

[Home](#) > [Journal](#) > [Medicine & Healthcare](#) > [OJOG](#)[Indexing](#) [View Papers](#) [Aims & Scope](#) [Editorial Board](#) [Guideline](#) [Article Processing Charges](#)[OJOG](#) > Vol.3 No.1, January 2013

OPEN ACCESS

In vivo effect of 17- β -estradiol, progesterone, hCG and expression of P53 and P21 in endometrial Ishikawa cells

PDF (Size: 345KB) PP. 105-110 DOI: 10.4236/ojog.2013.31020

Author(s)

Faruk Abike, Canan Aslan, Gokhan Kilic, Meral Koyuturk, Gozde Koksai, Nedret Altioik, Melike Ersoz

ABSTRACT

Purpose: The study examined the effect of 17- β -estradiol, progesterone and hCG on cell proliferation and the effect of cell cycles regulating P53 and P21 protein on expression levels of Ishikawa endometrium epithelium cells. Methods: Ishikawa cells was growed in flasks including DMEM-F12 medium. It was addicted 17- β -estradiol (0.4 μ M) in mediums for shows the estrogen effects to cells. Besides, hydroksiprogesteron caproate (1 μ g/ml) and hCG (20 ng/ml) were addicted to cells for shows to effect of progesterone and hCG to cells. Cell culture groups were incubated at 24 hours for assesment of cell proliferation. Although it was incubated at 48 hours for determination of P53 and P21 in cell groups. Cells in the G₁, S, G₂ and M phases of cellular cycle were marked with immunohistochemical marking of proliferated cell nuclear antigen (PCNA). S phase cell rates were also assessed using 5-bromo 2-deoxi-uridine (BrdU) marking method. Results: No difference was determined between the PCNA marked cells and control group subject to 17- β -estradiol however, a significant increase was recorded in the rate of S-phase proliferation. No relation was indicated in the comparison of increase in proliferation rate and P53 and P21 protein expression levels. The proliferation rates of cells subject to progesterone and hCG and P53 and P21 protein expression levels were identified to have very close values to control group. Conclusions: It has been concluded that the 17- β -estradiol, progesterone and hCG hormones at concentrations and durations of experiment, do not effect the P53 and P21 protein expression levels during the proliferation regulation of Ishikawa cells.

KEYWORDS

Ishikawa cells; 17- β -estradiol; Progesterone; hCG; P53; P21; Endometrium

Cite this paper

Abike, F. , Aslan, C. , Kilic, G. , Koyuturk, M. , Koksai, G. , Altioik, N. and Ersoz, M. (2013) *In vivo* effect of 17- β -estradiol, progesterone, hCG and expression of P53 and P21 in endometrial Ishikawa cells. *Open Journal of Obstetrics and Gynecology*, 3, 105-110. doi: 10.4236/ojog.2013.31020.

References

- [1] Bergeron, C. (2002) Effect of estrogens and antiestrogens on the endometrium. *Gynécologie Obstétrique & Fertilité*, 30, 933-937. doi:10.1016/S1297-9589(02)00486-1
- [2] Fanchin, R., Peltier, E., Frydman, R. and de Ziegler, D. (2001) Human chorionic gonadotropin: Does it affect human endometrial morphology in vivo? *Seminars in Reproductive Medicine*, 19, 31-35. doi:10.1055/s-2001-13908
- [3] Matsumoto, K., Moriuchi, T., Koji, T. and Nakane, P.K. (1987) Molecular cloning of cDNA coding for rat proliferating cell nuclear antigen (PCNA)/cyclin. *The EMBO Journal*, 6, 637-642.
- [4] Baserga, R. (1991) Growth regulation of the PCNA gene. *Journal of Cell Science*, 98, 433-436.
- [5] Cellis, J.E. and Madsen, P. (1986) Increased nuclear cyclin/ PCNA antigen staining of non S-phase transformed human amnion cells engaged in nucleotide excision DNA repair. *FEBS Letters*, 209, 277-283. doi:10.1016/0014-5793(86)81127-9

[Open Special Issues](#)[Published Special Issues](#)[Special Issues Guideline](#)[OJOG Subscription](#)[Most popular papers in OJOG](#)[About OJOG News](#)[Frequently Asked Questions](#)[Recommend to Peers](#)[Recommend to Library](#)[Contact Us](#)

