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Leptin and adiponectin levels in middle-aged postmenopausal women: associations with lifestyle habits, hormones, and inflammatory markers—a cross-sectional study

Yves M. Rolland[a,c,*], Horace M. Perry III[a,b], Ping Patrick[a], William A. Banks[a,b], John E. Morley[a,b]

[a]*Geriatric Research, Education and Clinical Center, St Louis VA Medical Center, Saint Louis, MO 63104, USA*
[b]*Department of Medicine, Division of Geriatrics, St Louis University School of Medicine, Saint Louis, MO 63104, USA*
[c]*Inserm, U558, F-31073 Toulouse, France; Univ Toulouse III, Toulouse, F-31062 France; CHU Toulouse Hôpital Casselardit, Service de Médecine interne et gérontologie clinique, Toulouse, F-31059 France*

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Abstract

To investigate the relationships between blood levels of leptin or adiponectin and lifestyle habits, hormones, and inflammatory markers, we measured parameters of alcohol intake, smoking, physical activity, and blood levels of leptin, adiponectin, testosterone, estrone, estradiol, cortisol, dihydroepiandrostenedione, luteinizing hormone, thyroxin, C-reactive protein (CRP), and interleukin 6 and interleukin 2 receptor in 76 healthy middle-aged postmenopausal women. Anthropometric measures and body composition (evaluated by dual-energy x-ray absorptiometry) and lipid profiles were also assessed. By simple regression, leptin correlated positively with fat and lean masses, glucose, triglycerides, low-density lipoprotein cholesterol, and total cholesterol, and negatively with high-density lipoprotein cholesterol. Adioponectin correlated negatively with fat and lean masses and low-density lipoprotein cholesterol, and positively with high-density lipoprotein cholesterol. Leptin concentration was correlated inversely with adiponectin ($r = -0.26$, $P < .05$) and positively with CRP ($r = 0.56$, $P < .01$). Adiponectin concentration was negatively correlated with time since last alcoholic drink ($r = -0.24$, $P < .05$) and CRP ($r = -0.27$, $P < .05$) and positively with testosterone level ($r = 0.23$, $P < .05$). By multiple regression analysis, leptin concentration was predicted by age ($P < .05$), testosterone ($P < .05$), adiponectin ($P < .05$), CRP ($P < .01$), and interleukin 6 receptor ($P < .01$). Adiponectin concentration was predicted by the time since last alcoholic drink ($P < .05$), testosterone ($P < .05$), leptin ($P < .05$), and C-reactive protein ($P = .05$). Similar results were found when leptin or adiponectin concentration was adjusted for fat mass. These results suggested that levels of leptin and adiponectin in middle-aged postmenopausal women are partially determined by sexual hormones and inflammatory marker levels, and both predicted one another. Moreover, adiponectin level may be modulated by alcohol intake.

1. Introduction

Leptin and adiponectin are both adipokines secreted from fat, and their blood levels correlate with adiposity and biological markers of adiposity. Increased body mass index (BMI) is associated with a decreased plasma adiponectin concentration [1] and increased leptin concentration [2]. However, fat mass (FM) is an insufficient parameter to explain the wide range of adiponectin and leptin levels reported. Adipokine secretions are also regulated by a variety of other factors such as lifestyle habits, hormones, and inflammatory markers. Most of the factors and mechanisms affecting adiponectin and leptin levels are poorly described or a matter of debate. Moreover, most of the literature addressing this issue concerns obese population. Factors affecting adiponectin and leptin levels in healthy subject within a normal range of weight have received less attention.

A better understanding of factors affecting leptin or adiponectin levels could have clinical significance [3]. Actually, adiponectin has been shown to improve insulin secretion, insulin sensitivity, and fatty acid oxidation; to have anti-inflammatory action; and to prevent atherosclerosis [4].

