Research Article

# Relationship between postprandial endotoxemia in nonobese postmenopausal women and diabetic nonobese postmenopausal women

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## Abstract

**Background:** We hypothesised that nonobese postmenopausal women (NoPoW) and diabetic NoPoW (DNoPoW) may be independently associated with postprandial endotoxemia. **Materials and Methods:** NoPoW and DNoPoW were evaluated for weight, eating habits, physical activity, body circumferences, fasting plasma glucose level, postprandial plasma glucose level, and insulin level. The lipopolysaccharide (LPS) levels and circulating LPS-binding protein (LBP) were determined in serum at fasting, 1 h, 2 h, 3 h, and 4 h after meal intake and their levels were co-related in 80 NoPoW and 80 DNoPoW. **Results:** Both DNoPoW group and NoPoW group showed a significant increase ($P < 0.05$) in LPS levels and circulating LBP in plasma after the meal intake, interestingly the increase was higher in the DNoPoW group. **Conclusions:** Elevated LPS and circulating LBP were associated significantly with DNoPoW group and NoPoW, especially after a meal intake. These findings suggested a role of LPS and LBP in postprandial systemic inflammation in DNoPoW group. Prospective studies are needed to confirm these results.

**Key words:** Endotoxemia, lipopolysaccharide, lipopolysaccharide-binding protein, postmenopause

## INTRODUCTION

Lipopolysaccharide (LPS) is an endotoxin (molecular weight >100,000 Daltons) and is composed of two major parts, the hydrophobic lipid A portion and the hydrophilic polysaccharide portion (commonly called the "O" region). Circulating endotoxin may derive from bacteria or gut microflora causing either overt acute infections or common chronic inflammatory conditions or cardiometabolic abnormalities including obesity, insulin resistance, and diabetes.[1] Endotoxin circulates in the plasma of healthy human subjects at low concentrations (between 1 and 200 pg/Ml).[2-5] However as the human gut is host to 100 trillion commensal organisms, which together contribute to an enteric reservoir of 1 g LPS, it is hypothesized that most of the circulating endotoxin may derive from the gut and that a small amount of commensally derived LPS may cotransit with dietary fat from the gut after a high-lipid meal, which thereby increases plasma endotoxin concentrations postprandially.

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The increased cardiovascular disease (CVD) risk after menopause seems to be associated with the emergence of the features of metabolic syndrome.[6,7] In fact, women with type 2 diabetes mellitus (T2DM), compared with age-matched nondiabetic women, exhibit several-fold higher rates of death-related to coronary artery disease, with event rates nearly identical to those observed in T2DM men.[8] Traditional cardiovascular risk factors cannot completely account for these sex differences in cardiovascular mortality.[9] Hence, more studies are needed to understand the precise influence of menopause in the risk for CVD, especially in diabetic patients, in order to achieve effective preventative and disease management

strategies to reduce the CVD risk particularly in postmenopausal women. Estrogen exerts cardioprotective action by maintaining a high level of high-density lipoprotein cholesterol and lowering the low-density lipoprotein cholesterol and triglycerides.[10-13] Loss of this protection after menopause may, therefore, be responsible for increased risk of developing CVD in postmenopausal women.[14-17] Increases in body weight are associated with greater risks of type 2 diabetes,[18-20] and weight gain in postmenopausal women are of special concern.[21] Estrogen has remarkable effects on body fat distribution, and the decreased estrogen production after menopause is associated with increased total body fat,[22,23] especially in the central/abdominal region.[24,25]

## MATERIALS AND METHODS

### Patients inclusion and exclusion criteria

This study was conducted in accordance with the ethical rules of the Helsinki Declaration. The study was approved by the Ethics Committee of the hospital, and all women gave written informed consent. Prior to the study, participants were informed that their confidentiality would be maintained, and consent was obtained. Eighty nonobese postmenopausal women (NoPoW) and 80 diabetic NoPoW (DNoPoW) were selected for the study. For the 80 NoPoW patients were excluded if they had CVD, arthritis, acute inflammatory disease, infectious disease, renal disease, were receiving treatment for hyperlipidemia or diabetes or were taking medications that could influence gastric emptying or the absorption time. For the 80 DNoPoW, all the above criteria except diabetes cases were excluded.

