Original Communication

Lycopene alleviates Western diet-induced elevations in anthropometrical indices of obesity, adipose lipids, and other nutritional parameters

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Abstract: *Objective:* Given the unrelenting surge in the prevalence of obesity and the intensified efforts aimed at elucidating underlying mechanisms and proffering effective treatments, this study investigated the effects of lycopene on various anthropometrical indices of obesity. *Methods:* Thirty female Wistar rats were equally divided into two groups and fed either control diet or Western diet. After eight weeks, obese rats (fed Western diet) were divided into three groups (n=5); obese control received the vehicle, while the other two received lycopene (0.2 and 0.4 mg/kg body weight, respectively). Normal rats were grouped into three (n=5) and treated similarly. This treatment lasted for another two weeks, in addition to their respective diets. Afterwards, anthropometrical indices were taken. *Results:* The weight gain, adiposity index, abdominal and thoracic circumference, body mass index, and Lee index were significantly increased ($p<0.05$) in the obese rats compared to the normal control, by 108.3%, 102.1%, 81.5%, 97.6%, 47.4%, and 13.9%, respectively. The obese rats had significantly ($p<0.05$) higher adipose tissue lipid contents, daily feed (37.4%) and energy intake (66.0%), daily weight gain (108.3%), and feed efficiency (25.5%) compared to control. However, the treatment of obese rats with lycopene occasioned a dose-dependent reduction in the elevated anthropometrical and nutritional parameters. In addition, lycopene elicited significant reductions ($p<0.05$), ranging from 16–54%, in the adipose lipid contents. *Conclusion:* The data presented here illustrate the positive effects of lycopene on indices of obesity and other anthropometric parameters in obese female rats.

Keywords: obesity, anthropometric measurements, lycopene, western diet, BMI

Introduction

The wind of change that is Westernization has brought, among other things, changes in lifestyle and eating habits [1]. These social and environmental changes (such as the increasingly sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization) are accompanied by increased consumption of foods high in saturated fats and refined sugars (as such, are energy-dense) and an augmented lack of physical activity [2]. The interplay between these factors is thought to be responsible for the escalating incidences of obesity [3].

The ever-growing obesity epidemic is now a foremost global health concern, affecting both adults and children. Obesity, formerly deemed a problem of high-income nations, now afflicts even the low- and middle-income nations [2]. As of 2019, over 39% of the world population (over 2 billion out of ~7.6 billion) were overweight, while between 2013 and 2014, about 70% of adults in the USA (the Western society's quintessential nation) were either overweight or obese [2, 4]. More than 40 million children (<5 years of age) were projected to be overweight or obese, in 2019. Globally, most of the world's populace reside in regions where death rates due to obesity are higher than those from underweight. Further, there are more obese individuals than underweight individuals in almost all regions of the world, except in parts of sub-Saharan Africa and Asia. If these worrying drifts persist, about 2.7 billion

adults will become overweight or obese by 2025 [2, 4]. This health burden is a prominent risk factor for the development of many diseases such as cardiovascular diseases, diabetes, cancer, cognitive impairment, and dyslipidemia [5].

Given these points, efforts are being intensified to elucidate the mechanisms at play and possible ways of mitigating the obesity epidemic. Although several animal models of studying obesity exist, one commonly utilized model is the diet-induced model of obesity (DIO), which simulates a more human-like obesity model compared to other models, like genetic modification [3, 6]. The western diet used in this study is characterized by its high energy density (high saturated fatty acids and high amounts of simple sugars), low fibre content, and high salt content [3, 7]. Several parameters such as body mass index (BMI), Lee index, energy intake, body weight gain, thoracic circumference (TC), abdominal circumference (AC), and AC to TC ratio are used as indicators to demonstrate increased adiposity and investigate the mechanisms underlying obesity [8, 9, 10]. Anthropometric measurements have made enormous contributions to the assessment of nutritional status and diagnosis of nutritional abnormalities such as obesity and protein-energy malnutrition, as well as monitoring the efficacies of treatment regimens [11].

