

RESULTS: Outcomes are summarized in table 1. Case #2 underwent 2 stimulation cycles 1 year apart. On average 13.8 ± 5.6 oocytes were retrieved, of which 49.3% were mature. Of the immature, 16.6% were matured in vitro, increasing the mature oocyte yield to 59%. All children tolerated the procedures well and there were no complications.

CONCLUSION: Oocyte cryopreservation is a feasible technique in selected female children at risk for premature ovarian failure. During ovarian stimulation, LH supplementation is needed due to the relative immaturity of pituitary-ovarian axis and sensitivity to suppression by GnRH antagonists.

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ESTIMATED NUMBER OF MATURE OOCYTES NEEDED FOR FERTILITY PRESERVATION PATIENTS BASED ON THE NUMBER OF EUPLOID BLASTOCYSTS DIAGNOSED FOLLOWING PRE-IMPLANTATION GENETIC SCREENING (PGS). J. Barritt,^a D. Hill,^a M. Surrey,^{a,b} S. Tormasi,^c C. Welch,^c S. Munne.^c

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OBJECTIVE: For oocyte fertility preservation patients there are no well-established guidelines for how many eggs should be frozen to attain a pregnancy in the future. The goal of this study is to determine how many mature oocytes (MII) are needed to be frozen to produce enough euploid blastocysts to achieve implantation and pregnancy, categorized by SART age group.

DESIGN: Retrospective data analysis at a private fertility clinic.

MATERIALS AND METHODS: Patients who underwent PGS cycles, using micro-array comparative genomic hybridization (aCGH) and blastocyst biopsy, were analyzed to determine number of MII oocytes collected, number of blastocysts produced, euploid embryos diagnosed and implantation rate.

RESULTS: See Table

Age Group	Cycles Analysed	Average MII Oocytes	Average Blastocysts	Average Euploid Embryos	Average MII's/Euploid Embryo	Implantation Rate
<35	50	11.7	7	4.7	2.5	31.1%
35-37	33	11.7	5.6	3.6	3.3	31.5%
38-40	31	11.5	5.2	2.3	5.0	25.5%
41-42	28	9.7	4.3	0.9	10.8	26.3%
>42	30	12	3.0	0.5	24	33.3%

CONCLUSION: Our results demonstrate that the number of MII oocytes needed to obtain a diagnosed chromosomally normal embryo is highly variable across the SART age groups. Increases of greater than 100% in the number of oocytes required between the ages 38-40, 41-42 and >42, are extremely important findings for patients. Additionally, we discovered that the implantation rates between age groups do not differ when diagnosed euploid embryos are transferred after trophectoderm biopsy and aCGH screening. Counseling of potential oocyte fertility preservation patients, as to the number of mature oocytes needed to achieve an expectation of a euploid embryo in the future, can now be accomplished for each SART age group based on the clinical results from >100 PGS cycles analyzed at a single fertility clinic.

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THE IMPACT OF LONG-TERM TAMOXIFEN TREATMENT ON OVARIAN RESERVE MARKERS IN WOMEN WITH BREAST CANCER: A PROSPECTIVE-LONGITUDINAL STUDY. K. Oktay,^{a,b} G. Bedoschi,^{a,b} M. Dickler,^c S. Goldfarb,^c V. Turan,^{a,b} F. Moy,^{a,b}

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OBJECTIVE: Based on the recent ATLAS study (Lancet, 3/2013), tamoxifen (Tmx) treatment is now recommended for 10 years for women with ER+ breast cancer. This creates a dilemma for women who have not completed childbearing as Tmx is contraindicated during pregnancy. One possible strategy is to monitor ovarian reserve markers during Tmx treatment; the treatment is paused and pregnancy or fertility preservation is attempted before the reserve becomes depleted. However, reliability of ovarian reserve assessment by serum markers while on long-term Tmx treatment is unknown.

DESIGN: In this prospective study, we longitudinally studied the impact of Tmx primarily on serum AMH, and secondarily on Inhibin-B. E2 levels were also measured as Tmx is an ovarian stimulant and to control for the confounding effects of high E2 levels.

MATERIALS AND METHODS: 210 menstruating women with stage I-III breast cancer were enrolled prior to treatment and underwent longitudinal sampling at baseline, 4- and 8-months after initiating Tmx. Of those, 17 (mean age 39.5 ± 3.4 years, range 33-44) received Tmx without adjuvant chemotherapy. Patients with undetectable baseline AMH levels (n=3) were excluded.

RESULTS: Tmx treatment did not result in a statistically significant change in serum AMH, Inhibin B or Estradiol levels over 8 months follow up.

Impact of tamoxifen on ovarian reserve

Marker	Baseline (n=14)	4 Months (n=10)	8 Months (n=9)	P value
AMH (ng/ml)	1.3 ± 0.2	1.2 ± 0.3	0.9 ± 0.1	0.57
Inhibin B (pg/ml)	162.9 ± 51.6	135.5 ± 83.4	108.9 ± 61.3	0.84
Estradiol (pg/ml)	119.0 ± 28.0	166.7 ± 75.8	152.1 ± 39.0	0.75

When the analysis was limited to those from whom the complete longitudinal data were available (n= 9), the results remained the same.

CONCLUSION: This is the first study reporting the impact of long-term Tmx treatment on ovarian reserve markers. Tmx treatment does not appear to alter ovarian reserve assessment by serum AMH. This information has crucial value for breast cancer survivors, when making fertility decisions.

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RISK OF DIMINISHED OVARIAN RESERVE IN BRCA 1/2 MUTATION CARRIERS. E. T. Wang, M. D. Pisarska, C. Bresee, Y. D. I. Chen, C. Alexander, B. Y. Karlan. Cedars Sinai Medical Center, Los Angeles, CA.

OBJECTIVE: BRCA 1/2 mutation carriers have significant reproductive pressures. Not only do they face the choice of risk-reducing salpingo-oophorectomy, emerging data indicates a lower oocyte yield in fertility preservation. We set out to determine if BRCA 1/2 mutation carriers have diminished ovarian reserve (DOR), based on serum anti-Mullerian hormone (AMH) levels, compared to non-mutation carriers.

DESIGN: Cross-sectional study.

MATERIALS AND METHODS: We studied 123 women, aged 24-45, recruited from the Gilda Radner Hereditary Cancer Program from 1991-2008. All participants underwent genetic testing to detect mutations in BRCA 1/2. Participants were limited to those with intact ovaries, age ≤ 45 , and no history of breast/ovarian cancer. AMH was assayed using stored serum samples (AMH Gen II ELISA, Beckman Coulter). DOR was defined as AMH <1ng/mL. Linear and logistic regression models adjusted for age and body mass index (BMI) were performed to determine the association between BRCA 1/2 mutations and AMH.

RESULTS: Of 123 women included in this study, 75 women were BRCA 1/2 mutation carriers and 48 women were non-carriers. BRCA 1/2 mutation carriers were slightly younger (see Table 1). In multivariable linear regression analyses, BRCA 1/2 mutation carriers had a lower AMH compared to non-carriers (-0.20 ng/mL, $P=0.03$). Furthermore, BRCA 1/2 mutation carriers had >2-fold increased odds of DOR (odds ratio 2.64, 95% CI 1.11-6.27, $P=0.03$). There was no difference noted when BRCA1 and BRCA2 mutation carriers were analyzed separately.