Original Article

Visceral Adiposity Index and Lipid Accumulation Product as a Predictors of Insulin Resistance in Women with Polycystic Ovary Syndrome in Karbala Holly

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Abstract

Background: Insulin resistance (IR) and hyperandrogenism are the two main characteristics of polycystic ovarian syndrome (PCOS), in addition to central obesity and dyslipidemia. The visceral adiposity index (VAI) and the lipid accumulation product (LAP) are markers that include anthropometric parameters and blood lipids, and they have shown accuracy in detecting visceral obesity and predicting IR. **Objectives:** This study aims to determine the association between body mass index (BMI) and PCOS characteristics as well as to identify the most effective markers that can predict IR in PCOS in Karbala city. **Materials and Methods:** This study is a case-control study and included 170 women with ages ranging between 18 and 40 years. We divided PCOS patients and healthy control into groups depending on their BMI: less than 25 kg/m^2 and more than 25 kg/m^2. Each person underwent a medical examination, ultrasonogram, hormones (serum luteinizing hormone, follicle stimulation hormone, prolactin, and free testosterone), metabolic parameters (lipids profile, insulin resistance), and anthropometric parameters were assessed. **Results:** Group 2 PCOS patients (BMI ≥25 kg/m^2) had significantly higher levels of insulin resistance and the luteinizing hormone/follicle-stimulating hormone ratio compared to group 1 PCOS patients (their BMI ≤ 25 kg/m^2) *P* value 0.001. The LAP and VAI were significantly different in PCOS2 compared to PCOS1, the sensitivity of LAP and VAI to predictors IR in groups 2PCOS was 71% and 81%, respectively. The beck anxiety inventory was used to calculate anxiety, resulting in PCOS2 having a high mean than PCOS1. **Conclusion:** We can conclude that VAI and LAP can help to early detect IR in PCOS patients with a BMI of more than 25 kg/m^2.

Keywords: Hormonal imbalance, insulin resistance, lipid accumulation product (LAP), obesity, polycystic ovarian syndrome (PCOS), visceral adiposity index (VAI)

Introduction

Polycystic ovary syndrome is a prevalent condition that affects women of reproductive age and is characterized by reproductive, hyperandrogenic, and dysmetabolic characteristics. The pathophysiology is still vague and can result in severe complications such as cardiovascular diseases[1] and type 2 diabetes mellitus.[2] The World Health Organization estimates that more than 116 million women (3.4%) worldwide are impacted by this syndrome. The prevalence of polycystic ovarian syndrome (PCOS) varies notably by geographic location,[3] and its prevalence is approximately 6%-10% of women of reproductive age.[4] A diagnosis is made based on the internationally agreed "Rotterdam" diagnostic criteria for PCOS.[5] The syndrome is associated with features of central obesity,[6] dyslipidemia,[7] mood disorders,[8] anxiety,[9] a higher chance of developing obstructive sleep apnea,[10] and acne.[11]

Access this article online

Quick Response Code:

Website: https://journals.lww.com/mjby

DOI: 10.4103/MJBL.MJBL_907_23

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Submission: 05-Jul-2023 **Accepted:** 11-Sep-2023 **Published:** 30-Apr-2026

How to cite this article: Ali ZA, Odda AH, Al-Kazaz FFM, Jafar NN, Al-Alaq MM, *et al*. Visceral adiposity index and lipid accumulation product as a predictors of insulin resistance in women with polycystic ovary syndrome in Karbala holly. Med J Babylon 2026;23:206-12.

Hyperinsulinemia activates excessive ovarian androgen production,[12] this condition is associated with elevated luteinizing hormone pulses have been reported to indicate hyperactivity of the hypothalamic-pituitary-gonadal axis due to impaired negative feedback of gonadotropins, resulting in decreased aromatase activity in granulosa cells, consequently non-convert testosterone to estrogen, as well as low progesterone hormone.[13] Additionally, it's important to note Prolactin (PRL) is a polypeptide hormone that has a variety of physiological roles. It is released by the pituitary gland.[14] Lipid profiles are closely linked to the level of steroid hormones. The fact that cholesterol (CHO) is a precursor to steroid hormones highlights their important role in hormonal disorders and PCOS development[15]; around 70% of people with PCOS have dyslipidemia.[16]

