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## JOURNAL ARTICLE

# Spontaneous lipid peroxidation and production of hydrogen peroxide and superoxide in human spermatozoa. Superoxide dismutase as major enzyme protectant against oxygen toxicity

J. G. Alvarez, J. C. Touchstone, L. Blasco and B. T. Storey  
Department of Obstetrics and Gynecology, University of Pennsylvania School of Medicine, Philadelphia 19104-6080.

Spontaneous lipid peroxidation in washed human spermatozoa was induced by aerobic incubation at 32 C and measured by malonaldehyde production; loss of motility during the incubation was determined simultaneously. Malonaldehyde production at the point of complete loss of motility, defined as the lipoperoxidative lethal endpoint (LLE), was  $0.10 \pm 0.03$  nmol/10(8) cells (mean  $\pm$  SD,  $n = 40$ ), and was independent of the time to complete loss of motility. Human spermatozoa produced both  $H_2O_2$  and  $O_2^-$  during aerobic incubation. Inhibition of superoxide dismutase in these cells with KCN showed that all the  $H_2O_2$  production is due to action of the dismutase. The superoxide dismutase activity of individual human sperm samples varied between 1 and 10 U/10(8) cells, variations between samples from a single donor being nearly as great as those between different donors. The time to complete motility loss (tL) showed equal variation of 1 to 10 hours among samples. The rate of spontaneous lipid peroxidation, calculated as LLE/tL, for a given sperm sample and the superoxide dismutase activity of the same sample, determined prior to aerobic incubation, gave a good linear correlation ( $r = 0.97$ ). Glutathione reductase, glutathione peroxidase, and glutathione were found to be present in human spermatozoa, but showed little variation among samples. These results suggest that superoxide dismutase plays the major role in protecting human spermatozoa against lipid peroxidation. In addition, the superoxide dismutase activity of a fresh sperm sample appears to be a good predictor of the lifetime (up to the complete loss of motility) of that particular sample, and so may prove useful in semen analysis.

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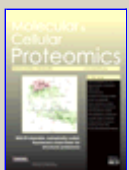
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▶ HOME

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