Although obesity is correctable, especially by caloric restriction and moderate exercise, sustaining such intensive behavioural changes over a long time is challenging, contributing to the failure of such lifestyle intervention. Moreover, the most suitable dietary style for enduring weight loss is still elusive [2, 12]. Furthermore, previously used medications for managing obesity, such as orlistat, lorcaserin, and phentermine-topiramate, have been largely limited by side effects, ranging from cardiovascular and gastrointestinal issues to cancer [13, 14]. Several bioactive compounds – like curcumin, resveratrol, quercetin, among others – have been researched and have been demonstrated to possess various direct or indirect effects on relevant molecular pathways, linked to the aetiology of cardiovascular diseases, diabetes, obesity, metabolic syndrome, and cancer. Being natural constitutents of foods, they are much safer than synthetic drugs and are emerging therapeutic agents for several conditions, including obesity [15, 16].

Lycopene ($C_{40}H_{56}$) is an unsaturated, lipophilic, non-provitamin-A carotenoid, found in tomatoes, watermelon, papaya, red grapefruits, apricots, and guava [16]. High lycopene intake has been associated with low visceral and subcutaneous fat mass, as well as a lower incidence of metabolic syndrome in men [17]. Lycopene has been reported to express anti-oxidative, lipid-lowering, anti-atherosclerotic, anti-inflammatory, antiplatelet, antihypertensive, and cardio-protective properties [18, 19]. Moreover, a careful review of existing literature revealed that most studies on the anti-obesity effects of lycopene were preventive [19, 20, 21], which raises the question; is there hope for those already obese? Besides, despite the encouraging reports concerning the effects of bioactive compounds, many of the conducted studies have different experimental designs and their result are often controversial or inconclusive, therefore, more studies are required in order to clarify their beneficial effects [15]. On this backdrop, this study sought to investigate the effects of lycopene on various anthropometrical indices of obesity in an animal model of DIO.

Methodology

Chemicals

Lycopene (crystalline; all-trans; $\leq$98% purity) was procured from Solarbio Life Science and Co. Ltd. (Tongzhou District, Beijing, China).

Ethical statement

This study received ethical approval from the ethics committee of the Department of Biochemistry, College of Biosciences, Federal University of Agriculture, Abeokuta (FUNAAB), with Ref No: FUNAAB/CBS/BCH/PG/17-0370. All conditions of animal experimentation followed the ARRIVE guidelines of the Percie du Sert et al. [22].

Experimental animals

Thirty female Wistar rats (weighing between 150–200 g; 8–10 weeks old) used in this study were obtained from a reputable animal farm, housed in separate cages under ambient conditions, in the animal house of the Department of Biochemistry, FUNAAB.

Experimental diet

Western diet was used to simulate an animal model of diet-induced obesity, which in addition to the control diet, was compounded as described by Bartolin et al. [3] (Table 1).

Experimental design

After two weeks of acclimatization, the Western diet was fed to fifteen rats for eight weeks to establish obesity, while the other fifteen rats were fed the control diet. After the first eight weeks, the rats were further divided into six groups (n=5) and treated for another two weeks, as follows:

Table 1. Composition of experimental diets

Diet composition (g per kg of chow)	Control diet	Western diet
Soy protein	200	200
Lard	0	180
Soy oil	40	40
Corn starch	547	177
Simple sugars	100	300
Fiber	50	25
Table salt	5	20
Vitamin mix	10	10
Mineral mix	40	40
Choline	2	2
Methionine	3	3
Lysine	3	3
Energy (kcal/g)	~3.76	~4.54

- Group A (Normal control): Control diet
- Group B (Obese control): Western diet
- Group C (Obese): Western diet+lycopene [0.2 mg/kg body weight (BW)]
- Group D (Obese): Western diet+lycopene (0.4 mg/kg BW)
- Group E (Normal): Control diet+lycopene (0.2 mg/kg BW)
- Group F (Normal): Control diet+lycopene (0.4 mg/kg BW)

Lycopene was dissolved in olive oil and administered to the animals by oral gavage for two weeks, while the whole study lasted for ten weeks. The dose of lycopene used was based on the estimated amount shown to be beneficial in studies, i.e., 9–21 mg/70 kg BW (0.13–0.3 mg/kg BW) [19, 23].

Feed intake and body weight

The diets were pelletized and given fresh daily. The feed intake per day was calculated as the difference in weight between feed given and the remnants after each day. The body weight of each animal was taken weekly.

