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Original Article

Vitamin C intake is a novel potential therapeutic approach in the secretion of adiponectin and in the treatment of abdominal obesity

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Abstract

Background: The prevalence of abdominal obesity is increasing rapidly worldwide and is a leading cause of morbidities and mortalities. Abdominal obesity is associated with various health complications including insulin resistance (IR), type 2 diabetes etc. The present research studied the outcome of VC intake on adiponectin (ACRP30) levels and its relationship with waist circumference (WC) and IR.

Methods: The present study was carried out on a total of 80 subjects. 42 obese (20 males and 22 females) and 38 non-obese (19 males and 19 females) were enrolled into two groups. Vitamin C (500 mg) was provided to all study subjects and instructed to consume it three times a day over a period of 90 consecutive days. Fasting blood samples were collected at the baseline and end of the study. Adiponectin levels were measured. Data were analyzed by using a two-tailed Student's t-test.

Results: Vitamin C supplementation significantly increased serum adiponectin (ACRP30) levels in obese males ($P = 0.0485$), obese females ($P = 0.0235$), non-obese males (0.0457), and non-obese females (0.0245). Adiponectin is inversely correlated with WC and IR. No significant differences were found in the levels of serum adiponectin between study participants gender-wise.

Conclusion: 500 mg of vitamin C intake, three times a day for 90 days, is a novel potential therapeutic approach to restore the capacity of adipose tissue in secreting adiponectin and for the treatment of abdominal obesity and allied complications.

Keywords: Vitamin C, Obesity, Adiponectin, ACRP30, Waist Circumference, HOMA-IR.

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Globally the prevalence of abdominal obesity is increasing rapidly, and it is becoming a leading cause of morbidities and mortalities (1-3). Abdominal or central obesity is characterized by an abnormally high distribution of adipose tissue around the internal viscera of the abdomen (4). Sedentary lifestyles, less physical activity, and intake of saturated fat and refined carbohydrates are the main reasons of abdominal obesity (5). According to a study, nearly 13% of world's adult population is obese and 41.5% of the adults have abdominal obesity (5). Obesity is at an alarming stage in various South Asian countries (6). Genetically, Asians store much fat around their abdomen as compared to other races (6). Rapid industrial growth, urbanization, shift in the economy, and replacement of traditional healthy food with Western diet (5, 6) are the main reasons behind the development of abdominal obesity in Asian people (7, 8). At present around 350 million obese subjects are present in India (9) with an annoyingly rising trend of abdominal obesity (10). Abdominal obesity is associated with insulin resistance, type 2 diabetes mellitus (T2DM), chronic inflammation, cardiovascular diseases, various types of cancers, etc. (1-3).

Adiponectin (ACRP30), is a protein hormone secreted by the adipocytes (11). It controls energy intake, insulin sensitivity, glucose and lipid metabolism, body weight, and protects against chronic inflammation (12, 13). Studies have shown that, plasma adiponectin mediates the association between central obesity and type 2 diabetes, which attenuates the adverse progression from adiposity to type 2 diabetes (14-17). As the ACRP30 has an inverse association with insulin resistance (IR) (18), obesity, and atherosclerosis (19), various studies have been carried out regarding the pharmacological elevation of ACRP30, but the concerns were raised up as pharmacotherapy adversely affected many vital organs like; use of thiazolidinediones (TZDs) increased the risk of heart failure (20) and bones fractures (21, 22). Also, several deaths due to acute liver failure were reported after the use of troglitazone (23). Use of pioglitazone resulted in bladder cancer (24) and macular edema in diabetic patients (25). That pose limitations for use of drugs in the treatment of metabolic disorders. Therefore, a non-invasive yet effective approach is needed to treat abdominal obesity. As obesity is often been allied with oxidative stress, antioxidant treatments are considered as an interesting approach to counteract central obesity and related complications (26-28).