Downloads: 52,685

Visits: 128,999

[Sponsors >>](#)

- [6] Hegele-Hartung, C., Mootz, U. and Beier, H.M. (1992) Luteal control of endometriyal receptivity and its modification by progesterone antagonists. *Endocrinology*, 131, 2446-2460. doi:10.1210/en.131.5.2446
- [7] Coskun, M. and Coskun, M. (2003) Biological dosimeter and related developments. *Cerrahpasa Journal of Medicine*, 34, 207-218.
- [8] Durmaz, R. and Vural, M. (2007) Genetics in primary and secondary glioblastoma. *Turk Norosirurji Dergisi*, 17, 80-90.
- [9] Sherr, C.J. (1996) Cancer cell cycles. *Science*, 274, 1672-1677. doi:10.1126/science.274.5293.1672
- [10] Ay, M.E., Terzioglu, O., Terzi, C. and Izci Ay, O. (2006) Kolorektal kanserlerde, P21, p27, p57 siklin bagimli kinaz inhibitor geni (CDKI) ekspresyonlarrinin degerlendirilmesi. *Akademik Gastroenteroloji Dergisi*, 5, 20-25.
- [11] LaBaer, J., Garrett, M.D., Stevenson, L.F., Slingerland, J.M., Sandhu, C., Chou, H.S. et al. (1997) New functional activities for the P21 family of CDK inhibitors. *Genes & Development*, 11, 847-862. doi:10.1101/gad.11.7.847
- [12] Dotto, G.P. (2000) P21(WAF1/Cip1): More than a break to the cell cycle? *Biochim Biophys Acta*, 1471, 43-56.
- [13] Tsihlias, J., Kapusta, L. and Slingerland, J. (1999) The prognostic significance of altered cyclin-dependent kinase inhibitors in human cancer. *Annual Review of Medicine*, 50, 401-423. doi:10.1146/annurev.med.50.1.401
- [14] Heneweer, C., Schmidt, M., Denker, H.W. and Thie, M. (2005) Molecular mechanisms in uterine epithelium during trophoblast binding: The role of small GTPase RhoA in human uterine Ishikawa cells. *Journal of Experimental & Clinical Assisted Reproduction*, 2, 4. doi:10.1186/1743-1050-2-4
- [15] Nishida, M. (2002) The Ishikawa cells from birth to the present. *Human Cell*, 15, 104-117. doi:10.1111/j.1749-0774.2002.tb00105.x
- [16] Croxtall, J.D., Elder, M.G. and White, J.O. (1990) Hormonal control of proliferation in the Ishikawa endometriyal adenocarcinoma cell line. *Journal of Steroid Biochemistry*, 35, 665-669. doi:10.1016/0022-4731(90)90306-D
- [17] Kayisli, U.A., Aksu, C.A.H., Berkkanoglu, M. and Arici, A. (2002) Estrogenicity of isoflavones on human endometriyal stromal and glandular Cells. *The Journal of Clinical Endocrinology & Metabolism*, 87, 5539-5544.
- [18] Shiozawa, T., Horiuchi, A., Kato, K., Obinata, M., Konishi, I., Fujii, S. and Nikaido, T. (2001) Up-regulation of p27Kip1 by progestins is involved in the growth suppression of the normal and malignant human endometriyal glandular cells. *Endocrinology*, 142, 4182-4188. doi:10.1210/en.142.10.4182
- [19] Taskin, S., Ozmen, B. and ünlü, C. (2006) Therapeutic use of the levonorgestrel releasing intrauterine system. *Journal of the Turkish German Gynecological Association*, 7, 63-67.
- [20] Wolkersdorfer, G.W., Bornstein, S.R., Hilbers, U., Zimmermann, G., Biesold, C., Lehmann, M., et al. (1998) The presence of chorionic gonadotrophin beta subunit in normal cyclic human endometriyum. *Molecular Human Reproduction*, 4, 179-184. doi:10.1093/molehr/4.2.179
- [21] Lei, Z.M., Reshef, E. and Rao, V. (1992) The expression of human chorionic gonadotropin/luteinizing hormone receptors in human endometriyal and myometrial blood vessels. *The Journal of Clinical Endocrinology & Metabolism*, 75, 651-659. doi:10.1210/jc.75.2.651
- [22] Kornyei, J.L., Lei, Z.M. and Rao, C.V. (1993) Human myometrial smooth muscle cells are novel targets of direct regulation by human chorionic gonadotropin. *Biology of Reproduction*, 49, 1149-1157. doi:10.1095/biolreprod49.6.1149
- [23] Prapas, N., Tavaniotou, A., Panagiotidis, Y., Prapa, S., Kasapi, E., et al. (2009) Low-dose human chorionic gonadotropin during the proliferative phase may adversely affect endometrial receptivity in oocyte recipients. *Gynecological Endocrinology*, 25, 53-59. doi:10.1080/09513590802360769
- [24] Stevenson, A.F. (2000) Human granulosa cells in vitro: Characteristics of growth, morphology and influence of some cytokines on steroidogenesis. *Indian Journal of Experimental Biology*, 38, 1183-1191.