Anthropometrical parameters

Anthropometrical parameters were measured as described by Novelli et al. [8]. The abdominal circumference (AC; immediately anterior to the forefoot), thoracic circumference (TC; immediately behind the fore-leg), and body length (nose-to-anus length) were measured in all groups of rats at the end of the treatment. Furthermore, body weight and body length were used to calculate the following anthropometrical parameters:

a. Body mass index (BMI; g/cm^2)=*body weight (g)/body length^2 (cm^2)*
b. Lee index (g^{-3}/cm^{-1})=*cube root of body weight (g)/body length (cm)*

Intake and feed efficiency

Using calorie and feed intake, the following nutritional parameters were calculated [8, 24]:

a. Feed intake (g/day)=*total feed consumed (g)/study duration (day)*
b. Energy intake (kcal/day)=*feed consumed (g/day)*×*energy content of diet (kcal/g)*
c. Feed efficiency (%)=*[mean body weight gain (g)/feed intake (g)]*×*100*

Adiposity index

At the end of the experiment, animals were sacrificed by decapitation, and body fat rapidly isolated, washed in ice-cold 50 mM phosphate buffer saline (PBS; pH 7.4), patted dry, and weighed. To calculate the adiposity index, the total body fat was divided by body weight as follows [9]:

a. Adiposity index (%)=*[total body fat (g)/final body weight (g)]*×*100*

Where total body fat=*Epididymal fat+Visceral fat +Retroperitoneal fat*

Adipose tissues lipid profiling

Lipids were extracted from the visceral adipose tissues (VAT) and the levels of phospholipids, triacylglycerols, and cholesterol were then determined with commercial kits obtained from Randox Laboratories (Crumlin, County Antrim, United Kingdom), following the methods previously described [25]. Free fatty acids (FFA) were also determined in the VAT, following the method described by Soloni and Sardina [26], and based on the principle that cuprizone reagent reacts with the chloroform extract of FFA in an alkaline medium to produce a coloured complex, whose intensity is directly proportional to the FFA level at 620 nm.

Statistical analyses

Quantitative variables were expressed as mean±standard error mean (SEM) of five rats. The normality of data distribution and homogeneity of variance was tested using the

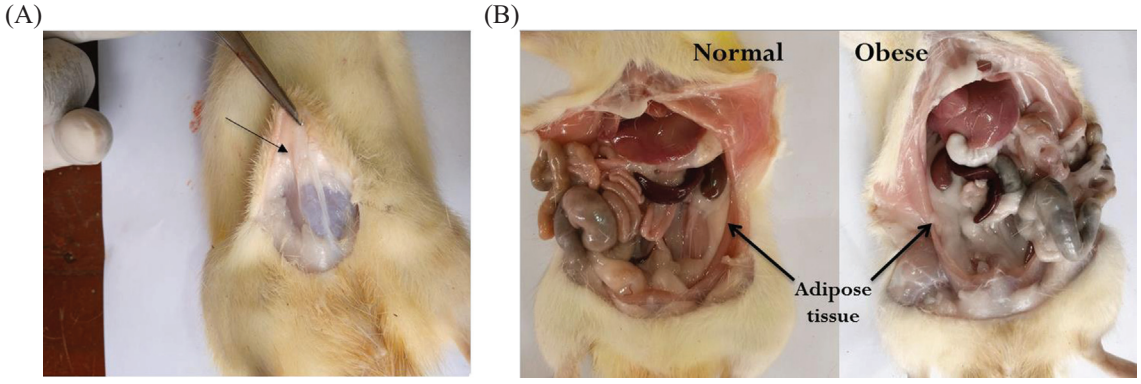

Figure 1. Representative snapshots of (A) flap (showing fat depots) and (B) adipose tissues of normal and obese rats.

