Hematologic parameters as the predictors for metabolic syndrome in perimenopausal and postmenopausal women living in urban area: a preliminary report

Patsama Vichinsartvichai, Siriwan Sirirat

Department of Obstetrics and Gynecology, Faculty of Medicine Vajira Hospital, Navamindradhiraj University, Bangkok, Thailand

Abstract

Introduction: Prevalence of metabolic syndrome increases drastically during menopausal transition. Chronic inflammation is proposed as the basic pathophysiology of metabolic syndrome (MetS).

Aim of the study: To compare mean white blood cell count between perimenopausal and postmenopausal women with and without MetS and find the prevalence of MetS in this patient group.

Material and methods: A total of 140 healthy perimenopausal and postmenopausal women were interviewed and underwent anthropometric measurements, biochemical investigations for MetS and hematologic parameters. MetS was defined according to the Joint Interim Statement 2009 criteria. The outcome measures were the hematologic parameters between women with and without MetS, correlation of hematologic parameters with MetS components and optimum cutoff for MetS prediction.

Results: The mean age of participants was 50 years. 63.6% were perimenopausal and 36.4% were postmenopausal ones. The prevalence of MetS was 21.4% (95% CI: 15.0-27.9). The women with MetS had a significantly higher level of white blood cell (WBC) counts (7,466.7 and 6,514.6; \( p = 0.006 \)) and total lymphocyte counts (2,572.0 and 2,207.7; \( p = 0.003 \)). The optimum cutoff of WBC counts and total lymphocyte counts for prediction of metabolic syndrome was 6,750 cells/ml (sensitivity = 0.633; specificity = 0.591, \( p = 0.019 \)) and 2,232 cells/ml (sensitivity = 0.667; specificity = 0.518, \( p = 0.016 \)), respectively.

Conclusion: White blood cell and total lymphocyte counts were higher in perimenopausal and postmenopausal women with MetS. However, both hematologic parameters were poor predictors for MetS in peri- and postmenopausal women.

Key words: metabolic syndrome, white blood cell count, total lymphocyte count, menopause, hematologic parameters.

Introduction

In the recent years, metabolic syndrome (MetS) has been unquestionably recognized as the major predisposing cardiovascular risk factor [1] (impaired glucose tolerance, hypertension, dyslipidemia and central obesity) and other chronic conditions [2-6], all of which increase the mortality rate [7]. The prevalence of metabolic syndrome increases steeply during the menopausal transition [8-10]. This surge of prevalence was explained by a substantial increase in waist circumference and fat mass, especially visceral fat mass throughout the estrogen recessional period [6, 11, 12].

In spite of unascertained etiology of MetS, the chronic systemic inflammatory state seems to be the pivotal mechanism underlying MetS development [13] through complex pathways such as monocyte chemoattractant protein-1 (MCP-1), tumor necrosis factor \( \alpha \) (TNF-\( \alpha \)) and interleukin (IL-6) [14], serine phosphorylation of insulin receptor substrate-1 (IRS-1) increment through activation of c-Jun N-terminal kinase (JNK) and I\( \kappa \)B kinase (IKK) [15], and toll-like receptor (TLR4) signaling pathway [16]. White blood cell (WBC) count is a routinely measured marker of systemic inflammation and elevated WBC count or its subtype is intimately linked to the prevalence of MetS in previous population-base studies [17-19]. Other hematologic parameters including platelet count and hemoglobin are also associated with MetS and its components in some studies [18].

To the best of our knowledge, there is no study focusing on the association between hematologic parameters and MetS in perimenopausal and postmenopausal women. The objectives of the present study were to compare mean WBC count between perimenopausal and postmenopausal women with and without MetS, to determine the prevalence of MetS in this patient group,
to determine a correlation of hematologic parameters with MetS components, and to find a predictive value and optimum cutoff level of hematologic parameters associated with MetS.

**Material and methods**

The cross-sectional study was carried out in the Women Health Clinic, Department of Obstetrics and Gynecology, Faculty of Medicine Vajira Hospital, Naresuan University, a tertiary-care university hospital. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki, and the study protocol was approved by the Vajira Institutional Review Board.