Obesity has a marked impact on the metabolic complications of PCOS.[17] According to Shi *et al.*[18] study, obese individuals with PCOS have more glucose and lipid metabolism abnormalities than PCOS patients with a normal body mass index (BMI), whereas hyperinsulinemia affected PCOS patients with normal BMI as well as obesity. The term "central obesity" describes abdomen fat hidden between the internal organs. Because of central obesity, insulin sensitivity increases, which results in insulin resistance (IR) which promotes the development of hyperandrogenism.[19] The lipid accumulation product (LAP) and the visceral adiposity index (VAI) are calculated to evaluate visceral obesity more easily and high precision.[20,21]

Aims of the Study

1. Determine the association between body mass index and PCOS characteristics.
2. Identify the most effective markers that can predict IR in women with PCOS in Karbala city.

Materials and Methods

The case-control study included 170 women with ages ranging between 18 and 40 years that has a mean age of 24.37±4.7 standard deviation (SD) collected at the reproductive fertility consultant of Teaching Hospital for Obstetrics and Gynecology, Kerbela Health Directorate, Iraq from November 2022 to August 2023. Raosoft's sample size calculator was used to determine the sample size. PCOS patients and healthy control were divided into groups depending on their BMI: less than 25 kg/m^2 and more than 25 kg/m^2. The participants in the control group had hirsutism-free, normoandrogenic, regular menstrual periods, as well as morphologically normal ovaries on an ultrasound.

The 2003 Rotterdam ESHRE/ASRM PCOS Consensus Workshop Group diagnostic criteria were used to determine PCOS's diagnosis by the presence of at least two of the three criteria: Oligo/anovulation, clinical and/or biochemical hyperandrogenism, and polycystic ovaries on ultrasound (more than 12 follicles larger than 2-9 mm in diameter or an ovarian volume greater than 10 mL in at least one ovary),[22] biochemical tests, measuring BMI and waist hips ratio had been done. The researcher designed a paper-based questionnaire that was used to interview participants. It consists of a number of questions to know the age, social status, and the period of diagnosis, and make sure that you do not take treatments for long periods of more than 3 months, and used beck anxiety inventory (BAI) to evaluate their anxiety.[23] Exclusion criteria included: thyroid dysfunction, non-classic congenital adrenal hyperplasia, cushing syndrome, androgen-secreting tumors, hyperprolactinemia, kidney or liver failure, hypertension, diabetes mellitus, and any contraception or anti-androgen medication also metformin (within the last 3 months).

Study protocol

A physical examination was done for every woman. Clinical hyperandrogenism-hirsutism (defined as more than four points on the Ferriman-Gallwey score),[24] and the presence of acne were evaluated. Oligo/amenorrhea was defined as a delay in menses of >35 days to 6 months.[25] The participant's height, weight, waist circumference, and hip circumference were measured. Fasting blood collection of the elbow veins was on the 2nd–3rd days of the menstrual period.

Measurements

Luteinizing hormone (LH), follicle-stimulating hormone (FSH), and prolactin hormone levels were measured by the chemiluminescent automates immunoassay system (ECL) (Cobase 411, Roche Diagnostic, Germany). The measurement of free testosterone hormone (T) concentration in (ng/mL) was done by enzyme-based competitive immunoassay (Snibe/Germany). Total cholesterol, triglyceride, low-density lipoprotein cholesterol (LDL), and high-density lipoprotein cholesterol (HDL) were measured by Autoanalyzer Biochemistry (smart-120) (GIESSE/ Italy). Enzyme-linked immunosorbent assay based on the sandwich principle was used for the fasting insulin assay method (Cobas/ Germany). The colorimetric method for using the Glucose kit was used to assess the fasting serum glucose (Biorex/UK).