Table 2. Effects of lycopene (Lyc) on some nutritional parameters in obese and normal rats

Groups	Feed intake (g/day)	Energy intake (kcal/day)	Weight gain (g/day)	Feeding efficiency (%)
Normal control	42.07±0.59	158.19±2.21[a]	1.62±0.05	1.02±0.03
Obese control	57.79±0.34*	262.52±1.54*	3.37±0.04*	1.29±0.02*
Obese+Lyc (0.2 mg/kg BW)	55.64±0.53*[#]	252.79±2.42*[#]	3.21±0.03*[#]	1.27±0.01*
Obese+Lyc (0.4 mg/kg BW)	56.64±0.33*[#]	257.33±1.50*[#]	3.19±0.05*[#]	1.24±0.02*
Normal+Lyc (0.2 mg/kg BW)	46.79±0.35*[#]	175.91±1.32*[#]	1.65±0.01[#]	0.94±0.01[#]
Normal+Lyc (0.4 mg/kg BW)	43.79±0.31*[#]	164.63±1.18[#]	1.67±0.03[#]	1.01±0.02[#]

Values, which are mean±SEM of five rats, bearing * and/or [#] are significantly different from normal control and/or obese control, respectively ($p<0.05$). BW: body weight.

Shapiro-Wilk test and Levene's test, respectively. Significance of difference between groups was tested with one-way Analysis of Variance and then Tukey's test, with a $p<0.05$ considered significant. All analyses were done with the Statistical Package for Social Sciences (version 20.0).

Results

Representative snapshots of the abdominal flap of obese rat, as well as the adipose tissues of normal and obese rats are presented in Figure 1.

Table 2 summarizes the effects of lycopene on some nutritional parameters in obese and normal rats. The feed and energy intake, as well as weight gain of the obese rats, were significantly higher ($p<0.05$) than those of normal control, by 37.4%, 66.0%, and 108.3% respectively. Treatment of obese rats with lycopene (0.2- and 0.4 mg/kg BW) significantly reduced ($p<0.05$) the feed and energy intake as well as weight gain, when compared to the obese control rats, with percentage decreases ranging from 2 to 8. Finally, though the Western diet showed significantly higher ($p<0.05$) feeding efficiency, lycopene did not have any significant effect on the feeding efficiency in obese rats.

The body weight gain and adiposity index were markedly increased in the obese rats compared to the normal control, by over one fold in both cases. Treatment with lycopene (0.2- and 0.4 mg/kg BW) significantly reduced ($p<0.05$) the gain in body weight and adiposity index (5 to 8 percentage decrease). Moreover, the administration of lycopene to normal rat did not affect the body weight gain or adiposity index (Figure 2).

The AC and TC of obese rats were significantly increased ($p<0.05$) by 83.1% and 71.7% respectively, when compared to the normal control. Treatment with lycopene occasioned a dose-dependent reduction in the AC and TC of obese rats, whereas, in normal rats, lycopene occasioned no significant change ($p>0.05$). Furthermore, the ratio of AC to TC was significantly higher ($p<0.05$) in the obese rats compared to the control (by 6.7%), while treatment with lycopene reduced the AC/TC in obese rats (Figure 3).

The effects of lycopene on anthropometric measurements of obese and normal rats are shown in Table 3. The final body weight of obese rats was significantly increased ($p<0.05$) by 46.8% when compared to the control. Obese rats treated with lycopene (0.2- and 0.4 mg/kg BW) showed a significant decrease ($p<0.05$) in final body weight (5.4% and 5.2% respectively) when compared to the obese control group. There was no significant difference ($p>0.05$) in the body length of normal and obese rats, with or without lycopene treatment. The BMI and Lee index of the obese rats were significantly increased ($p<0.05$) compared to the normal control, by 47.4% and 13.9%, respectively. Whereas

