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Original Article

# Breastfeeding effects on visfatin levels in postpartum women

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ABSTRACT

*Objective:* To investigate the effect of breastfeeding on the change of visfatin levels in postpartum women.
*Materials and methods:* Twenty-nine postpartum women were enrolled into the study. All measurements were completed at the 2nd and 16th week postpartum. Women who had been continuously breastfeeding (10 women), those who had never breastfed (9 women) and those who had breastfed for a short period of time but then ceased (10 patients) were enrolled into the study. Serum levels of insulin, glucose, cholesterol, triglycerides, and visfatin were measured.
*Results:* In the continuously breastfeeding group, the women at the 16-week point had higher levels of visfatin (14.5 ± 4.1, 17.0 ± 5.1 ng/mL; $p = 0.001$). In the never breastfed group, the women at the 16-week point had higher levels of visfatin (22.6 ± 3.9, 19.1 ± 3.0 ng/mL; $p = 0.005$). The visfatin levels in the breastfed but ceased group were found to not be significantly different between the two test points (13.3 ± 2.5, 13.4 ± 2.5 ng/mL; $p = 0.815$). Regarding the association between serum visfatin and markers of lipid metabolism, significant correlations were found between visfatin and hemoglobin A1c ($r = -0.425$, $p = 0.022$) at the 2-week point and triglycerides ($r = -0.387$, $p = 0.038$) at the 16th week.
*Conclusion:* Continuous breastfeeding for at least 16 weeks could induce increasing visfatin levels. The findings of our study might shed light on the necessity of further exploration of the mechanisms through which lactation may influence the occurrence of diabetes.


## Introduction

Visfatin, known as a pre-B cell colony-enhancing factor or nicotinamide phosphoribosyltransferase (Nampt), is a 52-kDa protein originally isolated as a secreted factor. Visfatin synergizes with interleukin-7 and stem cell factors to promote the growth of B cell precursors [1]. More recently, visfatin has been identified as an adipokine, predominantly expressed in and secreted from visceral adipose tissue while exerting a number of insulin-mimetic effects [2]. The elevation in visfatin concentration has been found in obesity, insulin resistance states, type 2 diabetes mellitus (DM), and polycystic ovary syndrome (PCOS) [3–8]. These studies suggested that increased visfatin in women with PCOS and severe obesity may be a compensatory response to insulin resistance [9,10]. Some studies, however, have shown contradictory results: visfatin levels were found to be reduced in women with type 2 DM and those with gestational DM [11–13]. These results suggested that a decrease in serum visfatin levels could be related to the failure of glucose homeostasis.

Lactation imposes a substantial metabolic burden and total energy expenditure on mothers [14,15]. Lactation may attenuate adverse metabolic risk factor changes that occur with pregnancy and therefore might affect a woman's future risk of cardiovascular and metabolic diseases. Duration of lactation and sustained lactation-associated metabolic changes were found to be associated with reduced incidence of type 2 DM [16–18]. Prolonged lactation was associated with a "lasting protective effect" on insulin secretion [19]. A study by McManus et al showed that lactating women did have a higher disposition index, indicating more efficient pancreatic β cell function [20]. The relationship between lactation and metabolic risk factors may be related to higher energy use or other effects on metabolism [21]. However, mechanisms that

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explain the relationship between lactation and reduced incidence of type 2 DM are still unclear. Because visfatin has been suggested to exert protective effects on pancreatic β cell function, we hypothesize that the breastfeeding-related physiological changes influence serum visfatin levels. The objective of the current study was to investigate: (1) the change of visfatin levels in breastfeeding in postpartum women, and (2) the correlation of visfatin levels with insulin resistance, glucose, and lipid levels.