Calculations

The body mass index (BMI) = weight (kg)/height (m)^2.[26]

Waist-to-hips ratio (WHR) = waist/hip.[27]

Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated = fasting glucose (mg/dL) × fasting insulin (μU/mL)/405.[28]

LAP (Female) = ([WC (cm) - 58] · TG).[29]

VAI (Female) = [WC/(39.68 + 1.88 · BMI)] · (TG/0.81) · (1.52/HDL-C).[30]

Statistical analysis

All participant answers to the questionnaire were entered into a data sheet. The participant's data were analyzed to descriptive statistics. Values for categorical data were represented by *n* (%). Results were regarded as statistically significant if their *P*-values were less than 0.05 (two-sided). The Shapiro-Wilk test was used to evaluate the distribution of the data in terms of normality. The Student's *t*-test was used to compare normally distributed data, whereas the Mann-Whitney *U* test was used for comparing nonparametric data. A correlation between two variables was tested by using Spearman's correlation analysis for non-normal distribution and Pearson's correlation analysis for normal distribution. The Statistical Package for the Social Sciences software, version 28.0 (IBM, SPSS, Chicago, Illinois), was used to generate the data analysis for this study.

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the declaration of Helsinki. It was carried out with patients verbal and analytical approval before sample was taken. The study protocol and the subject information and consent form were reviewed and approved by a local ethics committee according to the document number 3110 (including the number and the date in December 8, 2022) to get this approval.

Results

The study included 170 women, 85 healthy women, and 85 patient women. They had been divided according to their BMI into groups. Groups with BMI < 25 kg/m^2 contain (42 healthy controls1, 24 PCOS1) and groups with BMI > 25 kg/m^2 contain (43 controls 2, 61 PCOS2). Those ages ranged between 18 and 40 years old. The demographic and the assessment of anthropometric measurements were given in Table 1.

As shown in Table 2 the level of LH was high significant in both groups PCOS (1, 2) in comparison to controls (1, 2), respectively, (*P* value = 0.00) with an increase in mean in PCOS2 compare to PCOS1. Also, the level of LH/FSH was variable groups PCOS1, control1 and PCOS2, control 2 with high significance (*P* value = 0.00), Otherwise the levels of FSH were non-significant (*P* value more than 0.05) and PCOS1 has mean slight elevation compare to PCOS2, while free testosterone hormone levels were significant (*P* = 0.001) for both groups. The level of prolactin was a slight increase in PCOS 1, 2 compared to control 1, 2 with highly significant (*P* value = 0.00).

The mean levels of blood sugar PCOS 1, 2 women had higher mean values in comparison with healthy control 1, 2 in both groups respectively. Insulin level and HOMA-IR were significantly higher (*P* < 0.001) in both groups PCOS (1, 2) in comparison to control (1, 2) note an increase in means PCOS2 comparison in PCOS1.

Triglycerides, total cholesterol, LDL, and HDL in PCOS1 groups women were insignificantly in comparison to control1. Otherwise LDL and HDL in PCOS2 in comparison to control2 significantly (*P* < 0.001) but total cholesterol and TG were not significant. The VAI and LAP were higher in the PCOS2 group compared to the PCOS1 group, as well as significantly in both groups PCOS 1, 2 compared to control 1, 2.

As shown in Figure 1, the receiver operator characteristic curves (ROC) for VAI and LAP as predictors for HOMA-IR. PCOS1 and PCOS2 have area under curve (AUC) value which was for LAP (0.327, 0.766) and sensitivity (62%, 71%) and specificity (30%, 67%), respectively, whereas PCOS1 and PCOS2 have VAI with

Table 1: Demographic characteristics of study participants

Feature	PCOS1 Mean ± SD	Control 1 Mean ± SD	*P*-value	PCOS2 Mean ± SD	Control 2 Mean ± SD	*P*-value
Age (years)	23.2 ± 3.9	26.9 ± 4.4	0.03 (S)	24.8 ± 5.0	28.5 ± 4.4	0.039 (S)
BMI (kg/m^2)	22.2 ± 1.91	23.12 ± 1.4	0.8 (NS)	31.1 ± 4.72	29.1 ± 2.7	0.9 (NS)
WHR	0.81 ± 0.04	0.7 ± 0.03	0.08 (NS)	0.81 ± 0.05	0.83 ± 0.06	0.5 (NS)
Hirsutism	18 (75%)	0%	0.001 (S)	38 (62.3%)	0%	0.001 (S)
Acne	15 (62.2%)	0%	0.001 (S)	41 (67.9%)	0%	0.001 (S)
Alopecia	21 (87%)	0%	0.001 (S)	52 (85%)	0%	0.00 (S)
Oligo menorrhea	24 (100%)	0%	0.001 (S)	46 (75.4%)	0%	0.001 (S)
Smoking	Active 1 (4.2%)	Active 0 (0%)	0.05 (S)	Active 4 (6.6%)	Active (0%)	0.05 (S)
	Passive 14 (58.3%)	Passive 18 (41.9%)		Passive 37 (60.7%)	Passive 19 (45.2%)	
	Nonsmoking 9 (37.5%)	Nonsmoking 25 (58.1%)		Nonsmoking 20 (32.8%)	Nonsmoking 29 (54.8%)	
Married	Married (75%)	Married (54%)	0.6 (NS)	Married (73.8%)	Married (60%)	0.7 (NS)
	Single (25%)	Single (46%)		Single (26.2%)	Single (40%)	
BAI	15.4 ± 2.7			27.0 ± 5.37	0%	0.001 (S)