**Study design and participants**

The study was conducted in perimenopausal and postmenopausal women defined according to the STRAW+10 definition [20], aged at least 40 years, living in the urban area of the capital city of Thailand, who attended health checkups at the women health clinic from September 2014 to April 2015. Participants excluded from the study were women having a history of cancer, cardiovascular disease, stroke, immunosuppressive therapy, hysterectomy, diagnosed with inflammatory disease (arthritis, inflammatory bowel disease, psoriasis, etc.), polycystic ovary syndrome, steroid or contraceptive therapy, hysterectomy, diagnosed with inflammatory disease (arthritis, inflammatory bowel disease, psoriasis, etc.), polycystic ovary syndrome, steroid or contraceptive therapy. The study was conducted in perimenopausal and postmenopausal women defined according to the STRAW+10 definition [20], aged at least 40 years, living in the urban area of the capital city of Thailand, who attended health checkups at the women health clinic from September 2014 to April 2015. Participants excluded from the study were women having a history of cancer, cardiovascular disease, stroke, immunosuppressive therapy, hysterectomy, diagnosed with inflammatory disease (arthritis, inflammatory bowel disease, psoriasis, etc.), polycystic ovary syndrome, steroid or contraceptive therapy.

After written informed consent was obtained, all study participants were subjected to clinical and biochemical investigations. The socioeconomic data and medical history were collected, which included demographic data, lifestyle (alcohol consumption, smoking), menstrual history and family history of metabolic diseases.

The physical examinations of participants were performed including height (in cm), weight (in kg), waist circumference (in cm), and blood pressure (in mmHg). Waist circumference was measured at a level midpoint between the lower rib margin and the top of the iliac crest. Blood pressure of the participants was measured twice with a standardized mercury sphygmomanometer in a sitting position at least 60 seconds apart. The average of the two measurements was recorded. The body mass index (BMI) was then calculated and categorized into normal (BMI < 23.0 kg/m²), overweight (BMI 23.0–29.9 kg/m²), and obese (BMI ≥ 30.0 kg/m²), according to the classification adopted by the World Health Organization [21].

After overnight fast, the biochemical blood tests including complete blood count, fasting blood glucose, triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) were performed. The biochemical assays were conducted in the ISO 15189 certified biochemical laboratory of the Department of Clinical Pathology.

**Criteria for diagnosis of metabolic syndrome**

In the present study, we used the Joint Interim Statement (JIS) 2009 criteria [22]. The participants were diagnosed with metabolic syndrome if they had at least three out of five of the following factors: 1) abdominal obesity defined as waist circumference ≥80 cm for Asian women; 2) elevated triglycerides ≥150 mg/dl or drug treatment for elevated triglycerides; 3) reduced HDL-C <50 mg/dl or drug treatment for reduced HDL-C; 4) elevated blood pressure defined as systolic ≥130 mmHg and/or diastolic ≥85 mmHg or antihypertensive drug treatment; 5) elevated fasting glucose ≥100 mg/dl or drug treatment of elevated glucose.

**Statistical analysis**

Sample size was calculated using the formula for a descriptive study. When the estimated prevalence of metabolic syndrome (p) was 8% [18] and α = 0.05, a sample size of at least 127 cases was needed.

All data were analyzed by SPSS software (version 22.0). Data were presented as mean ± standard deviation (SD), number (%), or percentage (95% confidence interval – CI), as appropriate. Data comparisons were analyzed using the independent sample t test for continuous data and χ² for categorical data. Pearson’s correlation coefficient was determined for the correlation between WBC, total lymphocyte count and MetS components. Receiver operating characteristic (ROC) curve analysis for diagnosing MetS was performed to obtain area under ROC curve (AUC) and optimal cutoff points of WBC and total lymphocyte count for diagnosing MetS. An optimal cutoff point was defined as a point on a ROC curve nearest to the point where both sensitivity and specificity were one. A p value of < 0.05 was considered statistically significant.

**Results**

The characteristics of 140 participants are summarized in Table I. The overall mean age was 50.0 ± 7.4 years. Most participants were perimenopausal, married and multiparous. More than 60% of all participants had a healthy lifestyle; healthy foods, regular exercise, no smoking or alcohol-drinking habits (data not shown). The overall means of BMI and waist circumference were 24.0 ± 4.3 kg/m² and 82.1 ± 9.4 cm, respectively.