* Corresponding author. Geriatric Research, Education and Clinical Center, St Louis VA Medical Center, Saint Louis, MO, USA. Tel.: +1 314 894 6510; fax: +1 314 894 6614.
E-mail address: yvesmrolland@yahoo.fr (Y.M. Rolland).

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On the other hand, leptin may be a factor contributing to obesity and its associated morbidities in humans [5].

Because lifestyle habits are modifiable and profoundly affect health, their effect on adipokine secretion in healthy nonobese subjects is an important issue. They may modulate the cardiovascular risk factors, at least in part, through their action on adipokine levels and independently of FM. Moderate alcoholic intake has been shown to increase leptin [6] and adiponectin concentrations [7,8]. Smoking may reduce leptin concentration [9], and there are conflicting data whether physical activity has any effect on leptin [10] or adiponectin level [11].

Numerous other hormones have been studied in relation to leptin and adiponectin. Leptin appears to interact with glucagon, the insulin-like growth factors, growth hormone, and glucocorticoids [5]. Testosterone is implicated in the regulation of leptin [12] and adiponectin levels [13,14]. Adiponectin gene expression is reduced by glucocorticoids [15]. A possible relationship between thyroid hormones and leptin [16] or adiponectin [17] has also been suggested. In addition, there is a growing list of adipokines involved in inflammation. Physiologic relationship may exist between leptin and adiponectin levels [18] and these 2 adipokines may be cross-regulated. Adiponectin exhibits anti-inflammatory properties [19], and various cytokines increase leptin concentration in human subjects [20].

The aim of this study was to investigate the relationships between leptin or adiponectin and lifestyle habits, hormones, and inflammatory markers in healthy middle-aged postmenopausal women.

2. Research design and methods

2.1. Subjects

Seventy-six white women with a mean age of 54.1 ± 0.5 years (range, 45-62 years) volunteered to participate in this study. Participants were recruited by advertisement. To be included, women had to be postmenopausal (at least 6 months since their last menses), to have no hormone replacement therapy, no diabetes, no treatment of lipid or thyroid disorder, no steroid treatment, and no infection in the previous 2 months. Mean length since last menses was 41.5 ± 27 months (range, 6-99 months).

This study was approved by the institutional review boards of the St Louis VA Medical Center (St Louis, MO). Each participant provided written informed consent.

2.2. Methods

Participants were screened by medical history questionnaire, physical examination, and fasting blood profiles.

Lifestyle habits assessment was performed with standardized scales and questionnaires. Physical activity levels were obtained by standardized validated measurements [21]. History of smoking was obtained by direct inquiry. Alcohol intake was quantified by self-report in a structured interview using a questionnaire for both the previous week and previous 3 months as previously reported [22]. Time since last alcoholic drink was recorded on a scale from 1 (today) to 6 (>10 days ago). Lifetime alcohol consumption was estimated using a questionnaire (Lifetime Drinking History) [22].

Anthropometric measurements were performed in a research laboratory. Body mass index was defined by the weight/height^2 ratio. Hip circumference was determined at the level of the maximum posterior protrusion of the buttocks. Waist circumference was measured 1 cm above the iliac crests. The waist-to-hip ratio was then calculated. The mid-upper arm circumference (MAC) was measured with the left arm at a right angle to the forearm. The triceps fold thickness was measured on left arm with a calibrated Holtain skinfold calipers (Holtain Ltd, Crymych, Dyfed, UK) to the nearest 0.2 mm.

2.2.1. Direct body composition measurement

Lean mass (LM) and FM were evaluated by dual-energy x-ray absorptiometry (Hologic QDR 4.500 W; Hologic, Waltham, MA). The coefficient of variation was 1.9% for total body FM. Trunk FM and trunk LM were recorded. Correlated LM and correlated FM were defined by the LM/height^2 ratio or FM/height^2 ratio, respectively. Percentage of LM and FM corresponded to (LM/weight) × 100 and (FM/weight) × 100, respectively.