## Methods

Twenty-nine postpartum women were enrolled into the study. According to their breastfeeding status, they were divided into three groups: women who had been continuously breastfeeding (10 patients); those who had never breastfed (9 patients); and those who had breastfed for a short period of time but then ceased (10 patients). Blood samples were obtained directly from a cannulated vein on the 2nd week and the 16th week postpartum. The serum was separated by centrifugation and stored at −20°C until further analysis. Serum visfatin concentration was analyzed using an enzyme immunosorbent assay according to the manufacturer's instructions (Phoenix Pharmaceuticals, Belmont, CA, USA). The intra-assay and interassay coefficients of variation were 5.5% and 10.1%, respectively. Glucose, hemoglobin A1c, blood urea nitrogen (BUN), creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT), total cholesterol, and triglycerides were analyzed using LX-20 pro chemistry analyzers (Beckman Coulter, Brea, CA, USA). Insulin was measured using a Coat-A-count radioimmunoassay kit (Diagnostic Products Corp, Los Angeles, CA, USA). All women were of Han Chinese origin and delivered and had follow-up visits at the Department of Obstetrics at the Kaohsiung Medical University Hospital. The women were in good health and had not used medications known to affect sex hormones, lipids, or carbohydrate metabolism. Women who had children with fetal anomalies, or who had thyroid disease, preexisting hypertension, DM, or other chronic diseases were excluded from the study. A precise medical history, including body mass index (BMI), was obtained. The approval of the institutional review board (IRB) was obtained and the consent procedure was approved by the Ethics Committee of Kaohsiung Medical University Hospital. Written informed consent was received from all participants involved in the study.

Data were evaluated using SPSS software for Windows (version 12.0; SPSS Inc, Chicago, IL, USA) and presented as mean ± standard deviation. Differences between two points were evaluated using a paired $t$ test. Pearson correlation and linear univariate analysis were carried out to determine the relationship between the variables. All tests were two-tailed, and the significance level was defined as $p < 0.05$.

## Results

The clinical features are shown in Tables 1–3 for the three groups. There were no major differences between the two time points (2nd week and 16th week postpartum) with respect to the levels of AST, ALT, creatinine, glucose, insulin, and hemoglobin A1c in all three groups. The patients at the 16-week point had lower levels of cholesterol, body weight, BMI, and BUN in all three groups. However, only the continuously breastfeeding group had lower levels of triglycerides at the 16-week point. In the continuously breastfeeding group, the women at the 16-week point had higher levels of visfatin (14.5 ± 4.1, 17.0 ± 5.1 ng/mL; $p = 0.001$). In the never-breastfed group, the patients at the 16-week point had higher levels of visfatin (22.6 ± 3.9, 19.1 ± 3.0 ng/mL; $p = 0.005$). The visfatin levels in the breastfed but ceased group were found

**Table 1**
Clinical data of the "feeding" group.

| | Feeding | | |
|---|---|---|---|
| | 2nd wk ($n = 10$) | 16th wk ($n = 10$) | $p$ |
| Age (y) | 32.8 ± 3.9 | | |
| Body weight (kg) | 60.2 ± 7.2 | 58.3 ± 6.1 | 0.005** |
| BMI (kg/m$^2$) | 24.1 ± 2.5 | 23.4 ± 2.1 | 0.005** |
| AST (IU/L) | 19.6 ± 5.5 | 19.3 ± 4.7 | 0.790 |
| ALT (IU/L) | 25.1 ± 12.7 | 21.7 ± 7.4 | 0.281 |
| BUN (mg/dL) | 15.0 ± 3.4 | 12.8 ± 3.3 | 0.041* |
| Creatinine (mg/dL) | 0.6 ± 0.1 | 0.6 ± 0.1 | 0.103 |
| Insulin (μIU/mL) | 3.5 ± 2.9 | 3.1 ± 1.4 | 0.721 |
| Glucose (AC) (mg/dL) | 88.0 ± 14.5 | 91.6 ± 5.1 | 0.434 |
| Cholesterol (mg/dL) | 235.6 ± 50.8 | 198.7 ± 28.8 | 0.044* |
| Triglycerides (mg/dL) | 94.9 ± 35.1 | 63.2 ± 37.3 | 0.043* |
| Hemoglobin A1c (%) | 5.4 ± 0.3 | 5.4 ± 0.3 | 0.591 |
| Visfatin (ng/mL) | 14.5 ± 4.1 | 17.0 ± 5.1 | 0.001** |

Values are expressed as mean ± standard deviation. "Feeding" refers to women who had been continuously breastfeeding.
*$p < 0.05$, **$p < 0.01$.
ALT = alanine aminotransferase; AST = aspartate aminotransferase; BMI = body mass index; BUN = blood urea nitrogen.