- [25] Smith, M.L. and Seo, Y.R. (2002) P53 regulation of DNA excision repair pathways. *Mutagenesis*, 17, 149-156. doi:10.1093/mutage/17.2.149
- [26] Maia Jr., H., Maltez, A., Studart, E., Athayde, C. and Coutinho, E.M. (2004) Ki-67, Bcl-2 and P53 expression in endometriyal polyps and in the normal endometriyum during the menstrual cycle. *British Journal of Obstetrics and Gynaecology*, 111, 1242-1247. doi:10.1111/j.1471-0528.2004.00406.x
- [27] Isaksson, E., Cline, J.M., Skoog, L., Soderqvist, G., Wilking, N., von Schoultz, E., et al. (1999) P53 expression in breast and endometriyum during estrogen and tamoxifen treatment of surgically postmenopausal cynomolgus macaques. *Breast Cancer Research and Treatment*, 53, 61-67. doi:10.1023/A:1006172025349
- [28] Herr, D., Keck, C., Tempfer, C. and Pietrowski, D. (2004) Chorionic gonadotropin regulates the transcript level of VHL, P53, and HIF-2alpha in human granulosa lutein cells. *Molecular Reproduction and Development*, 69, 397-401.
- [29] Yaron, Y., Schwartz, D., Evans, M.I., Aloni, R., Kapon, A. and Rotter, V. (1999) P53 tumor suppressor gene expression in the mouse ovary during an artificially induced ovulatory cycle. *Journal of Reproductive Medicine*, 44, 107-114.
- [30] Wang, H.B., Lu, S.H., Lin, Q.X., Feng, L.X., Li, D.X., Duan, C.M., et al. (2010) Reconstruction of endometrium in vitro via rabbit uterine endometrial cells expanded by sex steroid. *Fertility and Sterility*, 93, 2385-2395. doi:10.1016/j.fertnstert.2009.01.091
- [31] Chiang, C.H., Cheng, K.W., Igarashi, S., Nathwani, P.S. and Leung, P.C. (2000) Hormonal regulation of estrogen receptor alpha and beta gene expression in human granulosa-luteal cells in vitro. *The Journal of Clinical Endocrinology & Metabolism*, 85, 3828-3839. doi:10.1210/jc.85.10.3828
- [32] Karlsson, S., Iatropoulos, M.J., Williams, G.M., Kangas, L. and Nieminen, L. (1998) The proliferation in uterine compartments of intact rats of two different strains exposed to high doses of tamoxifen or toremifene. *Toxicologic Pathology*, 26, 759-768. doi:10.1177/019262339802600608
- [33] El-Deiry, W.S., Tokino, T., Velculescu, V.E., Levy, D.B., Parsons, R., Trent, J.M., et al. (1993) WAF1, a potential mediator of P53 tumor suppression. *Cell*, 75, 817-825. doi:10.1016/0092-8674(93)90500-P
- [34] Zhang, H., Hannon, G.J. and Beach, D. (1994) P21-containing cyclin kinases exist in both active and inactive states. *Genes & Development*, 8, 1750-1758. doi:10.1101/gad.8.15.1750
- [35] Macleod, K.F., Sherry, N., Hannon, G., Beach, D., Tokino, T., Kinzler, K., et al. (1995) P53-dependent and independent expression of P21 during cell growth, differentiation, and DNA damage. *Genes & Development*, 9, 935-944. doi:10.1101/gad.9.8.935