2.2.2. Serum studies

Women were scheduled for blood sampling in the morning and instructed to fast after midnight the night before the evaluation. Hormonal and inflammatory marker measurements were performed by the clinical geriatrics laboratory.

Leptin and adiponectin measurements were determined using a commercially available radioimmunoassay (RIA) kit (Linco Research, St Charles, MO). In our laboratory, leptin and adiponectin have an intra-assay coefficient of variation of 4.7% and 5.3% and an inter-assay coefficient of variation of 5% and 8.1%, respectively.

Serum estrone and serum estradiol were measured using an RIA kit (ICN-Biomedicals, Costa Mesa, CA, and Diagnostic Systems Laboratories, Santa Monica, CA, respectively). The intra- and interassay coefficients of variation were 7.2% and 11.1%, respectively, for serum estrone and 6.5% and 9.7%, respectively, for serum estradiol. Luteinizing hormone (LH) was measured using an RIA (ICN-Biomedicals). The intra- and interassay coefficients of variation were 7.3%, and 8.6%, respectively. Serum cortisol was measured using an RIA kit (Diagnostic Products, Los Angeles, CA). The intra- and interassay coefficients of variation were 3.5% and 4.0%, respectively. Serum dihydroepiandrostenedione sulfate (DHEA) was measured using an RAI (Diagnostic Products). The intra- and interassay coefficients of variation were 5.3% and 7.0%, respectively. Testosterone was measured using an RIA kit (ICN-Biomedicals). The intra- and interassay coefficients of variation were 6.7% and 7.3%, respectively. Soluble

interleukin 6 (IL-6) receptor assay and soluble interleukin 2 (IL-2) receptor assay were performed using a commercially available enzyme-linked immunosorbent assay kit (INC-Biomedicals and Endogen, Woburn, MA, respectively). These kits have an intra- and interassay coefficients of variation of 5.9% and 5.0%, respectively, for IL-6 receptor and 9.8% and 9.6%, respectively, for IL-2 receptor.

Other routine serum studies including glucose, thyroxin, total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and C-reactive protein (CRP) were performed in a commercial clinical laboratory (Smith Kline-Beecham, St Louis, MO).

2.3. Statistical analysis

Study population characteristics and anthropometric and laboratory measurements were presented as proportions or median values with their SD and range. Adiponectin and leptin concentrations presented a normal distribution. Pearson correlation coefficients were presented to show the associations between adiponectin and leptin and anthropometric measurements, lifestyle habits, hormones, and inflammatory markers. Linear regression analysis was applied to explore the relation between adiponectin and leptin and potential variables. Linear regression analysis provides insight into linear associations and allows adjustment for the influence of potential confounders. To avoid the major impact of body fat and their self-confounding effects, we did not take into account anthropometric

Table 1
Physical characteristics, anthropometric and body composition measures, lifestyle habits, lipid profiles, hormones, and inflammatory marker concentrations (mean, SD, range) of the 76 healthy middle-aged postmenopausal women