Note: Values are expressed as mean ± SD

BMI: body mass index, WHR: waist-to-hips ratio, BAI: beck anxiety inventory, S: significant, SD: standard deviation, NS: non-significant

Table 2: Biochemical and anthropometric indexes characteristics of the studied groups

Parameters	PCOS1	Control1	*P*-Value	PCOS2	Control2	*P*-values
LH IU/L	9.6±3.3	4.4±1.2	0.001S	11.8±5.2	4.2±0.6	0.001S
FSH IU/L	6.05±1.52	8.84±1.6	0.44	5.5±1.4	8.5±1.05	0.15
LH/FSH	1.6±0.5	0.4±0.1	0.001	2.1±0.9	0.5±0.1	0.001
PRL ng/mL	20.8±5.9	11±2.9	0.001	15.1±6.3	10.9±3.6	0.001
Free.T pg/mL	2.1±0.7	1±0.28	0.001	2.4±01.5	1.1±0.36	0.001
TC mg/dL	142.6±26	150±21	0.37	167±26	160±22	0.166
HDL mg/dL	57.7±7.7	68.9±11	0.68	54.7±6.2	75.6±12.4	0.001
LDL mg/dL	82.8±22.6	69.4±15	0.11	95.4±29	72.6±15	0.001
TG mg/dL	77.3±18.8	69.8±13	0.06	108±42	93±23	0.004
FI µU/mL	10.9±3.6	6.15±1.3	0.001	15.2±4.6	6.3±0.8	0.001
FG mg/dL	96.5±9.3	83.8±4.8	0.01	93.5±9.8	87.5±5.5	0.001
HOMA-IR	2.6±0.9	1.3±0.3	0.001	3.5±1.2	1.4±0.23	0.001
VAI	2.9±1.0	1.7±0.42	0.001	3.1±1.2	2.2±0.5	0.001
LAP	34.5±8.8	20±5.5	0.008	45,4±14.3	40±11.5	0.05

Note: Values are expressed as mean ± SD

BMI: body mass index, LH: luteinizing hormone, FSH: follicle-stimulating hormone, PRO: prolactin, Free.T; free testosterone, FG: fasting glucose, FI: fasting insulin, HOMA-IR: homeostasis model assessment-insulin resistance, CHO: total cholesterol, HDL: high-density lipoprotein cholesterol, LDL: low-density lipoprotein cholesterol, TG: triglyceride, VAI: visceral adiposity index, LAP: lipid accumulation product, SD: standard deviation

AUC value of 0.568, 0.829 and sensitivity (75%, 81%) and specificity (46%, 62%), respectively, as shown in [Tables 3a and Table 4]. In normal weight, the sensitivity and specificity of the LAP and VAI for predictors of IR were noticeably low, whereas LAP and VAI are the best predictors of IR in overweight or obese patients. The correlation analyses between BMI, biochemical, and anthropometric indexes characteristics of the studied groups are shown in [Table 5].