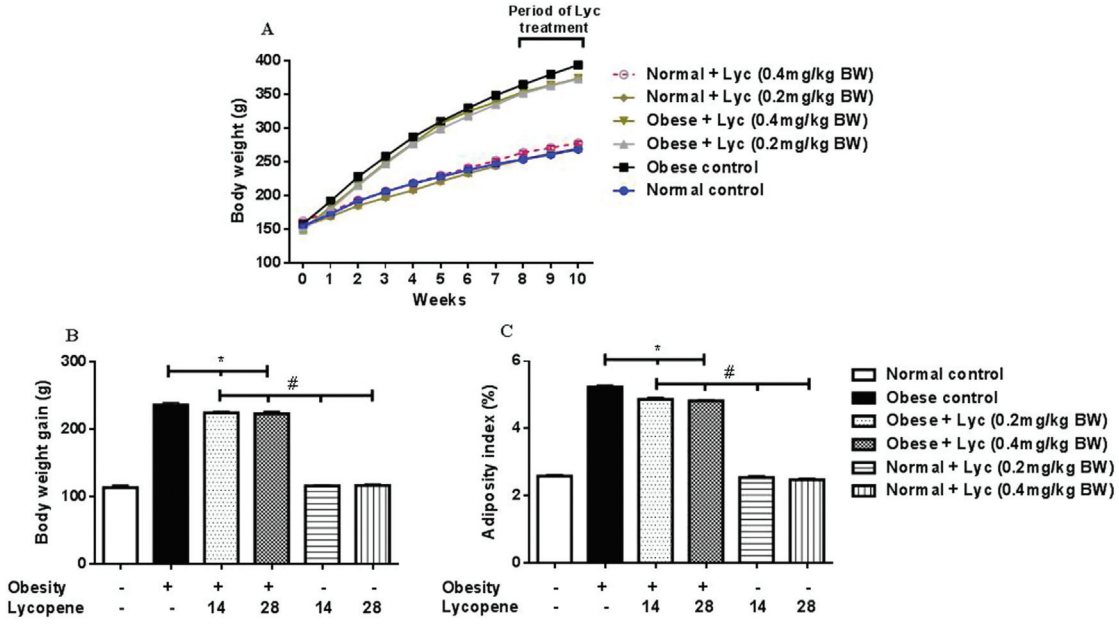

Figure 2. Effects of lycopene (Lyc) on weekly body weight (BW), body weight gain (in grams) and adiposity index (%) in normal and obese rats. Bars, which are mean±SEM of five rats, bearing * and/or # are significantly different from normal control and/or obese control, respectively ($p<0.05$).

treatment with lower dose of lycopene (0.2 mg/kg BW) showed no significant ($p>0.05$) on the BMI and Lee index, the higher dose occasioned a significant ($p<0.05$) reduction in the BMI and Lee index of obese rats. Besides, these anthropometric measurements, in normal rats treated with both doses of lycopene, were statistically similar to those of control.

Ingestion of Western diet induced significant elevations ($p<0.05$) in the levels of phospholipids, triacylglycerols, cholesterol, and FFA levels in the adipose tissues of obese rats. The observed elevations were by 42.1%, 30.1%, 1.5 folds, and 47.6%, respectively. However, lycopene elicited a reduction in these adipose lipid contents, with percentage decreases ranging from 16 to 54 (Table 4).

Discussion

Obesity, persistently linked to several leading causes of diseases worldwide, is presently a global menace. More alarming is the unprecedented rise in its prevalence, especially among children, with the cases of obesity now three times that of 1975 [2]. Management and treatment of obesity usually involve sustained weight loss and decreasing the risk factors for obesity [27]. Different anthropometric parameters are often employed to monitor the efficacy of these treatments [8, 11]. In this study, we appraised the effects of lycopene on relevant anthropometric parameters and indices of obesity in rats.

We chose the Western diet because it closely resembles the diets humans being are exposed to, and produces a more robust diet-induced obesity model than conventional high-fat and cafeteria diets [3]. Indeed, the diet effectively induced obesity, evidenced by the increased body weight gain and adiposity index in the Western diet-fed rats (Figure 2). The observed increases in weight gain and adiposity index have been previously reported in different DIO models [3, 8, 10], and is substantiated by the snapshots presented in Figure 1, which reveals accumulation of fats under the skin and hypertrophy of visceral adipose tissues in obese rats. The Western diet is similar to the diets human ingest (high-fat, high-sugar, high-salt, energy-dense, and micronutrient-poor foods), which are more convenient, easily accessible, relatively cheaper, but also lower in nutrient quality [2, 3]. More so, these dietary patterns, along with reduced physical activity, are believed to be responsible for the surge in the prevalence of obesity in adults and children [2]. Treatment with lycopene significantly attenuated the gain in body weight and reduced the adiposity index, an observation corroborated by the findings of Wang et al. [28] and Wang et al. [29]. Wang et al. [28] has previously demonstrated that lycopene reduced fat mass by down-regulating the expressions of lipogenesis genes (such as genes coding for acetyl-CoA carboxylase and fatty acid synthase) and increasing the expressions of lipolysis related genes, including thermogenic and mitochondrial functional genes. In another study [29], lycopene was shown to suppress lipid accumulation in tissues of mice fed with high-fat diet, by upregulating the expression of PPARα and