	Mean (SD)	Range
Age (y)	54.14 (4.24)	45.08-61.92
Anthropometric measurements		
Weight (kg)	65.07 (8.40)	47.72-81.59
BMI (kg/m^2)	24.56 (3.34)	18.09-31.36
WHR	0.81 (0.06)	0.66-0.96
MAC (cm)	30 (3)	23-36
Triceps (cm)	31.6 (7)	13-50
Direct body composition measures		
LM (kg)	36.23 (3.45)	29.51-43.59
FM (kg)	21.37 (5.47)	9.87- 32.99
Lifestyle habits		
Physical activity[a] (range, 1-34)	18.88 (5.72)	6-34
Alcohol		
Time since last drink (1- 6)	3.49 (0.16)	1-6
Previous week (average of mL/wk)	89 (164)	0-1181
Previous 3 mo (average of mL/wk)	91 (98)	0-603
Lifetime drinking history (average of mL/mo)	242 (236)	2-1471
Serum lipid profiles		
Triglycerides (mmol/L)	0.241 (0.135)	0.087-0.982
Total cholesterol (mmol/L)	5.41 (0.92)	3.64-7.60
HDL-C (mmol/L)	1.65 (0.37)	0.95-2.74
LDL-C (mmol/L)	3.27 (0.78)	2.03-5.27
Hormones		
Leptin (ng/mL)	17.71 (11.63)	2-54
Adiponectin (μg/mL)	15.42 (6.21)	5-30.5
Estrone (pmol/L)	125.73 (98.97)	11.01-715.84
Estradiol (pmol/L)	63.03 (51.12)	18.49-262.62
LH (ng/mL)	35.75 (13.28)	5-63
Cortisol (mmol/L)	33.63 (12.60)	11.03-63.45
DHEA (mmol/L)	305.13 (159.70)	76.27-839.01
Testosterone (pmol/L)	48.32 (33.62)	3.46-169.88
Thyroxine (nmol/L)	88.41 (17.50)	60.48-137.70
Inflammatory markers		
CRP (μg/mL)	2.23 (2.91)	0.1-20.2
IL-6 receptor (μg/mL)	82.76 (49.91)	20-274
IL-2 receptor (μg/mL)	371.53 (153.18)	122-774

WHR indicates waist-to-hip ratio.
[a] Higher score indicates higher physical activity.

Table 2
Unadjusted Pearson correlation coefficients between leptin or adiponectin and physical characteristics, anthropometric and body composition measures, lifestyle habits, lipid profiles, hormones, and inflammatory marker concentrations

	Leptin		Adiponectin	
	n = 76	*P*	n = 76	*P*
Age	0.23	.24	−0.006	.08
Creatinine	0.18	.33	−0.16	.12
Glucose	0.35	.03	−0.14	.42
Lifestyle habits				
Physical Activity	−0.11	.11	−0.17	.13
Alcohol (last week)	−0.14	.22	0.17	.21
Time since last alcoholic drink	0.07	.56	−0.24	.04
Anthropometric measurements				
BMI	0.73	<.001	−0.31	.01
WHR	0.25	.03	−0.20	.09
MAC	0.66	<.01	−0.44	<.001
Triceps	0.64	<.001	−0.24	.10
Direct body composition measures				
Fat				
FM	0.78	<.01	−0.31	.01
CorFM	0.70	<.01	−0.29	.02
%FM	0.69	<.01	−0.26	.03
Trunk FM	0.73	<.01	−0.30	.02
Lean				
LM	0.42	.02	−0.28	.04
CorLM	0.23	.30	−0.22	.13
%LM	0.41	.02	−0.25	.08
Trunk LM	0.42	.05	−0.28	.04
Serum lipid profiles				
Triglycerides	0.36	.03	−0.14	.15
Total cholesterol	0.31	.04	−0.09	.49
HDL-C	−0.24	<.001	0.47	<.001
LDL-C	0.33	<.01	−0.26	.02
Hormones				
Adiponectin	−0.26	.02	–	–
Leptin	–	–	−0.26	.02
Estrone	0.12	.76	0.15	.92
Estradiol	0.08	.77	0.02	.28
Cortisol	−0.15	.81	−0.03	.82
DHEA	0.01	.88	0.22	.30
LH	0.05	.77	0.03	.77
Testosterone	0.22	.11	0.23	.03
Thyroxin	−0.12	.49	0.13	.86
Inflammatory markers				
CRP	0.56	<.01	−0.27	.02
IL-6 receptor	0.22	.24	0.13	.67
IL-2 receptor	−0.01	.86	−0.08	.92

CorFM indicates correlated FM; CorLM, correlated LM.

measures and biological markers of adiposity (eg, lipid profiles) for adjustment in the first initial models. Finally, similar models were performed with leptin adjusted for FM or adiponectin adjusted for FM. This approach was applied because leptin is secreted by the adipocytes in direct proportion to adipose tissue mass and adiponectin is exclusively secreted by the adipocytes. For these reasons, we expressed the leptin adjusted for FM index (leptin/FM) and the adiponectin adjusted for FM index (adiponectin/FM) as potential indicators of the secretory activities of fat tissue for each adipokine.