Discussion

Early IR detection and treatment are essential for PCOS patients.[31] Insulin stimulates the release of pituitary LH in addition to the production of ovarian steroids because it has a gonadotropin-enhancing action. IR may be a crucial feature in PCOS's metabolic and reproductive processes,[32] and it is an independent risk factor for diabetes mellitus and cardiovascular diseases,[33] The purpose of this study was to find out more about the relationship between PCOS and BMI and identify the most effective markers that can predict IR in women with PCOS in Karbala city. The study participants were divided into groups based on their BMI: less than 25 kg/m^2 and more than 25 kg/m^2. One of the many signs of PCOS in females is obesity.[34] That there was no significant difference between PCOS women and the control group concerning age. Nevertheless, it is more common in women under 35 years, because women in that age group have biological with ovulation and fertility.[35] The results of the study found a significant difference in LH, prolactin, and free T levels between PCOS (1, 2) women and the control (1,2) groups, respectively. There were no significant differences in FSH levels between the PCOS (groups 1, 2) and the control group (1, 2) respectively. This agrees with research done by Mohanraj *et al.*[36] Nonetheless, PCOS2 has increased slightly prolactin level; therefore, our results are in agreement with the study by Naina and Agarwal.[37] After years of research, no specific, clear findings have been found about the PRL level of PCOS women. Despite study indicated into a slight elevation.[38] While no notable differences in its levels were discovered between the control and PCOS groups in the study by Jiang *et al*.[39]

Our study found that group 2 PCOS has significantly high levels of insulin resistance. Since insulin influences lipid and glucose metabolism, hence insulin resistance has a big impact on those processes. Under insulin stimulation, both glycogenolysis and lipolysis are inhibited. Insulin resistance, however, increases hepatic glycogenolysis and accelerates lipolysis in adipose tissue inappropriately, which upregulates hepatic *de novo* lipogenesis and plays a role in the pathogenesis of PCOS.[40] Our study found a significant difference in HDL, LDL, and TG in group 2 PCOS with high BMI compared to the high BMI of group 2 control but insignificantly for cholesterol in both groups. Dyslipidemia is one of the most common phenomena observed in women with the result of the study by Rashidi *et al.*[41] which is generally consistent with the results of our study.

The both VAI and LAP showed high precision in detecting visceral obesity, and the VAI was one of the more accurate measures of adiposity and dysfunction of adipose tissue because it includes BMI, TG, HDL-C, and WC.[42] They are reliable predictors of IR in PCOS.[43,44] In the study the VAI and the LAP high significant difference between PCOS2 and PCOS1, and sensitivity of LAP and VAI to predictors IR in overweight and obese individuals was 71% and 81% respectively, this agreement with the study by Wang in 2023.[20] and the sensitivity of LAP and VAI to predictors IR in normal weight individuals was 62%