It has been reported that, when isolated human adipocytes were treated with vitamin C, resulted in adiponectin gene (ADIPOQ) expression and secretion of HMW adiponectin (29, 30). Vitamin C is a potent antioxidant and its intake is negatively associated with obesity, diabetes mellitus, atherosclerosis, stroke, etc. (28). Therefore, the present work was aimed to study the effect of VC on plasma adiponectin (ACRP30) and its correlation with WC and IR.

Methods

Ethical consideration: The Institutional Ethics Committee of Krishna Institute of Medical Sciences (Deemed to be University), Karad had approved this study. Registration number is KIMSDU/KIMS/IEC/3/2013. Present study was carried out at Krishna Institute of Medical Sciences (KIMS), Karad, Maharashtra, India.

Study period: Study was carried out for 18 months from January 2015 to June 2016.

Sample size calculation: Other similar studies were considered for sample size calculation (31, 32). Considering leptin with 95% CI, power 80%, difference between leptin levels of obese and non-obese = 9.43±13.68 ng/ml, at least 35 participants and by considering 10% dropout, minimum 39 subjects should be enrolled in both obese and non-obese groups. One was dropped from the non-obese group, therefore finally 38 subjects remained in the non-obese group. A total of 42 obese (20 males, 22 females) and 38 non-obese (19 males, 19 females) subjects, aged 20-45 years, were enrolled in this study.

Inclusion criteria: Apparently healthy non-smokers, non-alcoholic, male and female obese and non-obese subjects.

Exclusion criteria: To prevent heterogeneity in results and misconception and biased assessment of effectiveness of the vitamin C intervention, following subjects were excluded from the present study. Subjects with tuberculosis, malignancy, diabetes mellitus, renal disease, hepatitis, hypothyroidism, Cushing's syndrome, pregnant women, any infectious diseases during the study, subjects taking NSAID, and those on hypolipidemic drugs were omitted from the study. Subjects on medication for any illnesses or food supplements, also excluded from present study.

Study design: A purpose of the study, objectives and risk factors were properly explained to all the study subjects and their informed written consent was obtained. Utmost care was undertaken in accordance with the Declaration of Helsinki (33) during a study period. WC was recorded twice to the nearest 0.10 cm with stress-resistant tape to confirm relentless tension during measurement. The WC was measured at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest by using stretch-resistant tape. Indian WC guidelines were followed to define abdominal obesity (34, 35).

Blood samples of the participants were collected after the 12-14 hrs. Of overnight fasting. The serum was separated and analyzed for ACRP30 levels by using the standard ELISA protocol. Subjects were provided with and asked to consume vitamin C tablets (500 mg), three times a day after breakfast, lunch, and dinner for 90 consecutive days. Previous similar studies were considered while selecting the dose and duration of the vitamin C intervention for the present study (36-40). Subjects were advised to continue their daily routine. Weekly follow-up was taken and compliance was monitored through phone calls, in-person meetings, subjects' self-reports, tablets count etc.

Statistical analysis: Data were reported as mean±standard deviation (SD) and all statistical analyses were performed using SPSS, Version 20. The two-tailed student 't' test (paired 't' test) was used to estimate differences between ACRP30, assessed before and after the supplementation of vitamin C in obese and non-obese subjects. On the contrary, an unpaired t-test was used to compare baseline study variables between obese and non-obese subjects (obese versus non obese). Pearson correlation coefficients between the study variables like; WC, HOMA-IR and adiponectin of

obese and non-obese were determined. If $p < 0.05$ then the differences and correlation coefficients were considered significant.

