

17th World Congress *of* the Academy of Human Reproduction

15–18 March 2017

Hotel Parco dei Principi - Rome, Italy

final program

Chairmen:

*Giuseppe Benagiano, Gian Carlo Di Renzo, Andrea R. Genazzani,
Pasquale Patrizio, Tommaso Simoncini*





Poster Sessions

Thursday 16th 11:45 - Poster Area

SESSION 1 Endometrium, Endometriosis, Fibroids, Social Science, Psychology, Cancer

This group of posters will be available for consultation from 8:45 to 18:15. Their authors will be present for discussion at 11:45. Posters considered by the Scientific Committee "of particular interest" will be presented in printed form.

Presented as printed posters:

- P1** Methylation profile of inhibitors of metalloproteinases in endometriosis
Magdalena Balkowiec (PL), Radosław Maksym (PL), Joanna Jacko (PL), Monika Grymowicz (PL), Paweł Włodarski (PL)
- P2** The impact of sexual abuse of the teenagers during highschool
Mário Cardoso (PT), Maria Presado (PT), Tiago Nascimento (PT), Andrea Carvalho (PT), Ana Frade (PT)
- P3** Mammography self-efficacy and breast cancer fear of nurses
Nülüfer Erbil (TR), Azize Nuran Kabraman (TR)
- P4** Hope and fertility related quality of life among women undergoing assisted reproductive techniques: possible mediating role of anxiety and depression
Saeedeh Lotfinikoo (IR), Reza Omani Samani (IR)
- P5** Endometrial Receptivity Defect in Endometriosis Woman Study: Insulin Growth Factor (IGF-1), Matrix Metallo Protein-9 (MMP-9), and Leptin Expression
Uki Budibastuti (ID), Tedjo Oepomo (ID), Eriana Melinawati (ID), Kautsar Heridho (ID), Andy wijaya (ID), Fajar Alam (ID)
- P6** Ceramide-1-phosphate (C1P) protects against cyclophosphamide-induced gonadotoxicity in a mouse model
Natalia Pascuali (AR), Leopoldina Scotti (AR), Mariana Di Pietro (AR), Gonzalo Oubiña (AR), María May (AR), Antonio Gómez Muñoz (AR), Dalhia Abramovich (AR), Fernanda Parborell (AR)
- P7** The presence of membrane-bound progesterone receptor induces growth of breast cancer with norethisterone but not with progesterone: a xenograft model
Xiangyan Ruan (CN), Yue Zhao (CN), Muqing Gu (CN), Alfred Otto Mueck (DE)
- P8** Fertility Preservation by Ovarian Tissue Cryopreservation among Cancer Patients in Italy: a multicentric study in Assisted Reproductive technology (ART) centers
Giulia Scaravelli (IT), Ettore Cittadini (IT), Alberto Revelli (IT), Raffaella Fabbri (IT), Raffaella De Palo (IT), Vincenzo Vigilano (IT), Roberta Spoletini (IT), Roberto De Luca (IT), Lucia Speciale (IT)
- P9** DNA-methylation in uterine myoma
Farida Eseneeva (RU), Marina Khamoshina (RU), Victor Radzinsky (RU), Vsevolod Kiselev (RU), Andrey Poloznikov (RU), Oleg Shalaev (RU), Leyla Salimova (RU)
- P10** The effect of anti-recurrent treatment of uterine fibroid with mifepristone on the state of the mammary glands in women of reproductive age
Vladislava Novikova (RU), Vadim Khorolskiy (RU)
- P11** The effect of perceived social support and marital status on hope in women with infertility
Afsaneh Kermanizadeh (IR), Adis Kraskian Mujembari (IR), Reza Omani Samani (IR), Zabra Ezabadi (IR), Abbas Rahimi Forooshani (IR), Abad Alizadeh (IR)
- P12** The experience of infertility: a qualitative research
Narges Bagheri Lankarani (IR)

Presented as e-posters:

Ceramide-1-phosphate prevents cyclophosphamide toxicity in mouse ovary

Pascuali, N¹; Scotti, L¹; Di Pietro, M¹; Oubiña G¹; de Zúñiga I²; Tesone, M¹; Abramovich, D¹; Irusta, G¹; Gómez Muñoz, A ³ and Parborell, F¹.