The overall prevalence of MetS diagnosed by JIS 2009 criteria was 21.4% (95% CI: 15.0-27.9). The prev-
Tab. I. Characteristics of 140 participants stratified by metabolic syndrome (MetS) status

|                         | MetS (n = 30) | Non-MetS (n = 110) | p     |
|-------------------------|--------------|--------------------|-------|
| Age (years)             | 52.0 ±8.0    | 49.5 ±7.2          | 0.092*|
| Menopausal status, n (%)|              |                    | 0.009*|
| Perimenopause           | 13 (43.3)    | 76 (69.1)          |       |
| Postmenopause           | 17 (56.7)    | 34 (30.9)          |       |
| Alcohol consumption, n (%)| 3 (10.0) | 11 (10)            | 1.000*|
| BMI (kg/m²)             | 27.7 ±4.0    | 23.0 ±3.8          | < 0.001*|
| Normal weight           | 3 (10.0)     | 65 (59.1)          | < 0.001*|
| Overweight              | 20 (66.7)    | 41 (37.3)          |       |
| Obese                   | 7 (23.3)     | 4 (3.6)            |       |
| Components of MetS      |              |                    |       |
| Waist circumference (cm)| 90.1 ± 8.6   | 79.9 ± 8.4         | < 0.001*|
| Triglycerides (mg/dl)   | 163.9 ± 89.6 | 83.1 ± 31.1        | < 0.001*|
| HDL-C (mg/dl)           | 51.3 ± 14.0  | 61.5 ± 12.9        | < 0.001*|
| Systolic BP (mmHg)      | 136.7 ± 10.0 | 121.7 ± 12.3       | < 0.001*|
| Diastolic BP (mmHg)     | 83.6 ± 8.1   | 75.6 ± 8.6         | < 0.001*|
| Fasting glucose (mg/dl) | 100.1 ± 7.5  | 93.1 ± 6.9         | < 0.001*|

Tab. II. Pearson’s correlation coefficients between white blood cell count, total lymphocyte count and components of metabolic syndrome

|                         | WBC Total lymphocyte count |
|-------------------------|-----------------------------|
| BMI                     | 0.290* 0.230*               |
| Waist circumference     | 0.276* 0.260*               |
| Triglycerides           | 0.202* 0.324*               |
| HDL-C                   | -0.215* -0.161*             |
| Systolic blood pressure | 0.053 0.059                 |
| Diastolic blood pressure| 0.085 0.160                 |
| Fasting glucose         | 0.126 0.070                 |

WBC – white blood cell count, BMI – body mass index, HDL-C – high-density lipoprotein cholesterol
*p < 0.05

The hematologic parameters from the complete blood count were compared between women with and without MetS as presented in Table I. Perimenopausal and postmenopausal women with MetS had a significantly higher level of mean white blood cell count (7,466.7 ±2,293.4 and 6,514.6 ±1,452.8 in MetS and non-MetS group, respectively, p = 0.006) and total lymphocyte count (2,572.0 ±686.4 and 2,207.7 ±557.7 in MetS and non-MetS group, respectively, p = 0.003). Other hematologic parameters did not differ between perimenopausal and postmenopausal with or without MetS.

There were weak correlations between white blood cell count, total lymphocyte count and components of MetS, which are summarized in Table II. Both white blood cell count and total lymphocyte count were correlated with BMI, waist circumference and triglycerides level while HDL-C showed a weak correlation with white blood cell count only.

The ROC curve of white blood cell count and total lymphocyte count for prediction of MetS is presented in Fig. 1. Albeit the predictive performances for both hematologic parameters were poor, we proposed the optimal cutoff of 6,750 cell/ml for white blood cell count and 2,232 cell/ml for total lymphocyte count (Table III).
Discussion

In the current study, overall prevalence of MetS was approximately 20.4%, which was higher than in a previous study in Thai women [23]. The prior study reported that prevalence of MetS was 15.9% in Thai perimenopausal and postmenopausal women attending a menopause clinic [23] and 11.7% in women attending a health checkup clinic [24]. The higher prevalence in our study might be due to that all our participants lived in the urban area, which predisposed them to lead a more sedentary lifestyle since they had higher BMI, waist circumference and alcohol consumption. The most common MetS components in our study were abdominal obesity, elevated blood pressure and elevated fasting glucose, which was also in agreement with previous studies about the most prevalent components of metabolic syndrome among postmenopausal women with MetS [9, 25, 26].