Tests were 2-sided, and P values less than .05 were considered significant. Data analysis was performed using a commercially available statistical analysis program, Statistica (StatSoft, Oklahoma City, OK).

3. Results

3.1. Demographic characteristics

Age, physical characteristics, lipid profiles, lifestyle habits, and blood levels of hormones and inflammatory markers for the whole group are presented in Table 1. According to the Adult Treatment Panel III definition [23], no woman had the metabolic syndrome. Fifty participants (65.8%) previously smoked, but none were present smokers. Mean leptin and adiponectin concentrations were 17.71 ± 11.63 ng/mL and 15.42 ± 6.21 μg/mL, respectively, and both had a wide range of distribution (range, 2-54 ng/mL and 5-30 μg/mL, respectively). Correlations between leptin levels, adiponectin levels, and the other variables are presented in Table 2.

3.2. Leptin

In the simple analysis, leptin was not significantly correlated with age. Leptin correlated positively and significantly with all of the direct and indirect anthropometric measures. None of the anthropometric measures or derived FM measures (correlated FM, percentage of FM, trunk FM) had an association consistently superior than FM ($r = 0.78$, $P < .001$).

Leptin was negatively and significantly correlated with HDL-C ($r = -0.24$, $P < .001$), whereas it was positively and significantly correlated with glucose level ($r = 0.35$, $P < .05$), total cholesterol ($r = 0.31$, $P < .05$), LDL-C ($r = 0.33$, $P < .01$), and triglycerides ($r = 0.36$, $P < .05$).

None of the lifestyle habits (physical activity and alcohol) were significantly correlated with leptin concentration, and none of the hormones tested in this study except adiponectin were found significantly associated with leptin concentration ($r = -0.26$, $P < .05$). Among the inflammatory markers, CRP was positively and significantly associated with leptin ($r = 0.56$, $P < .01$).

In the multiple regressions analysis, when anthropometric and lipid measures were not included, statistically significant independent predictors of leptin for the final model included age, adiponectin, testosterone, CRP, and IL-6 receptor. Significant predictors of leptin adjusted for FM were the same except for age (Table 3).

3.3. Adiponectin

In the simple analysis, adiponectin was not significantly correlated with age. Adiponectin was correlated negatively and significantly with BMI, MAC, and FM (and its derived measures). Negative correlation between adiponectin and LM was significant ($r = -0.28$, $P = .046$). Among the anthropometric measures, the highest correlation coefficient was observed for MAC with adiponectin ($r = -0.44$, $P < .001$). Adiponectin was highly and positively correlated with HDL-C ($r = 0.47$, $P < .001$), whereas it was negatively correlated with LDL-C ($r = -0.26$, $P < .05$).

Time since last alcoholic drink was the only lifestyle parameter significantly correlated with adiponectin concentration ($r = -0.24$, $P < .05$).

Adiponectin levels were positively and significantly correlated with testosterone levels ($r = 0.23$, $P < .05$) and leptin. None of the other hormones tested in this study were found to be significantly associated with adiponectin concentration.

Among the inflammatory markers, CRP was negatively associated with adiponectin and was the only inflammatory marker significantly associated with adiponectin ($r = -0.27$, $P < .05$).