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Keywords: oncofertility, cyclophosphamide, C1P, ovarian reserve, fertility preservation

We investigated whether the administration of the sphingolipid ceramide-1-phosphate (C1P) can preserve ovarian function from gonadotoxicity caused by cyclophosphamide (CTX) in a mouse model. C1P protects against CTX-induced gonadotoxicity by preserving the ovarian reserve and contributing to blood vessel formation, providing a novel strategy to avoid chemotherapy-induced POF.

Premature ovarian failure (POF) is often a consequence of gonadotoxic chemoradiotherapy. Depleted follicle reserve can present with transient or permanent amenorrhea, infertility and premature menopause. In particular, alkylating agents such as cyclophosphamide (CTX) induce severe follicle loss, the proposed mechanisms being apoptosis and/or damage to ovarian microvascularization. Bioactive sphingolipids, such as C1P, are important regulators of cell homeostasis. As well as sphingosine-1-phosphate (S1P), C1P has a proangiogenic and anti-apoptotic role, with the advantage of a greater stability. Since the efficacy of gonadotropin-releasing hormone analogs is still controversial, we propose C1P as a potential protective molecule for fertility preservation. Based on these considerations, we investigated whether the administration of C1P can preserve ovarian function from gonadotoxicity caused by cyclophosphamide (CTX) in a mouse model. Twenty-four 8-week-old female F1 mice (BALB/c x C57BL/6) were separated into four groups (n=6/group). Mice received either a single intraperitoneal injection of saline (control group) or 75 mg/kg of CTX. Besides, CTX mice received intrabursal administration of vehicle (CTX group) or C1P in two different doses (CTX+C1P groups: 5ul/ovary; 0.5 mM or 10ul/ovary; 0.6 mM). Animals were euthanized on day 14, and their ovaries removed and fixed in Bouin's solution for further study. Bouin-fixed, paraffin-embedded slides of ovarian tissue were stained with hematoxylin and eosin (H&E) and the number of different stages of follicles was determined in 3 sections per ovary. Data are expressed as the percentage of each follicle type per ovary. Ovaries were also immunostained for anti-müllerian hormone (AMH) and for vonWillebrand factor by immunohistochemistry (IHC). Quantification of relative vascular areas was performed with ImageProPlus software. Firstly, we analyzed follicular structures in H&E-stained ovarian slides. In CTX-treated ovaries, percentages of primary and preantral follicles were lower (both p<0.01) and the percentage of atretic follicles was higher (p<0.01) when compared with control ovaries. Local administration of C1P in CTX-treated ovaries (both CTX+C1P groups) increased the percentage of primary and preantral follicles, and decreased the percentage atretic follicles compared to CTX alone (p<0.001). No significant differences were observed in the % of corpora

lutea between groups. Next, we studied the protein expression of AMH, a well-known marker of ovarian function, by IHC. Consistent with follicular count results, AMH expression decreased in the CTX group compared to the control group, while both doses of C1P increased AMH expression in CTX-treated ovaries. Furthermore, the IHC for Von Willebrand factor, an endothelial cell marker, showed decreased vascular area in CTX-treated ovaries compared to control ($p < 0.05$). Administration of C1P in both doses restored the vascular area to levels comparable to those of control ovaries ($p < 0.05$). In all cases, one-way ANOVA, followed by Tukey comparisons, were performed to test for statistical significances. P values < 0.05 were considered significant. Our results suggest that C1P preserves ovarian reserve in CTX-induced POF by improving follicular dynamics and ovarian angiogenesis. Given the increasing number of cancer survivors, finding effective, low-cost fertility-extending options for women undergoing life-preserving treatments is critical. C1P could be a novel strategy for fertility preservation in cancer patients.

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