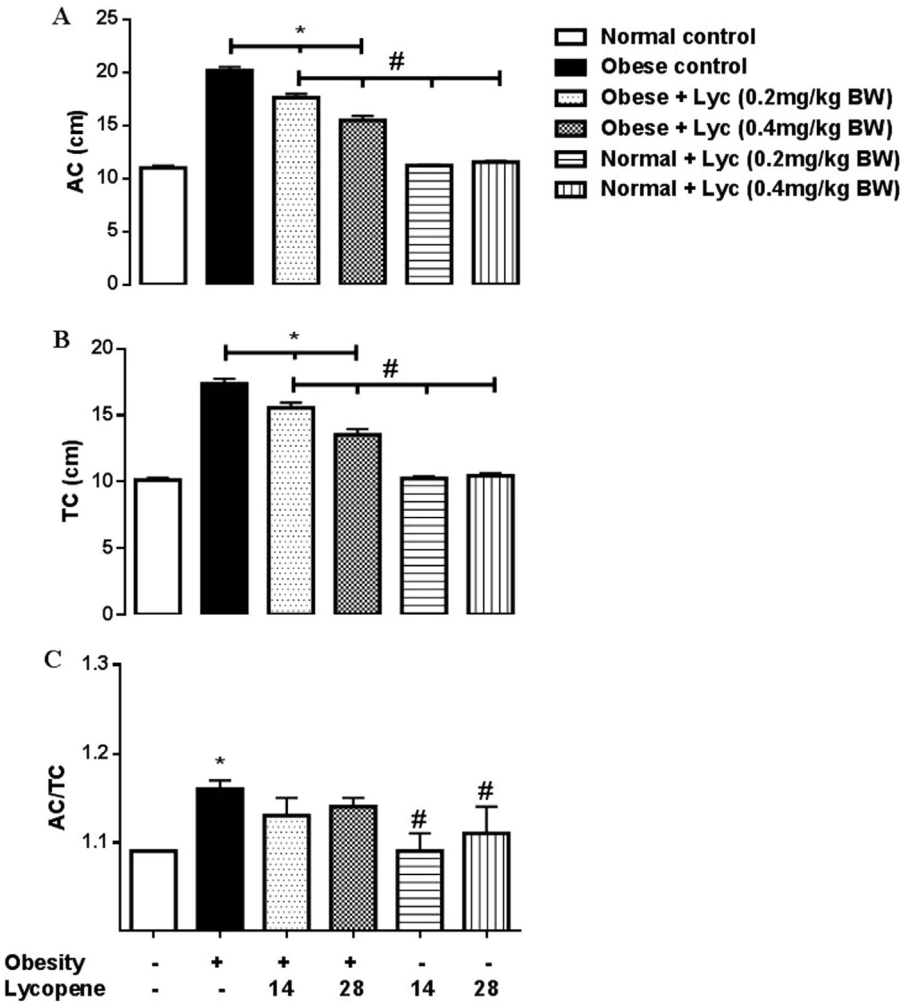

Figure 3. Effects of lycopene (Lyc) on abdominal circumference (AC), thoracic circumference (TC), and AC-to-TC in obese and normal rats. Bars, which are mean±SEM of five rats, bearing * and/or # are significantly different from normal control and/or obese control, respectively ($p<0.05$).

Table 3. Effects of lycopene (Lyc) on some anthropometric measurements in obese and normal rats

Groups	Final body weight (g)	Body length (cm)	BMI (g/cm^2)	Lee index ($g^{-3}cm^{-1}$)
Normal control	268.60±2.86	19.36±0.37	0.72±0.02	0.33±0.01
Obese control	394.40±2.69*	19.30±0.14	1.06±0.02*	0.38±0.00*
Obese+Lyc (0.2 mg/kg BW)	373.00±1.82*#	19.38±0.75	1.01±0.08*	0.37±0.01*
Obese+Lyc (0.4 mg/kg BW)	373.60±1.21*#	20.10±0.30	0.93±0.03*#	0.36±0.01
Normal+Lyc (0.2 mg/kg BW)	269.80±3.18#	18.86±0.11	0.76±0.01#	0.34±0.00#
Normal+Lyc (0.4 mg/kg BW)	278.40±2.96#	18.98±0.43	0.78±0.03#	0.34±0.01#