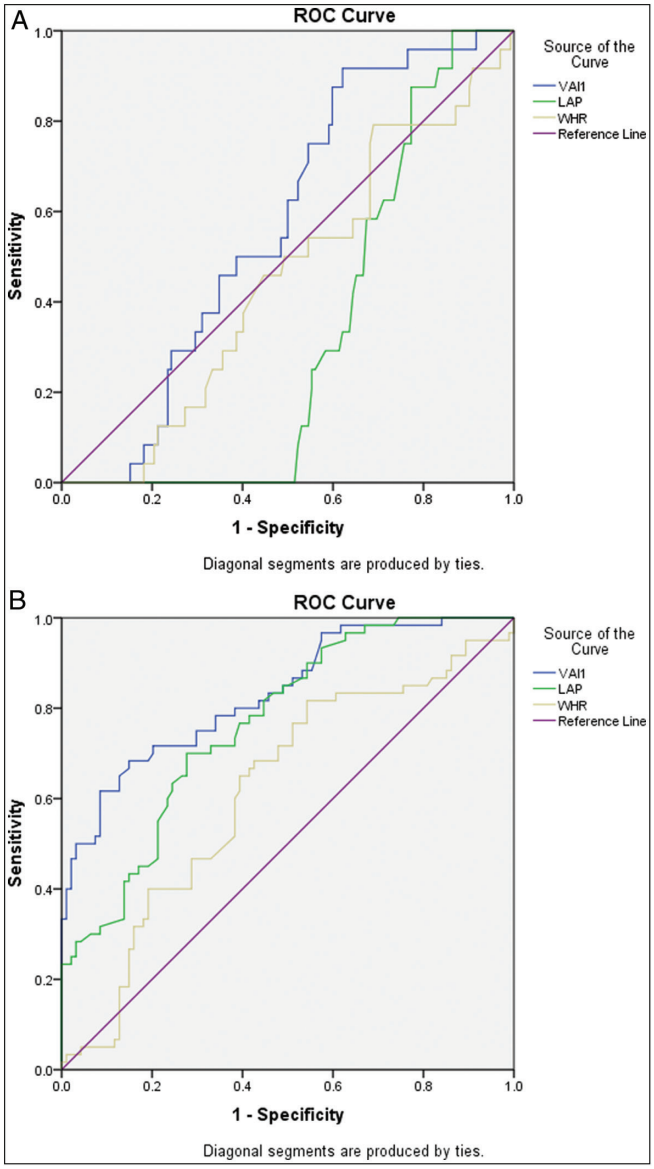

Figure 1: ROC curves for PCOS patients. (a) ROC curves for PCOS1 patients. (b) ROC curves for PCOS2 patients. HOMA-IR: homeostasis model assessment insulin resistance, VAI: visceral adiposity index, LAP: lipid accumulation product

Table 3: Receiver operating curve analysis of anthropometric indexes and HOMA-IR for PCOS1

Variables	AUC	*P*-value	Cut-off values	SS%	SP%	(95% CI)
LAP	0.327	0.007	30.8	62%	30%	0.331-0.532
VAI	0.568	0.288	2.19	75%	46%	0.468-0.669
WHR	0.448	0.422	0.81	54%	46%	0.334-0.563

SS: sensitivity, SP: specificity, AUC: area under curve, CI: confidence interval, VAI: visceral adiposity index, LAP: lipid accumulation product, HOMA-IR: homeostasis model assessment-insulin resistance

and 75% respectively. The VAI had the highest AUC value (AUC=0.829), followed by LAP (AUC=0.766) in group PCOS2.Which VAI is best to predict IR in PCOS. The difference in research results may be associated with different lifestyle and environmental variables.[45] The VAI and LAP were found to be more accurate predictors of IR of PCOS with obesity/overweight in our study. The connection between visceral obesity and IR supports these findings.[46]

Table 4: Receiver operating curve analysis of anthropometric indexes and HOMA-IR for PCOS2

Variables	AUC	*P*-value	Cut-off values	SS%	SP%	(95% CI)
LAP	0.766	0.001	39.43	71%	67%	0.692-0.839
VAI	0.829	0.001	2.251	81%	62%	0.762-0.896
WHR	0.616	0.006	0.824	62%	61%	0.525-0.708

SS: sensitivity, SP: specificity, AUC: area under curve, CI: confidence interval, VAI: visceral adiposity index, LAP: lipid accumulation product, HOMA-IR: homeostasis model assessment-insulin resistance

Table 5: Correlation analyses between BMI, biochemical, and anthropometric indexes characteristics of the studied groups

Parameters	Rho coefficients (*r*)	*P*-value
HOMA-IR	0.40	0.001
Age	0.13	0.077
WHR	0.31	0.001
LH	0.31	0.001
FSH	-0.20	0.007
Prolactin	0.03	0.63
Free.T	0.07	0.58
HDL	-0.10	0.18
LDL	0.30	0.001
TG	0.59	0.001
TC	0.40	0.001
FG	0.31	0.001
FI	0.40	0.001
LAP	0.43	0.001
VAI	0.30	0.001
BAI	0.6	0.001

LH: luteinizing hormone, FSH: follicle-stimulating hormone, Free. T, free testosterone: FG: fasting glucose, FI: fasting insulin, HOMA-IR: homeostasis model assessment-insulin resistance, TC: total cholesterol, HDL: high-density lipoprotein cholesterol, LDL: low-density lipoprotein cholesterol, TG: triglyceride, VAI: visceral adiposity index, LAP: lipid accumulation product, BAI: beck anxiety inventory

Conclusion

Collected data showed that the values for the hormone, lipid profile, and index all increased in obese or overweight when comparing the PCOS group with BMI > 25 kg/m^2 to the PCOS group with BMI 25 kg/m^2. We can conclude that BMI, VAI, and LAP may help early detection of IR in PCOS patients with a BMI of more than 25 kg/m^2.

Acknowledgment

The authors gratefully acknowledge the special funding supported from the Guangzhou Science and Technology Plan Project: 202102020118.

Financial support and sponsorship
Nil.

Conflicts of interest
The authors declare no conflicts of interest.

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