In the multiple regressions analysis, when anthropometric and lipid measures were not included, statistically significant independent predictors of adiponectin for the

Table 3
Results from stepwise multivariate linear regression analyses with serum leptin as dependent variable and potential predictors as independent variables[a]

	Predictor of leptin[b] (n = 75; $R = 0.61$; $R^2 = 0.38$; $P < .0001$)			Predictor of leptin adjusted for FM[c] (n = 73; $R = 0.56$; $R^2 = 0.32$; $P < .0001$)		
	β	SE	P	β	SE	P
Age	.22	0.09	.02	–	–	–
Adiponectin	−.23	0.10	.02	−.22	0.10	.03
Testosterone	.21	0.09	.03	.22	0.10	.03
CRP	.30	0.10	.003	.36	0.10	.0007
IL-6 receptor	.29	0.09	.002	.28	0.10	.006

[a] Potential predictors are age, alcohol intake, hormones, and inflammatory markers.
[b] Anthropometric measures and lipid profiles were not taken into account for adjustment in the model.
[c] Lipids profiles were not taken into account for adjustment in the model.

Table 4
Results from stepwise multivariate linear regression analyses with serum adiponectin as dependent variable and potential predictors as independent variables[a]

	Predictor of adiponectin[b] (n = 74; R = 0.45; R^2 = 0.18; P < .001)			Predictor of adiponectin adjusted for FM[c] (n = 73; R = 0.38; R^2 = 0.33; P < .0001)		
	β	SE	P	β	SE	P
Time since last alcoholic drink	−.24	0.10	.021	−.20	0.09	.04
Leptin	−.25	0.12	.04	−.54	0.10	.0001
Testosterone	.24	0.10	.02	–	–	–
DHEA	–	–	–	.20	0.09	.04
CRP	−.23	0.11	.05	−.12	0.09	.21
IL-6 receptor	.21	0.11	.06	.27	0.10	.008

[a] Potential predictors are age, alcohol intake, hormones, and inflammatory markers.
[b] Anthropometric measures and lipid profiles were not taken into account for adjustment in the model.
[c] Lipids profiles were not taken into account for adjustment in the model.

final model included time since last alcoholic drink, leptin, and testosterone. Significant predictors of adiponectin adjusted for FM included time since last alcoholic drink, leptin, DHEA, and IL-6 receptor (Table 4). C-reactive protein was close to significant (P = .05).

4. Discussion

In this cross-sectional study of healthy middle-aged postmenopausal women on lifestyle habits, hormones, and inflammatory markers, we found that leptin and adiponectin concentrations were predicted by testosterone and inflammatory markers and both cross-predicted one another. Moreover, this study suggested that adiponectin level may also be modulated by a recent alcohol intake.

This study suggested that in a healthy postmenopausal population, leptin level was partially predicted by testosterone concentration. Leptin has been shown to influence the hypothalamopituitary-gonadal function and to stimulate the LH-releasing hormone as well as LH and follicle-stimulating hormone secretion [24]. The role of leptin during amenorrhea of girls affected by anorexia nervosa [25] or athletic women [26] or low testosterone levels in anorexic boys [27] as a key determinant of gonadal function has been demonstrated by several studies. In young women, the leptin level is also a prerequisite for menstruation and reproductive function [28].

The positive association between leptin and testosterone levels is of interest as it also supports the hypothesis of a possible regulatory function of leptin on androgen secretion in postmenopausal women. The role of serum androgens during menopause still gives rise to controversy [29]. However, it has been repeatedly shown that androgens inhibit leptin production in adipose tissue [12] and may reduce the degree of central obesity [30]. In postmenopausal women, the moderate decrease of testosterone may cause an increase of FM and a central redistribution. On the other hand, recent reports suggest that adipose tissue and especially visceral adiposity [31] is also an important site for secretion of sex steroids [32]. During menopause, lowering the primary source of androgen from the gonad and the adrenal gland may be attenuated by the contribution of an increased visceral adiposity and an increased leptin level.