### Preparation of patients and sample collection

On the morning of the visit, blood pressure, weight, and height were measured, and compliance with dinner instructions was verified with a questionnaire. After that, each participant underwent a structured examination, which included an interview. Height, weight, waist circumference (WC) and hip measurements, a fasting venipuncture, and sequential determination of serum lipids were done. Height and weight were measured to the nearest 0.5 cm and 0.1 kg, respectively. Body mass index (BMI) was calculated as weight (kilogram) divided by height (in meter) squared. WC was determined to the nearest 0.1 cm using a measuring tape positioned at the midpoint between the lowest rib and the iliac crest and hips were measured at the largest gluteal circumference. These measurements were used to calculate the waist-to-hip ratio. Blood pressure was measured using a standard mercury sphygmomanometer. Blood samples were obtained from the antecubital vein and placed in vacutainer tubes. Postprandial blood samples were taken 1, 2, 3, and 4 h after the end of the study meal. Samples were centrifuged; serum was collected and stored at 20°C until analyzed. The diagnosis of DM was based on WHO criteria,[26] that is, a fasting plasma glucose level >7.0 mmol/L or >126 mg/dL, or a 2-h postprandial plasma glucose level >11.1 mmol/L or >200 mg/dL on more than one occasion, with symptoms of diabetes.

Serum LPS concentrations were measured by endotoxin assay, based on a Limulus amebocyte extract with a chromogenic Limulus amebocyte lysate (LAL) assay (QCL-1000, Lonza Group Ltd.). Samples were diluted in pyrogen-free water and heated at 70°C for 10 min to inactivate endotoxin-neutralizing agents that inhibit the activity of endotoxin in the LAL assay. Internal control of recovery calculation was included in the assessment. All samples were tested in duplicate. The endotoxin content was expressed as endotoxin units (EU)/mL. Exhaustive care was taken to avoid environmental endotoxin contamination and all material used for sample preparation, and the test was pyrogen-free. Plasma LPS-binding protein (LBP) levels were determined by a sandwich enzyme-linked immunosorbent assay Technology. Plasma samples were diluted at least 200 times and assayed according to the manufacturer's instructions. The assay has a sensitivity of 0.2 ng/ml. The intra-assay and inter assay coefficients of variation were <5 and <10%, respectively.

### Statistical analysis

All data are presented as mean ± standard deviation and were analyzed using GraphPad prism version 5 (San Diego, California, USA). Differences between two groups were analyzed by Student's *t*-test, while multiple groups were compared using ANOVA, followed by Bonferroni's multiple comparison tests. $P < 0.05$ was considered as statistically significant.

## RESULTS

The mean BMI values were 23.0 ± 1.4 kg/m$^2$ in NoPoW and 24.0 ± 2.1 kg/m$^2$ in DoPoW [Table 1]. The mean WCs

**Table 1: Patient characteristics**

| Characteristics | Nonobese postmenopausal women | Diabetic nonobese postmenopausal women |
|---|---|---|
| Age (years) | 48±5 | 48±6 |
| BMI (kg/m$^2$) | 23.0±1.4 | 24.0±2.1 |
| Waist circumference (cm) | 70.9±7.1 | 74±6.2 |
| Systolic blood pressure (mmHg) | 109.5±16.4 | 115.6±14.3 |
| Diastolic blood pressure (mmHg) | 71.9±11.8 | 78.9±4.9 |
| Fasting plasma glucose (mg/dL) | 91±11 | 164±37 |
| Postprandial plasma glucose (mg/dL) | 132±19 | 268±67 |

BMI: Body mass index

were 74 ± 6.2 in DoPoW and 70.9 ± 7.1 in NoPoW. The mean systolic blood pressure (mmHg) was 115.6 ± 14.3 in DoPoW and 109.5 ± 16.4 in NoPoW, whereas the diastolic blood pressure (mmHg) was 78.9 ± 4.9 in DoPoW and 71.9 ± 11.8 NoPoW Compared with NoPoW, DoPoW were more likely to have higher values for WC, blood pressure, and glucose. Fasting plasma glucose and postprandial plasma glucose were in the range of 91 ± 11 and 132 ± 19 in NoPoW and 164 ± 17 and 258 ± 27 in DoPoW.