We found that perimenopausal and postmenopausal with MetS had a higher level of WBC and total lymphocyte count. Although all previous studies reported the same finding of higher levels of WBC and its subtype or being in a higher quartile of people with MetS [17-19, 24, 27-31], none of these studies focused on their association in perimenopausal and postmenopausal women. Only a WHI observational study [32] reported the level of WBC as a predictor of cardiovascular events and mortality rate in postmenopausal women. They reported that WBC count in an upper quartile was an independent predictor of coronary heart disease even if adjusted for multiple other risk factors including CRP level and total cholesterol/HDL-C ratio.

Although the mechanism of higher WBC and lymphocyte count in perimenopausal and postmenopausal women with MetS remains unclear, there are some possible explanations. The chronic inflammation appears to be a crucial mechanism for the pathophysiology of MetS [13]. During menopausal transition, the body composition changes including increased waist circumference [6, 12], fat mass, and visceral fat deposition [6, 11] contribute to reduction in circulating adiponectin [33], a collagen-like protein expressed in adipose tissue that is associated with many metabolic processes [34]. Low levels of adiponectin lead to an increase in levels of TNF-α and IL-6 from macrophages and a decrease in levels of the anti-inflammatory cytokines, IL-10 and IL-1 receptor antagonist, thus causing a chronic inflammatory state and insulin resistance. Low levels of adiponectin also increase gluconeogenesis by inhibiting adenosine monophosphate-activated protein kinase (AMPK) and causing hyperglycemia [35]. Overall, this proinflammatory state in MetS might explain the elevation of WBC count in MetS patients. Further studies about the relationship between WBC count and serum adiponectin are needed to confirm our hypothesis.

In our study, the correlation of WBC count and total lymphocyte count with components of MetS that significantly changed during menopausal transition (waist circumference, triglycerides and HDL-C) was congruent with previous cross-sectional and longitudinal studies [19, 24, 28, 30]. This could be also explained by the inflammatory state in these patients. The inflammation, increased triglycerides, and decreased HDL-C may stem from TNF-α and IL-6, which stimulate lipolysis and increase circulating free fatty acids to the liver. This increase in free fatty acids induces hepatic triglyceride synthesis and increases very low-density lipoprotein

Tab. III. Areas under receiver operating characteristic curve (AUCs) and optimal cutoff points (OCPs) of white blood cell count and total lymphocyte count for the prediction of metabolic syndrome

|                          | AUC (95% CI) | p    | OCP  | Sensitivity | Specificity |
|--------------------------|-------------|------|------|-------------|-------------|
| WBC (cell/ml)            | 0.640       | 0.019| 6.750| 0.633       | 0.591       |
|                          | (0.523-0.757)|      |      |             |             |
| Total lymphocyte count   | 0.643       | 0.016| 2.232| 0.667       | 0.518       |
| (cell/ml)                | (0.530-0.756)|      |      |             |             |

CI – confidence interval; WBC – white blood cell count
secretion from the liver, this increases hepatic triglyceride production and secretion, and thus hypertriglyceridemia [36]. Tumor necrosis factor α and IL-6 also suppress lipoprotein lipase synthesis in adipose tissue, which may contribute to hypertriglyceridemia and low HDL-cholesterol concentrations observed in individuals with visceral obesity [6, 37].

The current recommendation for the optimal cutoff point for WBC is varied according to the final diagnosis and population. We proposed an optimal cutoff point of WBC level at 6,750 cell/ml in Thai perimenopausal and postmenopausal women for prediction of MetS (sensitivity 63.3% and specificity 59.1%). A previous report from Japan recommended a cutoff point at 5,000 cell/ml for the prediction of MetS (sensitivity 65% and specificity 63%) in women who attended a general medical check-up program [30]. In a WHI observational study, they recommended the level of 6,700 cell/ml for a high risk of cardiovascular disease and mortality in postmenopausal women [32]. Currently, no recommendation regarding total lymphocyte count for the prediction of MetS has been proposed. We suggest the total lymphocyte count at least 2,232 cell/ml to further investigate for MetS.

