CONCLUSIONS: Good pregnancy rates were achieved without significant differences among the sperm sources. The pregnancy and the delivery rate were dependent strictly on the age of the wife but not on her ovarian reserve.

Supported by: None.

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PREGNANCIES AND LIVE BIRTHS FOLLOWING INTRACYTOPLASMIC SPERM INJECTION OF TESTICULAR SPERMATOZOA AFTER REPEATED IMPLANTATION FAILURE WITH THE USE OF EJACULATED SPERM. A. Weissman, E. Horowitz, A. Ravhon, H. Nahum, A. Golan, D. Levan. IVF Unit, Department of Obstetrics and Gynecology, Edith Wolfson Medical Center, Holon, Israel; IVF Unit, Department of Obstetrics and Gynecology, Edith Wolfson Medical Center, Holon, Israel.

OBJECTIVE: The use of testicular sperm for IVF-ICSI is currently indicated exclusively for patients with azospermia, since a favorable outcome is expected even if very few spermatozoa are present in the ejaculate.

DESIGN: We describe a series of four couples with longstanding male factor infertility and multiple failed IVF-ICSI cycles. In all couples, the use of ejaculated sperm for ICSI resulted in poor embryo quality and repeated implantation failure.

MATERIALS AND METHODS: Testicular sperm aspiration was performed in subsequent cycles, and testicular sperm was used for ICSI.

RESULTS: Embryo implantation and ongoing pregnancies/deliveries were achieved in all four couples.

CONCLUSIONS: In selected patients, the use of testicular sperm for IVF-ICSI should be considered, even when motile spermatozoa can be identified in the ejaculate.

Supported by: None.

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OBJECTIVE: To assess the contribution of the male genome to the embryo’s ability to develop and implant.

DESIGN: Fertilization, embryo development, implantation and pregnancy outcomes were assessed in couples with a male partner suffering from spermatogenic failure and compared to normozoospermic couples.

MATERIALS AND METHODS: ICSI cycles from Sept 1993 to Mar 2008 were reviewed and ejaculated and TESE cycles were identified. Only normal semen samples with a concentration of ≥ 20x10^6/ml, motility of ≥ 40%, and morphology ≥ 4% normal forms as well as NOA men undergoing TESE were included in the study. Female patients were then allocated according to the age of their partner (≤ 35 yrs old) or > 35 yrs old. Fertilization, embryo cleavage, and quality, and pregnancy outcome together with losses were compared.

RESULTS: A total of 1652 ejaculated and 620 NOA patients were identified. When patients were categorized according to the maternal age and sperm source, women ≤ 35 yrs old inseminated with normozoospermic specimens (n=441) had a higher fertilization rate (79.1%) than the TESE (n=370) cycles (53.7%; p<0.001). The embryo quality and implantation rates were comparable between the two sperm sources (27.9 vs 30.6%). Furthermore, in spite of the similar number of embryos replaced, no differences were observed when clinical pregnancies (presence of a FSH) were compared (46.5 vs 44.6%). In addition, the incidence of pregnancy wastage for the ejaculated group was 4.9% (10/205) while for the NOA was 6.1% (10/165). When cycles with advanced maternal age (≥ 35 yrs old) were considered, the fertilization rate was similarly higher in the ejaculated cohort (76.7% vs 75.5; p<0.001) while the embryo quality and the average number of conceptuses transferred were comparable between the two groups. Further, the clinical pregnancy rates were similar at 31.4% and 33.2% between the ejaculated and TESE sources. The proportion of embryos that implanted in the ejaculated group was 14.5% (536/3689) while in the TESE cohort, 17.2% (122/707). Interestingly, the rate of pregnancy losses was not higher in the TESE-treated patients (10.8%) than the normozoospermic group (15.5%).

CONCLUSIONS: In contradiction to other studies implying that a subfertile male gamete is responsible for poor embryo development and impaired implantation, we observed that men with compromised spermatogenesis re-produce similarly to their normozoospermic counterparts. From these findings, maternal age remains the sole culprit for the impaired clinical outcome.

Supported by: Institutional.

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LYCOPENE ATTENUATES OXIDATIVE SPERM DNA DAMAGE IN VITRO. J. Libman, M. San Gabriel, A. Zini. Urology, McGill University Health Centre, Montreal, QC, Canada; McGill University Health Centre, Montreal, QC, Canada.