The mean plasma endotoxin (LPS) in NoPoW in EU/mL was 0.37, 0.44, 0.64, 0.59 and 0.57 at fasting, 1, 2, 3, and 4 h versus 0.39, 0.5, 0.72, 0.73 and 0.66 in the DoPoW during the same duration. The mean LBP μg/ml was 10.9, 14.6, 19.9, 14 and 13.5 at fasting, 1, 2, 3, and 4 h in the NoPoW versus 11.6, 15.3, 23.8, 21.8 and 16.3 in the DoPoW during the same duration. Plasma endotoxin activity had a significant positive correlation with menopause, but the activity was higher in the DoPoW than in the NoPoW [Table 2].

## DISCUSSIONS

Metabolic diseases are associated with a low-grade inflammatory status. In our quest to determine a triggering factor of the early development of metabolic disease, we looked for a molecule involved early in the cascade of inflammation and identified LPS as a candidate. Furthermore, LPS stimulates release of several cytokines that are key inducers of insulin resistance. The concept of dietary excess is essentially linked to high-lipid intake induced inflammation.[27] LPS is a putative factor for the triggering of metabolic diseases.[28,29] The mechanisms are allowing enteric LPS absorption are unclear but could be related to increased filtration of plasma LPS into lymph with fat absorption.[26]

Bacterial endotoxin is considered as a potential inflammatory mediator of atherosclerosis[2,30-32] and has emerged as an independent predictor of atherosclerosis risk,[33] although the mechanisms for increased endotoxin in the plasma of some healthy individuals remain unknown. Chylomicrons promote intestinal absorption of LPS. More than 1 g of LPS can be found in the gut lumen.[34] Even small amounts of this highly proinflammatory substance could elicit strong inflammatory responses systemically, and hence, it is likely that the gut epithelium acts to effectively block the "translocation" of LPS and other microbial proinflammatory substances. However, small amounts of LPS are absorbed from the gut in healthy animals.[35] Excessive LPS absorption, however, could evidently be harmful and could lead to acute or chronic inflammation. Increased LPS absorption, for example, could exacerbate the risk for several chronic diseases, such as alcoholic liver injury.[36,37] Hence, dietary fat could increase LPS absorption in several ways. One-way would be through promotion of the paracellular uptake of macromolecules as a result of deleterious effects of fatty acids (FA) on tight-junction integrity. An alternative mechanism explaining FA dependent LPS absorption may involve internalization of LPS by the enterocyte, followed by association of some of the internalized LPS with chylomicrons and concomitant basolateral secretion of LPS with the chylomicrons or by association of independently transcytosed LPS with newly released chylomicrons. Chylomicrons are associated with metabolic endotoxemia by promoting LPS absorption.[38,39]

Lipoprotein lipase (LPL) activity was greater in diabetic postmenopausal women. The lower LPL activity in estrogen-sufficient women is consistent with other reports.[40,41] Our finding that the association between LPL activity and meal FA storage was present in premenopausal, but not in postmenopausal women, suggests that the upregulated LPL activity in postmenopausal women is no longer rate-limiting for dietary FA storage, at least at this amount of dietary fat intake. Although some have found that postmenopausal women treated with estrogen increase fat oxidation, others have not observed differences in fat oxidation between postmenopausal women who were estrogen-deficient versus estrogen-sufficient.[42,43] Our findings of reduced postprandial FA oxidation in postmenopausal women are consistent with the other previous reports.[44] Ovariectomized mice administered estrogen upregulate the skeletal muscle expression of peroxisome proliferator-activated receptor-δ and peroxisome proliferator-activated receptor (PPAR)-α,[45,46] which should increase fat oxidation. Hence future work must look at PPAR profiles in these patients. Latest evidence suggests, however that bacterial LPS derived from the gut microbiota may trigger inflammation and oxidative stress in response to diets.[47-51] High intake of

