Journal of Fertilization : In Vitro, IVF-Worldwide, Reproductive Medicine, Genitics & Stem Cell Biology

Li, J IVF Reprod Med Genet 2014, 2:1
DOI: 10.4172/2375-4508.1000e105

Editorial

Crosstalk between brca1 and Hormone Receptor Signaling Pathways in Ovarian Cancer Progression

Da Li*

Department of Obstetrics and Gynecology, Shengjing Hospital, China Medical University, Shenyang 110004, China

Introduction

Ovarian cancer is characterized by a high rate of mortality among gynecologic oncology patients [1]. To date, although the exact cause of ovarian cancer are poorly understood, BRCA mutations are the well-known hereditary factor [2], and the risk of ovarian cancer conferred by BRCA mutations can be regulated by both genetic and environmental influences [3]. BRCA1 is a tumor suppressor gene which plays an important part in multiple cellular events, including DNA repair, cell-cycle checkpoints and genomic integrity [4]. Recent research has confirmed that BRCA1 is an important transcriptional regulator, and BRCA1-knockdown exhibited the changes of 7% mRNAs expression profiles in MCF-7 cells [4]. Moreover, our recent study also indicated that differential epigenetic regulation of transcription exist along with BRCA1 dysfunction [5,6]. Therefore, one can speculate that there are wide ranges of gene expression and regulation differences between BRCA1 inactivation and the basal phenotype. Notably, a growing body of data suggests that BRCA1 has extensive cellular effects on hormone receptor signaling pathways. For example, BRCA1 can inhibit Progesterone Receptor (PR) activity in the PR-positive T47D cells [7,8] and repress estrogen receptor-alpha activity in MCF-7 cells [9]. BRCA1 may also be a potential regulator of the insulin-like growth factor 1 receptor in HCC1937 cells [10]. In addition, the Epidermal Growth Factor Receptor (EGFR) is a member of the ErbB family of receptor tyrosine kinases that is a potential link between genetic and environmental interactions [11]. Angiotensin II Type 1 Receptor (AGTR1) interacts with genetic and environmental factors, which exert a potent effect on the proliferation and survival of the estrogen-induced Ishikawa cell line [12]. Our recent study has just confirmed that both EGFR and AGTR1 is downstream target for BRCA1 in SKOV3 cells [13,14]. In this way, there is an extensive crosstalk among BRCA1 signaling pathways and hormone receptor signaling pathways in ovarian cancer progression. Therefore, if we can clarify the complex interactions between BRCA1 and hormone receptor signaling pathways at the transcriptional, posttranscriptional, and epigenetic levels, this may improve our understanding of the basic molecular mechanism of ovarian cancer.

Acknowledgements

This work was supported by the Higher Specialized Research Fund for Doctoral Program of Ministry of Education of China (No. 20122104110027).

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***Corresponding author:** Da Li, Department of Obstetrics and Gynecology, Shengjing Hospital, China Medical University, Shenyang 110004, China, Tel: 86-024-96615-42111; Fax: 86-024-83955092; E-mail: leeda@ymail.com

Received January 07, 2014; **Accepted** January 08, 2014; **Published** January 10, 2014

Citation: Li D (2014) Crosstalk between brca1 and Hormone Receptor Signaling Pathways in Ovarian Cancer Progression. J IVF Reprod Med Genet 2: e105. doi:10.4172/2375-4508.1000e105