Need to search many journals at once?

ME HELP FEEDBACK SUBSCRIPTIONS ARCHIVE SEARCH TABLE OF CONTENTS

Q.

Journal of Andrology, Vol 15, Issue 3 255–265, Copyright $^{\odot}$ 1994 by The American Society of Andrology

JOURNAL ARTICLE

Journal of

Mechanism of superoxide dismutase loss from human sperm cells during cryopreservation

J. L. Lasso, E. E. Noiles, J. G. Alvarez and B. T. Storey Department of Obstetrics and Gynecology, University of Pennsylvania Medical Center, Philadelphia.

Earlier studies on human sperm cryodamage have shown that plasma membrane stress is the primary process and that phospholipid peroxidation in cryopreserved samples is not inhibited by addition of antioxidants. One consistent effect of cryopreservation is loss of enzymatic activity of the peroxidation defense enzyme, superoxide dismutase (SOD). To clarify this aspect of the freeze-thaw process and to develop a more complete resolution of the reactions leading to

cryodamage, we sought to identify which of the two most probable mechanisms, loss of enzyme protein from the cells of denaturation of the protein, operates. If the first operates, cellular enzymatic activity and enzyme protein as identified by immunocytochemistry should give a linear correlation. If the second operates, there should be no correlation. In this study, five individual samples were analyzed before and after cryopreservation for immunoreactive Cu/Zn SOD and cell intactness by flow cytometry, for SOD enzymatic activity by a highly sensitive fluorimetric method, and for motility characteristics by Hamilton-Thorn motility analyzer. Fresh samples were obtained by the "swim-up" method and had > 95% intact cells with > 78% motile cells. After freeze-thaw, about half the cells were intact. SOD enzymatic activity was determined on Triton X-100 cell extracts, a method that removes all enzymatic activity from the cell structure, and compared with immunoreactive SOD in the cells as determined by indirect immunofluorescence mean intensities. Residual immunofluorescence was observed in the cells after Triton X-100 treatment; if this was taken into account, a close linear correlation between SOD enzyme activity and SOD immunoreactivity was obtained (r = 0.90; P = 0.00014). There was no correlation between SOD enzyme activity ratios for cryopreserved and fresh cells and fraction of intact cells after freeze-thaw. We conclude that loss of SOD protein from the subset of cells undergoing acute membrane damage is the most probable primary mechanism of SOD enzymatic activity loss from the sample and that resistance to cryodamage and SOD activity in any given cell are guite independent of one another.

This article has been cited by other articles:

This Article

- Full Text (PDF)
- Alert me when this article is cited
- Alert me if a correction is posted

Services

- Similar articles in this journal
- Similar articles in PubMed
- Alert me to new issues of the journal
- Download to citation manager

Citing Articles

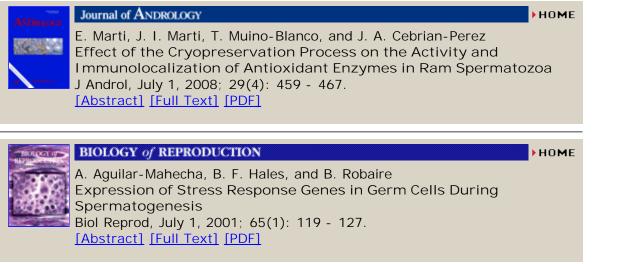
- Citing Articles via HighWire
- Citing Articles via Google Scholar

Google Scholar

- Articles by Lasso, J. L.
- Articles by Storey, B. T.
- Search for Related Content

PubMed

- PubMed Citation
- Articles by Lasso, J. L.
- Articles by Storey, B. T.



HOME HELP FEEDBACK SUBSCRIPTIONS ARCHIVE SEARCH TABLE OF CONTENTS

Copyright © 1994 by The American Society of Andrology.