Results

In the present study, after the intake of VC a significant increase in ACRP30 levels in all study participants was observed viz; OM, OF, NOM, and in NOF ($p<0.05$) (table 1). Some of the data from Table 1 are from our previously published original research work (26, 27). At the study baseline, no significant difference was seen in the ACRP30 levels between OM and OF and between NOM and NOF ($p>0.05$) (table 2). At the study baseline, no significant difference was seen in the ACRP30 levels between OM and NOM ($p>0.05$), but the ACRP30 level was significantly higher in NOF when compared with OF at the study baseline ($p<0.05$) (table 3). There was a negative correlation between WC and ACRP30 in all obese (OM, OF) and non-obese (NOM, NOF) subjects (table 4). A negative correlation was observed between HOMA-IR and ACRP30 in OM and OF, while positive correlation was seen between HOMA-IR and ACRP30 in NOM and NOF (table 4).

Table 1. Comparison of baseline and end values of adiponectin (ACRP30) in obese and non-obese subjects

Variables	OM (n=20) (Mean±SD)	OF (n=22) (Mean±SD)	NOM (n=19) (Mean±SD)	NOF (n=19) (Mean±SD)
WC (cm) (22)				
Baseline	103±7	91±6	81±4.73	75±7
End	99±7	89±6	79±4.75	73±7
T	8.718	13.667	15.62	10.875
P	**<0.0001**	**<0.0001**	**<0.0001**	**<0.0001**
FBG (mg/dl) (23)				
Baseline	94±13	97±19	101±19	87±10
End	89±9.5	90±9	89±10	84±3
T	2.301	2.503	3.686	1.582
P	**0.0329**	**0.0206**	**0.0017**	0.1311
Serum Insulin (µU/ml) (23)				
Baseline	18.45±14.55	13.46±5.60	12.18±10.74	7.56±3.58
End	14.05±14.26	9.76±6.69	6.84±3.53	6.10±3.34
T	2.272	2.406	2.069	1.528
P	**0.0349**	**0.0254**	0.0533	0.1440
HOMA-IR (23)				
Baseline	4.25±3.04	3.21±1.40	3.20±2.98	1.65±0.85
End	3.05±3.0	2.24±1.71	1.53±0.85	1.27±0.78
T	2.385	2.792	2.339	1.648
P	**0.0277**	**0.0109**	**0.0311**	0.1167
ACRP30 (µg/ml)				
Baseline	32±13	33±12	41±18	43±12
End	40±13	40±11	50.4±9	48±11
T	2.109	2.442	2.146	2.245
P	**0.0485**	**0.0235**	**0.0457**	**0.0245**

OM- obese males, OF- obese females, NOM- non obese males, NOF- non obese females, WC- waist circumference, FBS- fasting blood glucose, HOMA-IR- Homeostatic model assessment of insulin resistance, ACRP30- Adiponectin.

Table 2. Comparison of baseline ACRP30 levels between obese and non-obese subjects

Variable	OM	OF	t and p	NOM	NOF	t and p
	Baseline Mean±SD	Baseline Mean±SD		Baseline Mean±SD	Baseline Mean±SD	
ACRP30 (µg/ml)	32±13	33±12	t= 0.0920, p=0.9275	41±17.2	43±12	t= 0.3256, p= 0.7466

Table 3. Comparison of baseline ACRP30 levels between OM and NOM and between OF and NOF

Variable	OM	NOM	t and p	OF	NOF	t and p
	Baseline Mean±SD	Baseline Mean±SD		Baseline Mean±SD	Baseline Mean±SD	
ACRP30 (µg/ml)	32±13	41±17	t=1.863, p=0.0704	33±12	43±12	t=2.475, p=0.0178

Table 4. Correlation of ACRP30 with WC and HOMA-IR at study baseline in obese and non-obese individuals

Variables		OM	OF	NOM	NOF
ACRP30 (µg/ml)	WC (cm)	r=-0.180, p=0.449	r=-0.033, p=0.883	r=-0.097, p=0.693	r=-0.194, p=0.426
	HOMA-IR	r=-0.2553, p=0.2773	r=-0.051, p=0.8201	r=0.2047, p=0.4005	r=0.0284, p=0.4908

Discussion

Adiponectin (ACRP30) is an adipocytokine, it is associated with glucose and fat metabolism, intake of energy, insulin sensitivity, body's fat regulation, and protection from chronic inflammation (11-13, 18-19). A low plasma adiponectin concentration is associated with increased cardiovascular risk (18), hyperinsulinemia, IR (41), and type 2 diabetes mellitus (T2DM) (42).