**Table 2: Plasma endotoxin and LBP profile**

| Parameters | Fasting | 1 h | 2 h | 3 h | 4 h | *P* |
|---|---|---|---|---|---|---|
| Plasma endotoxin (LPS) NoPoW (EU/mL) | 0.37±0.02 | 0.44±0.01 | 0.64±0.02 | 0.59±0.01 | 0.57±0.02 | *P*<0.05 |
| Plasma endotoxin (LPS) in DNoPoW (EU/mL) | 0.39±0.03 | 0.5±0.03 | 0.72±0.04 | 0.73±0.03 | 0.66±0.02 | *P*<0.05 |
| LBP in NoPoW (μg/mL) | 10.9±0.3 | 14.6±0.4 | 19.9±0.5 | 14±0.6 | 13.5±0.4 | *P*<0.05 |
| LBP in DNoPoW (μg/ml) | 11.6±0.6 | 15.3±0.6 | 23.8±0.6 | 21.8±0.8 | 16.3±0.5 | *P*<0.05 |

LPS: Lipopolysaccharide, LBP: Lipopolysaccharide-binding protein, NoPoW: Nonobese postmenopausal women, DNoPoW: Diabetic nonobese postmenopausal women

fat or carbohydrates does not promote only endotoxemia, but also production of LPS transporting proteins and receptors. This "metabolic endotoxemia" has been shown to initiate or promote obesity, insulin resistance, metabolic syndrome, and finally diabetes.[47-49,52]