A positive significant association was found between adiponectin and testosterone. These results are not consistent with previous studies. Indeed, testosterone has been shown to inhibit adiponectin concentration [6,13] and may specifically inhibit the secretion of its high-molecular-weight form [18]. However, those interventional studies were performed in men and evaluated the effects of testosterone replacement over a period too short to result in body composition changes. The decreased adiposity caused by testosterone might increase adiponectin levels in the long-term and might offset the immediate decreased secretion of adiponectin. In one previous cross-sectional study in postmenopausal women, testosterone was not found to correlate with the adiponectin level [33]. Our results indicate that the long-term effects of testosterone on adiponectin levels in postmenopausal women deserve further investigation.

In our healthy postmenopausal women, CRP and IL-6 receptor were positively and significantly associated with leptin, and CRP was negatively associated with adiponectin. Increasing evidence suggests that the inflammatory state may be causal in the development of the disorders associated with the metabolic syndrome. Production of cytokines by adipose tissue is increased in FM and correlated with increased leptin levels and decreased adiponectin levels. Adiponectin has also been reported to exhibit a cytokine inhibitory function [34] and leptin to act as a pro-inflammatory cytokine [35]. Furthermore, oxidative stress in adipose tissue has been reported to decrease production of adiponectin [34], and leptin synthesis increases in response to sepsis and secretion of inflammatory mediators [35]. Our results are consistent with an interaction between leptin or adiponectin and inflammatory markers. Our results also suggested that even in healthy subjects, both adiponectin and leptin are inflammatory modulators, but also that an inflammatory process leading to the metabolic syndrome may start during this period.

We found a significant negative association between plasma adiponectin concentrations and the time since last alcoholic drink. This result suggested that the more recently

alcohol has been drunk, the higher the level of adiponectin. These findings support the notion that alcohol consumption increases adiponectin concentration as demonstrated in several recent studies [7,8,36,37]. Although previous work suggested that adiponectin concentration reflects long-term, moderate alcohol intake, our results suggest that an immediate dietary change is involved. Epidemiologic and experimental studies suggest that moderate alcohol intake reduces the risk of ischemic heart disease and type 2 diabetes mellitus [38]. This protective effect has not yet been elucidated; however, improvement of the lipid profiles cannot explain all of its protective effect. Adiponectin is an adipokine known to improve insulin sensitivity and exhibit antiatherogenic effect, which may partially explain the preventive effect of alcohol [39].

Like others [40], we found that leptin was a negative predictor of adiponectin levels independent of FM. Based on animal studies [14], a physiologic relationship may exist between leptin and adiponectin levels, but cross-sectional studies in humans reported discrepant results [33,41]. Our results suggest that leptin and adiponectin negatively determined each other, but interventional studies are necessary to confirm this hypothesis.

This study has several limitations. First, the selection of a healthy white population is not representative of the general population of the same age. However, our results provide background information on these poorly understood adipokines aside from pathologic situation. Understanding the mechanisms associated with leptin and adiponectin level is relevant in young postmenopausal women because this transition period is associated with an accelerated change in body composition [42]. Second, because of the cross-sectional nature of the study, the causal nature of associations between adiponectin or leptin and the different variables could not be addressed. Third, we had no data for men. Influence of sex hormones on adipokine levels seems important and our results cannot be generalized to men. Another, theoretical, limitation of our analyses is that adiponectin and leptin concentrations were both adjusted for total FM in the multiple models. This approach is critical. Actually, fat tissue distribution and the related sizes of the adipocytes have previously been mentioned as other major determinants of leptin and adiponectin levels. However, in the simple analysis, FM was the strongest direct anthropometric variable correlated with leptin and adiponectin. Fourth, adiponectin and leptin may cross-talk with many other hormones (such as insulin) not tested in our study or be modulated by other lifestyle habits like nutrients intake profile. Finally, a large series of correlations was performed, and some may be statistically significant by chance alone.

In summary, this cross-sectional study in middle-aged postmenopausal women suggested that leptin and adiponectin concentrations are partially determined by testosterone and inflammatory markers levels and are cross-determined. Moreover, adiponectin levels may also be modulated by a recent alcohol intake.

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