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Editorial

Visfatin, breastfeeding, and breast cancer

Breast cancer is a big health issue in the world, including Taiwan, since the actual incidence of invasive breast cancer increased significantly. In 2011, the incidence rate (IR) of breast cancer was 64.3 per 100,000 women after adjustment for agecontributing to a mortality rate of 15.99 per 100,000 women, which represented the fourth highest mortality rate of all cancer death, and the IR of breast cancer seemed to be continuously elevated [1,2]. Although hereditary factors play an important role in development of breast cancer [3], there is no doubt that the link between hormones, cytokines, metabolic parameters, environmental factors, and reproductive history and breast cancer growth and development has been recognized for more than a century [4,5].

Breastfeeding not only decreases the risk of developing breast cancer but also decreases the risk of recurrence in women treated for breast cancer. A recent United States study analyzed information on 1636 women with breast cancer from two prospective breast cancer cohorts, and results showed that women who had ever breast fed had a 30% lower risk of recurrence than women who had not [hazard ratio (HR), 0.70; 95% confidence interval (CI), 0.53–0.93], and an even lower risk was found in those who had breast fed for 6 months or longer (HR 0.63, 95% CI 0.46–0.87; p trend = 0.01) [6]. The benefits of breastfeeding are also significantly evident in the declined breast cancer deaths [6]. All suggested that breastfeeding may set up a molecular environment that makes the tumor less likely to metastasize, treatable with hormonal therapy such as tamoxifen, and generally have better outcomes. In the past issue of the *Taiwanese Journal of Obstetrics and Gynecology*, Dr. Shen et al [7] have an article addressing this important topic—breastfeeding effects on visfatin levels in postpartum women. Results showed that women with continuous breastfeeding had higher levels of visfatin than those without breastfeeding and a longer duration of breastfeeding showed a higher level of visfatin [7].

The authors highlighted the value of this article, because results showed that significant correlations were found between visfatin and hemoglobin A1c ($r = -0.425$, $p = 0.022$) at the 2nd week point and triglycerides ($r = -0.387$, $p = 0.038$) at the 16th week [7]. Therefore, their findings might shed a light on the necessity of further exploration of the mechanisms through which lactation may influence the occurrence of diabetes. We congratulate the authors on their success and also appreciate their contribution to this field.

Dr. Shen et al's [7] report favored the benefits of breastfeeding on metabolic status; however, their results might conflict with the recent understanding of the relationship between serum levels of visfatin and risk of breast cancer and other cancers, such as endometrial cancer and colon cancer. In addition, visfatin plays an important role in a variety of metabolic and stress responses, as well as in cellular energy metabolism as nicotinamide phosphoribosyl-transferase and contributes to the generation of nicotinamide adenine dinucleotide biosynthesis, which was significantly upregulated by a hypoxic microenvironment [8]. All hinted that visfatin might be important during the stress situation. Although not all, stress-related proteins are often reported to correlate with many unhealthy situations, including development of cancer and chronic illness [9]. Furthermore, a previous study showed a higher visfatin expression in breast cancer tissue correlating with more malignant cancer behavior, and estrogen receptor (ER) and progesterone receptor (PR) negativity, indicators of poor prognosis and survival [8], but Dr. Shen et al [7] found that continuous breastfeeding for at least 16 weeks could induce increasing visfatin levels.

Does the higher visfatin level increase the risk of development of breast cancer? Based on Dr. Shen et al's [7] report, it might be against the positive correlation between visfatin level and risk of breast cancer. Therefore, we would like to introduce the mechanisms of visfatin in cancers. Several pathways are suggested, including visfatin activating cell cycle progression by upregulation of cyclins, other cyclin-dependent kinases, increased synthesis of genes playing a significant role in tumor-related angiogenesis, such as vascular epidermal growth factor, in metastases and invasion, such as matrix metalloprotease [10]. Unlike the above findings, the role of visfatin on normal and abnormal metabolic function is relatively unclear. Dr. Shen et al's [7] report might support our understanding of the positive correlation between abnormal metabolism status and an increased risk of many hormone-dependent malignant tumors, including breast cancer, if an increased visfatin level might be against the metabolic diseases, such as diabetes mellitus.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

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