Early diagnosis and prompt treatment of MetS can prevent the morbidity and mortality from its complications [6]. In our study, WBC and total lymphocyte counts are higher in perimenopausal and postmenopausal women with MetS but the prediction power is poor. More studies are required before applying its utilities into clinical practice. Further research about the association between the adiponectin level and WBC count is also recommended which will let us better understand the role of inflammation in MetS.

To the best of our knowledge, our study is the first to demonstrate the association between WBC count and MetS in perimenopausal and postmenopausal women. However, with a cross-sectional study it is impossible to determine the direction of the association. A longitudinal study would be more appropriate for this question.

Conclusions

Metabolic syndrome is common among Thai perimenopausal and postmenopausal women living in the urban area. WBC and total lymphocyte counts were higher in perimenopausal and postmenopausal women with MetS. However, both hematologic parameters were poor predictors for MetS in this group. Further longitudinal studies are necessary to confirm the relationship between WBC, lymphocyte count and MetS in perimenopausal and postmenopausal women.

Acknowledgement

This study was supported by the grant from the Faculty of Medicine Vajira Hospital, Navamindradhiraj University. The authors would like to thank all staff members of the Women Health Clinic, Department of Obstetrics and Gynecology, Faculty of Medicine Vajira Hospital, Navamindradhiraj University for facilitating the subject recruitment process.

Disclosure

Authors report no conflict of interest.

References

1. Isomaa B, Almgren P, Tuomilehto J, et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome. Diabetes Care 2001; 24: 683-689.
2. Chen J, Mauritner P, Hamm LL, et al. The metabolic syndrome and chronic kidney disease in U.S. adults. Ann Intern Med 2004; 140: 167-174.
3. Watanabe H, Tanabe N, Watanabe T, et al. Metabolic syndrome and risk of development of atrial fibrillation: the Niigata preventive medicine study. Circulation 2008; 117: 1255-1260.
4. Xu S, Wan Y, Xu M, et al. The association between obstructive sleep apnea and metabolic syndrome: a systematic review and meta-analysis. BMC Pulm Med 2015; 15: 105.
5. Kotonen A, Yki-Jarvinen H. Fatty liver: a novel component of the metabolic syndrome. Arterioscler Thromb Vasc Biol 2008; 28: 27-38.
6. Stachowiak G, Pertynski T, Pertynska-Marczewska M. Metabolic disorders in menopause. Prz Menopauzalny 2015; 14: 59-64.
7. Wang J, Ruotsalainen S, Moilanen L, et al. The metabolic syndrome predicts cardiovascular mortality: a 13-year follow-up study in elderly non-diabetic Finns. Eur Heart J 2007; 28: 857-864.
8. Eshghi R, Esteghamati A, Nakhjavani M. Menopause is an independent predictor of metabolic syndrome in Iranian women. Maturitas 2010; 65: 262-266.
9. Kim HM, Park J, Ryu SY, Kim J. The effect of menopause on the metabolic syndrome among Korean women: the Korean National Health and Nutrition Examination Survey. 2001. Diabetes Care 2007; 30: 701-706.
10. Muchanga Sifa MI, Lipira FB, Longo AL, et al. Prevalence and predictors of metabolic syndrome among Congolese pre- and postmenopausal women. Climacteric 2014; 17: 442-448.
11. Abdoulin R, Douet E, Brochu M, et al. The effect of the menopausal transition on body composition and cardiometabolic risk factors: a Montreal-Ottawa New Emerging Team group study. Menopause 2012; 19: 760-767.
12. Sowers M, Zheng H, Tomey K, et al. Changes in body composition in women over six years at midlife: ovarian and chronological aging. J Clin Endocrinol Metab 2007; 92: 895-901.
13. Welty FK, Alfaddagh A, Elajami TK. Targeting inflammation in metabolic syndrome. Transl Res 2015.
14. Donath MY, Shoelson SE. Type 2 diabetes as an inflammatory disease. Nat Rev Immunol 2011; 11: 98-107.
15. Zhang J, Gao Z, Yin J, Quon MJ, Ye J. S6K directly phosphorylates IRS-1 on Ser-270 to promote insulin resistance in response to TNF-(alpha) signaling through IKK2. J Biol Chem 2008; 283: 35375-35382.
16. Glass CK, Ofteisky JM. Inflammation and lipid signaling in the etiology of insulin resistance. Cell Metab 2012; 15: 635-645.
17. Phillips AC, Carroll D, Gale CR, et al. Lymphocyte sub-population cell counts are associated with the metabolic syndrome and its components in the Vietnam Experience Study. Atherosclerosis 2010; 213: 294-298.
18. Tao LX, Li X, Zhu H, et al. Association of hematological parameters with metabolic syndrome in Beijing adult population: a longitudinal study. Endocrinol 2014; 46: 485-495.
19. Babio N, Ibarrola-Jurado N, Bullo M, et al. White blood cell counts as risk markers of developing metabolic syndrome and its components in the PREDIMED study. PLoS One 2013; 8(3): e58354.