## REFERENCES


1. Turnbaugh PJ, Ley RE, Mahowald MA, Magrini V, Mardis ER, Gordon JI. An obesity-associated gut microbiome with increased capacity for energy harvest. Nature 2006;444:1027-31.
2. Wiedermann CJ, Kiechl S, Dunzendorfer S, Schratzberger P, Egger G, Oberhollenzer F, *et al.* Association of endotoxemia with carotid atherosclerosis and cardiovascular disease: Prospective results from the Bruneck Study. J Am Coll Cardiol 1999;34:1975-81.
3. Goto T, Edén S, Nordenstam G, Sundh V, Svanborg-Edén C, Mattsby-Baltzer I. Endotoxin levels in sera of elderly individuals. Clin Diagn Lab Immunol 1994;1:684-8.
4. Hasday JD, Bascom R, Costa JJ, Fitzgerald T, Dubin W. Bacterial endotoxin is an active component of cigarette smoke. Chest 1999;115:829-35.
5. Bölke E, Jehle PM, Storck M, Nothnagel B, Stanescu A, Orth K. Endotoxin release and endotoxin neutralizing capacity during colonoscopy. Clin Chim Acta 2001;303:49-53.
6. Niebauer J, Volk HD, Kemp M, Dominguez M, Schumann RR, Rauchhaus M, *et al.* Endotoxin and immune activation in chronic heart failure: A prospective cohort study. Lancet 1999;353:1838-42.
7. Carr MC. The emergence of the metabolic syndrome with menopause. J Clin Endocrinol Metab 2003;88:2404-11.
8. Pan WH, Cedres LB, Liu K, Dyer A, Schoenberger JA, Shekelle RB, *et al.* Relationship of clinical diabetes and asymptomatic hyperglycemia to risk of coronary heart disease mortality in men and women. Am J Epidemiol 1986;123:504-16.
9. Pyörälä K, Laakso M, Uusitupa M. Diabetes and atherosclerosis: An epidemiologic view. Diabetes Metab Rev 1987;3:463-524.
10. Adashi EY. The climacteric ovary as a functional gonadotropin-driven androgen-producing gland. Fertil Steril 1994;62:20-7.
11. Barrett-Connor E, Bush TL. Estrogen and coronary heart disease in women. JAMA 1991;265:1861-7.
12. Grodstein F, Stampfer MJ, Manson JE, Colditz GA, Willett WC, Rosner B, *et al.* Postmenopausal estrogen and progestin use and the risk of cardiovascular disease. N Engl J Med 1996;335:453-61.
13. Wild RA, Taylor EL, Knehans A. The gynecologist and the prevention of cardiovascular disease. Am J Obstet Gynecol 1995;172:1-13.
14. Bush TL. The epidemiology of cardiovascular disease in postmenopausal women. Prevalence Med Part V Ann N Y Acad Sci, 1990;592:263-71.
15. Wasir JS, Misra A, Vikram NK, Pandey RM, Luthra K. C-reactive protein, obesity, and insulin resistance in post-menopausal women in urban slums of North India. Diabetes Metab Syndr Clin Res Rev 2007;1:83-9.
16. Maturana MA, Breda V, Lhullier F, Spritzer PM. Relationship between endogenous testosterone and cardiovascular risk in early postmenopausal women. Metabolism 2008;57:961-5.
17. Edmunds E, Lip GY. Cardiovascular risk in women: The cardiologist's perspective. QJM 2000;93:135-45.
18. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: Findings from a national cohort of US adults. Am J Epidemiol 1997;146:214-22.
19. Resnick HE, Valsania P, Halter JB, Lin X. Relation of weight gain and weight loss on subsequent diabetes risk in overweight adults. J Epidemiol Community Health 2000;54:596-602.
20. Colditz GA, Willett WC, Stampfer MJ, Manson JE, Hennekens CH, Arky RA, *et al.* Weight as a risk factor for clinical diabetes in women. Am J Epidemiol 1990;132:501-13.
21. Lemay A, Turcot L, Déchêne F, Dodin S, Forest JC. Hyperinsulinemia in nonobese women reporting a moderate weight gain at the beginning of menopause: A useful early measure of susceptibility to insulin resistance. Menopause 2010;17:321-5.
22. Guo SS, Zeller C, Chumlea WC, Siervogel RM. Aging, body composition, and lifestyle: The Fels Longitudinal Study. Am J Clin Nutr 1999;70:405-11.
23. Ley CJ, Lees B, Stevenson JC. Sex- and menopause-associated changes in body-fat distribution. Am J Clin Nutr 1992;55:950-4.
24. Lovejoy JC, Champagne CM, de Jonge L, Xie H, Smith SR. Increased visceral fat and decreased energy expenditure during the menopausal transition. Int J Obes (Lond) 2008;32:949-58.
25. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972;18:499-502.
26. American Diabetes Association. Diagnosis and classification of Diabetes Mellitus. Diabetes Care 2004;27:S5-10.
27. Mydel P, Takahashi Y, Yumoto H, Sztukowska M, Kubica M, Gibson FC 3rd, *et al.* Roles of the host oxidative immune response and bacterial antioxidant rubrerythrin during Porphyromonas gingivalis infection. PLoS Pathog 2006;2:e76.
28. Mitaka C. Clinical laboratory differentiation of infectious versus non-infectious systemic inflammatory response syndrome. Clin Chim Acta 2005;351:17-29.
29. Black DD, Tso P, Weidman S, Sabesin SM. Intestinal lipoproteins in the rat with D-(+)-galactosamine hepatitis. J Lipid Res 1983;24:977-92.
30. Stoll LL, Denning GM, Weintraub NL. Potential role of endotoxin as a proinflammatory mediator of atherosclerosis. Arterioscler Thromb Vasc Biol 2004;24:2227-36.
31. Ostos MA, Recalde D, Zakin MM, Scott-Algara D. Implication of natural killer T cells in atherosclerosis development during a LPS-induced chronic inflammation. FEBS Lett 2002;519:23-9.
32. Lehr HA, Sagban TA, Ihling C, Zähringer U, Hungerer KD, Blumrich M, *et al.* Immunopathogenesis of atherosclerosis: Endotoxin accelerates atherosclerosis in rabbits on hypercholesterolemic diet. Circulation 2001;104:914-20.
33. Berg RD. The indigenous gastrointestinal microflora. Trends Microbiol 1996;4:430-5.
34. Ravin HA, Rowley D, Jenkins C, Fine J. On the absorption of bacterial endotoxin from the gastro-intestinal tract of the normal and shocked animal. J Exp Med 1960;112:783-92.
35. Adachi Y, Moore LE, Bradford BU, Gao W, Thurman RG. Antibiotics prevent liver injury in rats following long-term exposure to ethanol. Gastroenterology 1995;108:218-24.
36. Caradonna L, Amati L, Magrone T, Pellegrino NM, Jirillo E, Caccavo D. Enteric bacteria, lipopolysaccharides and related cytokines in inflammatory bowel disease: Biological and clinical significance. J Endotoxin Res 2000;6:205-14.
37. Wellmann W, Fink PC, Benner F, Schmidt FW. Endotoxaemia in active Crohn's disease. Treatment with whole gut irrigation and 5-aminosalicylic acid. Gut 1986;27:814-20.
38. Kvietys PR, Specian RD, Grisham MB, Tso P. Jejunal mucosal injury and restitution: role of hydrolytic products of food digestion. Am J Physiol 1991;261:G384-91.
39. Ghoshal S, Witta J, Zhong J, de Villiers W, Eckhardt E. Chylomicrons promote intestinal absorption of lipopolysaccharides. J Lipid Res 2009;50:90-7.
40. Iverius PH, Brunzell JD. Relationship between lipoprotein lipase activity and plasma sex steroid level in obese women. J Clin Invest 1988;82:1106-12.
41. Price TM, O'Brien SN, Welter BH, George R, Anandjiwala J, Kilgore M. Estrogen regulation of adipose tissue lipoprotein lipase – possible mechanism of body fat distribution. Am J Obstet Gynecol 1998;178:101-7.
42. O'Sullivan AJ, Crampton LJ, Freund J, Ho KK. The route of estrogen replacement therapy confers divergent effects on substrate oxidation and body composition in postmenopausal women. J Clin Invest 1998;102:1035-40.
43. Jensen MD, Martin ML, Cryer PE, Roust LR. Effects of estrogen on free fatty acid metabolism in humans. Am J Physiol 1994;266:E914-20.