OBJECTIVE: Oxidative stress has been implicated as a potential factor in male idiopathic infertility, and, has been shown to cause sperm DNA damage and impaired sperm motility. The objective of this study was to examine in vitro effects of the anti-oxidant lycopene on human sperm motility and DNA damage.

DESIGN: Experimental human study.

MATERIALS AND METHODS: Fresh, Percoll-washed sperm suspensions (n=5) from proven fertile donors were pre-incubated for 30 minutes with or without lycopene (2 or 5 µM). The same suspensions were then incubated an additional 2 hours with hydrogen peroxide (0.50 or 100 µM) to induce DNA damage. Sperm motility (%) was recorded and DNA damage was assessed using the flow cytometry-based sperm chromatin structure assay and expressed as % DNA fragmentation index (%DFI).

RESULTS: The incubation of spermatozoa with hydrogen peroxide (50 µM) resulted in a significant decline in mean (±SD) percent motility (28 ± 13 vs. 73 ± 4%, respectively, p<0.05) and a significant increase in %DFI compared to samples incubated without hydrogen peroxide (29.8 ± 39.4 vs. 11.1 ± 14.6%, respectively, p<0.05). Pre-treatment of samples with 5 µM lycopene resulted in a significantly lower %DFI than samples treated with hydrogen peroxide alone (8.0 ± 7.9 vs. 29.8 ± 39.4 %, respectively, P<0.05). However, lycopene did not protect spermatozoa from the observed decline in motility after hydrogen peroxide treatment.

CONCLUSIONS: The data suggest that pre-incubation of spermatozoa with the anti-oxidant lycopene offers protection against oxidative stress-induced DNA damage in vitro. These data also highlight the differential protective effects of lycopene on sperm motility and sperm DNA integrity.

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CATALASE CAN PROTECT SPERMATOZOA OF FSH RECEPTOR KNOCK-OUT MICE AGAINST OXIDANT-INDUCED DNA DAMAGE IN VITRO. J. Libman, M. San Gabriel, M. R. Sairam, A. Zini. Urology, McGill University Health Centre, Montreal, QC, Canada; McGill University Health Centre, Montreal, QC, Canada; Clinical Research Institute of Montréal, Montreal, QC, Canada.

OBJECTIVE: The etiology of sperm DNA damage is likely multi-factorial: abnormal compaction of nuclear DNA, a testicular event, and oxidative stress, primarily a post-testicular event, have both been implicated as potential causes of DNA damage. The objective of this study was to evaluate sperm DNA damage in spermatozoa from wild-type (WT) and FSH receptor knock-out (FORKO) mice (a model of male infertility), compare the relative susceptibility of spermatozoa from these animals to oxidative DNA damage, and examine the protective effect of the anti-oxidant catalase on sperm DNA damage.

DESIGN: Experimental animal-based study.

MATERIALS AND METHODS: Epididymal spermatozoa from FORKO mice (n=5) and WT controls (n=5) were extracted and incubated in the presence or absence of catalase. Sperm DNA damage was assessed (1) immediately after epididymal extraction and (2) following a 2-hour incubation at 37°C. DNA damage was measured by the sperm chromatin structure assay and the results expressed as the % DNA fragmentation index or % DFI (reflecting the % of spermatozoa with DNA damage).

RESULTS: Untreated epididymal spermatozoa from WT mice had a significantly lower mean (±SD) % DFI than that of FORKO mice (2.7 ± 1.8 vs. 6.4 ± 2.9%, P<0.05). Prolonged (2-hour) incubation of FORKO mice spermatozoa with catalase resulted in a significantly lower % DFI than samples incubated in the absence of catalase (9.8 ± 4.1 vs. 17.9 ± 9.2%, respectively, P<0.05). However, incubation of WT mice spermatozoa with catalase (vs. no catalase) did not result in a significant reduction in %DFI (5.8 ± 5.0 vs. 7.7 ± 6.5%, respectively, P>0.05).

CONCLUSIONS: These data indicate that catalase may protect sperm nuclear DNA from oxidative stress in vitro. The data also demonstrate the differential susceptibility of WT and FORKO mice spermatozoa to oxidative stress.

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