20. Harlow SD, Gass M, Hall JE, et al. Executive summary of the Stages of Reproductive Aging Workshop +10: addressing the unfinished agenda of staging reproductive aging. J Clin Endocrinol Metab 2012; 97: 1159-1168.

21. WHO, IASO, IOTF. The Asia-pacific perspective: redefining obesity and its treatment. Melbourne 2000.

22. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society, and International Association for the Study of Obesity Circulation 2009; 120: 1640-1645.

23. Indhavivadhana S, Rattanachaiyanont M, Wongvananurak T, et al. Predictors for metabolic syndrome in perimenopausal and postmenopausal Thai women. Climacteric 2011; 14: 58-65.

24. Loosonthern V, Dhanamun B, Williams MA. Prevalence of metabolic syndrome and its relationship to white blood cell count in a population of Thai men and women receiving routine health examinations. Am J Hypertens 2006; 19: 339-3645.

25. Jouyandeh Z, Nayebsadreh F, Qorbani M, Asadi M. Metabolic syndrome and menopause. J Diabetes Metab Disord 2013; 12: 1.

26. Marjani A, Moghasemi S. The Metabolic Syndrome among Postmenopausal Women in Gorgan. Int J Endocrinol 2012; 2012: 953627.

27. Jesri A, Okonofua EC, Egan BM. Platelet and white blood cell counts are elevated in patients with the metabolic syndrome. J Clin Hypertens (Greenwich) 2005; 7: 705-711.

28. Jung CH, Lee WY, Kim BY, et al. The risk of metabolic syndrome according to the white blood cell count in apparently healthy Korean adults. Yonsei Med J 2013; 54: 615-620.

29. Oda E. High-sensitivity C-reactive protein and white blood cell count equally predict development of the metabolic syndrome in a Japanese health screening population. Acta Diabetol 2013; 50: 633-638.

30. Oda E, Kawai R. Comparison between high-sensitivity C-reactive protein (hs-CRP) and white blood cell count (WBC) as an inflammatory component of metabolic syndrome in Japanese. Intern Med 2010; 49: 117-124.

31. Ford ES. The metabolic syndrome and C-reactive protein, fibrinogen, and leukocyte count: findings from the Third National Health and Nutrition Examination Survey Atherosclerosis 2003; 168: 351-358.

32. Margolis KL, Manson JE, Greenland P, et al. Leukocyte count as a predictor of cardiovascular events and mortality in postmenopausal women: the Women’s Health Initiative Observational Study. Arch Intern Med 2005; 165: 500-508.

33. Chu MC, Cosper P, Orio F, et al. Insulin resistance in postmenopausal women with metabolic syndrome and the measurements of adiponectin, leptin, resistin, and ghrelin. Am J Obstet Gynecol 2006; 194: 100-104.

34. Diez JJ, Iglesias P. The role of the novel adipocyte-derived protein adiponectin in human disease: an update. Mini Rev Med Chem 2010; 10: 856-869.

35. Ukkola O, Santaniemi M. Adiponectin: a link between excess adiposity and associated comorbidities? J Mol Med (Berl) 2002; 80: 696-702.

36. Feingold KR, Doerrler W, Dinarello CA, et al. Stimulation of lipolysis in cultured fat cells by tumor necrosis factor, interleukin-1, and the interferons is blocked by inhibition of prostaglandin synthesis. Endocrinology 1992; 130: 10-16.

37. Goumi I, Oka K, Etienne J, Chan L. Endotoxin-induced hypertriglyceridemia is mediated by suppression of lipoprotein lipase at a post-transcriptional level. J Lipid Res 1993; 34: 139-146.