44. Lwin R, Darnell B, Oster R, Lawrence J, Foster J, Azziz R, *et al.* Effect of oral estrogen on substrate utilization in postmenopausal women. Fertil Steril 2008;90:1275-8.
45. D'Eon TM, Souza SC, Aronovitz M, Obin MS, Fried SK, Greenberg AS. Estrogen regulation of adiposity and fuel partitioning. Evidence of genomic and non-genomic regulation of lipogenic and oxidative pathways. J Biol Chem 2005;280:35983-91.
46. Muoio DM, MacLean PS, Lang DB, Li S, Houmard JA, Way JM, *et al.* Fatty acid homeostasis and induction of lipid regulatory genes in skeletal muscles of peroxisome proliferator-activated receptor (PPAR) alpha knock-out mice. Evidence for compensatory regulation by PPAR delta. J Biol Chem 2002;277:26089-97.
47. Cani PD, Amar J, Iglesias MA, Poggi M, Knauf C, Bastelica D, *et al.* Metabolic endotoxemia initiates obesity and insulin resistance. Diabetes 2007;56:1761-72.
48. Amar J, Burcelin R, Ruidavets JB, Cani PD, Fauvel J, Alessi MC, *et al.* Energy intake is associated with endotoxemia in apparently healthy men. Am J Clin Nutr 2008;87:1219-23.
49. Grunfeld C, Feingold KR. Endotoxin in the gut and chylomicrons: Translocation or transportation? J Lipid Res 2009;50:1-2.
50. Deopurkar R, Ghanim H, Friedman J, Abuaysheh S, Sia CL, Mohanty P, *et al.* Differential effects of cream, glucose, and orange juice on inflammation, endotoxin, and the expression of Toll-like receptor-4 and suppressor of cytokine signaling-3. Diabetes Care 2010;33:991-7.
51. Ghanim H, Sia CL, Upadhyay M, Korzeniewski K, Viswanathan P, Abuaysheh S, *et al.* Orange juice neutralizes the proinflammatory effect of a high-fat, high-carbohydrate meal and prevents endotoxin increase and Toll-like receptor expression. Am J Clin Nutr 2010;91:940-9.
52. Cani PD, Neyrinck AM, Fava F, Knauf C, Burcelin RG, Tuohy KM, *et al.* Selective increases of bifidobacteria in gut microflora improve high-fat-diet-induced diabetes in mice through a mechanism associated with endotoxaemia. Diabetologia 2007;50:2374-83.


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