%) when compared to that of control spermatozoa (Ham's F-10 alone, 12% +/- 2%). Nitric oxide released by the NONOates appeared responsible for this effect because sodium nitrate or nitrite or preincubated NONOates (to exhaust the formation of nitric oxide) had no influence on sperm capacitation. Catalase, but not superoxide dismutase, abolished the capacitating action of the NONOates. No nitric oxide synthase activity was detected in spermatozoa, whether they were in their basal state or already capacitated. Furthermore, the nitric oxide synthetase inhibitor L-NG nitroarginine methyl ester did not block sperm capacitation induced by fetal cord serum ultrafiltrate. It is therefore concluded that, although spermatozoa do not possess detectable nitric oxide synthase activity, low levels of nitric oxide induce human sperm capacitation, and this action likely involves hydrogen peroxide.

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