**Table 2**
Clinical data of the "stopped feeding" group.

| | Stopped feeding | | |
|---|---|---|---|
| | 2nd wk ($n = 10$) | 16th wk ($n = 10$) | $p$ |
| Age (y) | 30.4 ± 4.2 | | |
| Body weight (kg) | 58.1 ± 8.1 | 56.2 ± 8.4 | 0.027* |
| BMI (kg/m$^2$) | 23.0 ± 1.8 | 22.2 ± 2.0 | 0.026* |
| AST (IU/L) | 20.5 ± 3.6 | 20.2 ± 3.7 | 0.790 |
| ALT (IU/L) | 24.6 ± 6.9 | 21.1 ± 5.4 | 0.144 |
| BUN (mg/dL) | 12.9 ± 3.7 | 10.3 ± 2.8 | 0.085 |
| Creatinine (mg/dL) | 0.6 ± 0.1 | 0.6 ± 0.1 | 0.514 |
| Insulin (μIU/mL) | 3.2 ± 1.6 | 3.8 ± 2.0 | 0.335 |
| Glucose (AC) (mg/dL) | 91.0 ± 13.9 | 92.4 ± 9.7 | 0.535 |
| Cholesterol (mg/dL) | 231.8 ± 34.5 | 180.5 ± 26.6 | <0.001** |
| Triglycerides (mg/dL) | 89.4 ± 67.3 | 81.0 ± 62.6 | 0.191 |
| Hemoglobin A1c (%) | 5.1 ± 0.5 | 5.2 ± 0.4 | 0.460 |
| Visfatin (ng/mL) | 13.3 ± 2.5 | 13.4 ± 2.5 | 0.815 |

Values are expressed as mean ± standard deviation. "Stopped feeding" refers to those who breastfed for a short period of time but then ceased.
*$p < 0.05$, **$p < 0.01$.
ALT = alanine aminotransferase; AST = aspartate aminotransferase; BMI = body mass index; BUN = blood urea nitrogen.

**Table 3**
Clinical data of the "no feeding" group.

| | No feeding | | |
|---|---|---|---|
| | 2nd wk ($n = 9$) | 16th wk ($n = 9$) | $P$ |
| Age (y) | 31.2 ± 3.3 | | |
| Body weight (kg) | 60.5 ± 6.8 | 56.3 ± 5.6 | 0.012* |
| BMI (kg/m$^2$) | 23.8 ± 2.8 | 22.2 ± 2.5 | 0.012* |
| AST (IU/L) | 19.2 ± 2.1 | 18.7 ± 3.3 | 0.594 |
| ALT (IU/L) | 20.6 ± 7.3 | 16.3 ± 4.3 | 0.111 |
| BUN (mg/dL) | 14.5 ± 3.3 | 11.3 ± 1.9 | 0.004** |
| Creatinine (mg/dL) | 0.6 ± 0.1 | 0.6 ± 0.1 | 0.377 |
| Insulin (μIU/mL) | 2.7 ± 1.2 | 3.3 ± 1.0 | 0.107 |
| Glucose (AC) (mg/dL) | 89.7 ± 5.3 | 91.2 ± 3.9 | 0.607 |
| Cholesterol (mg/dL) | 238.3 ± 51.9 | 188.0 ± 31.1 | 0.015* |
| Triglycerides (mg/dL) | 83.2 ± 46.8 | 76.0 ± 33.3 | 0.622 |
| Hemoglobin A1c (%) | 5.1 ± 0.3 | 5.2 ± 0.3 | 0.347 |
| Visfatin (ng/mL) | 22.6 ± 3.9 | 19.1 ± 3.0 | 0.005** |

Values are expressed as mean ± standard deviation. "No feeding" refers to those who had never breastfed.
*$p < 0.05$, **$p < 0.01$.
ALT = alanine aminotransferase; AST = aspartate aminotransferase; BMI = body mass index; BUN = blood urea nitrogen.