In present work, intake of 500 mg of vitamin C three times a day, for 90 days resulted in a significant rise in ACRP30 levels, in both obese and non-obese subjects (table 1). It has been seen that, when isolated human adipocytes were treated with vitamin C, resulted in adiponectin gene (ADIPOQ) expression and secretion of high molecular weight adiponectin (29, 30). In the present study, intake of vitamin C might have stimulated adiponectin gene expression and enhanced biosynthesis of ACRP30 in all study participants. In the study by Johnston *et al*. on obese individuals, a baseline plasma ACRP30 level was found positively correlated with plasma vitamin C and negatively with body fat mass, also 8 weeks of 500 mg of vitamin C intake, did not change their circulating ACRP30 level (36), however in the present study, the dose and duration of the vitamin C intake, could have resulted in increased plasma ACRP30 levels in all study participants. Vincent *et al*. also reported that, antioxidant supplementation (vitamin C 500 mg, vitamin E 800 IU, and β-carotene 10 mg), significantly increased ACRP30 levels in both normal weight and overweight male and female subjects (43).

In the present study, there was found no significant difference in the ACRP30 levels between OM and OF (table 2). Similar findings on obese subjects were reported in a study by Abraham *et al*. (44). The present study reported no significant differences in the adiponectin levels between OM and OF and between NOM and NOF (table 2). This study concurs with the study of Vayghan *et al*. where they did not find significant differences in the levels of ACRP30 in healthy men and women having similar BMI (45). In the current study, it was also observed that OM have low ACRP30 levels as compared to NOM but that difference was insignificant, whereas ACRP30 was significantly lower in OF as compared to NOF (table 3). It may possible that, other factors like physical activities or diet have increased the plasma ACRP30 concentration in the NOF. Moreover, ACRP30 concentration is inversely aligned with the body fat mass (46) that explains why obese subjects have less adiponectin as compared to their non-obese counterparts. Obesity is associated with coronary endothelial dysfunction

(47), the circulating levels of adiponectin are decreased in obesity, which have antagonist effect on vascular function (48). In the present study, a negative correlation of ACRP30 with WC and HOMA-IR suggests that, plasma adiponectin is inversely aligned with the development of abdominal obesity and insulin resistance (table 4). Also, intake of vitamin C has increased the plasma adiponectin concentration and decreased WC and HOMA-IR levels (table 1). These results emphasize the importance of vitamin C in the treatment of abdominal obesity and insulin resistance.

Intake of vitamin C is associated with body fat reduction (26-28) improved coronary function and enhanced plasma adiponectin levels (19). Also, vitamin C treatment on isolated human adipocytes resulted in molecular expression of adiponectin gene and secretion of adiponectin (29, 30). Intake of vitamin C may have similar effects on adiponectin gene expression that would have resulted in enhanced plasma adiponectin levels in all study participants. 500 mg of vitamin C intake, three times a day for 90 days, is a novel potential therapeutic approach to restore the capacity of adipose tissue in secreting adiponectin and for the treatment of abdominal obesity and allied complications.

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Ethical Approval: The Institutional Ethics Committee of Krishna Institute of Medical Sciences (Deemed to be University), Karad had approved this study. Registration number is KIMSDU/KIMS/IEC/3/2013.

Conflict of interests: None

Author contributions: Ganesh H. Ghanwat and Ajit V. Sontakke planned the present study; Ganesh H Ghanwat carried out the study under the guidance of Ajit V. Sontakke. Ganesh H. Ghanwat, Anup S. Hendre and Bhagyashri Yadav collected the data and analysed. Ganesh H. Ghanwat, Rohan S Phatak and Amar R Mohite discussed and prepared the manuscript.

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