Values, which are mean±SEM of five rats, bearing * and/or # are significantly different from normal control and/or obese control, respectively ($p<0.05$). BMI: body mass index; BW: body weight.

improving mitochondrial function, thereby enhancing lipolysis. These postulations suggest that an underlying mechanism through gene expression may be involved in the positive effects observed and are further substantiated by the observation that the lipid levels in the adipose tissues, which were initially elevated following consumption of the Western diet, were markedly reduced upon treatment with lycopene, as shown in Table 2. In addition, Luvizotto et al. [30] revealed that lycopene was able to increase both the mRNA levels and plasma concentration of adiponectin, *in-vivo*, in diet-induced obesity. Adiponectin is an adipokine produced and secreted exclusively by the adipose tissues and, among other anti-obesity effects, stimulates fatty acids oxidation and lipolysis. Therefore, lycopene-evoked

Table 4. Effects of lycopene (Lyc) on lipid contents in the adipose tissues of obese and normal rats

Groups	Phospholipids (mg/g tissue)	Triacylglycerols (mg/g tissue)	Cholesterol (mg/g tissue)	Free fatty acids (mg/g tissue)
Normal control	56.62±1.82	89.08±1.07	15.95±1.62	5.20±0.07
Obese control	80.43±0.81*	115.87±2.37*	40.56±1.02*	7.67±0.47*
Obese+Lyc (0.2 mg/kg BW)	62.90±0.74*#	93.71±0.54#	28.05±3.00*#	6.40±0.18*#
Obese+Lyc (0.4 mg/kg BW)	47.07±1.63*#	85.14±1.55#	18.66±1.73#	6.16±0.15#
Normal+Lyc (0.2 mg/kg BW)	43.06±1.20*#	85.84±1.07#	18.24±1.32#	5.13±0.15#
Normal+Lyc (0.4 mg/kg BW)	41.83±4.16*#	81.84±0.94*#	16.89±1.11#	5.01±0.08#

Values, which are mean±SEM of five rats, bearing * and/or # are significantly different from normal control and/or obese control, respectively ($p<0.05$). BMI: body mass index; BW: body weight.

upregulated adiponectin levels [30] may be involved in the reduced adipose lipid levels, following treatment with lycopene.

The AC, TC, and the AC/TC ratio were markedly increased in the obese rats compared to normal rats. Increases in body circumference (with no change in height) often accompany weight gain and are good predictors of obesity-associated risks [31]. As obesity progresses (the energy balance becomes more positive and favors storage of energy), more fats are deposited, particularly around the abdominal and thoracic regions [32], and this may account for the increased AC, TC, and the AC/TC ratio. Furthermore, this deposition of fat in the thoracic-abdominal region has been implicated in altered lung volume, airway function, asthma, and other pulmonary complications [32]. A dose-dependent decrease in these body measurements ensued following treatment with lycopene, possibly owing to its ability to reduce weight gain and adiposity (as previously discussed). Besides, the decreased adipocytes' lipid contents following treatment with lycopene, observed in this study, may play a part in the reduction of these body measurements. In line with our results, Sluijs et al. [17] associated increased carotenoid intake with reduced waist circumference and other measures of adiposity in middle-aged and elderly men, accrediting lycopene and β-carotene with the strongest associations.

The BMI, expressed as kg/m^2, is defined as the body weight (in kilograms) divided by the square of the body length (in meters) and is used to measure obesity in humans. However, this index has long since been adapted for animal studies and expressed as g/cm^2. The Lee index, posited as an alternative to BMI in animal studies, is the cube root of body weight (in grams) divided by body length (cm). In both indices, the nose-to-anus length is used as the height or body length [8, 10]. Obesity increased body weight but had no significant effect on body length. Also, the body mass and Lee indices of obese rats were significantly increased following the induction of obesity, as similarly reported by Novelli et al. [8] and Nderitu et al. [33]. Shabbir et al. [10] did not observe any significant difference ($p>0.05$) in rats fed with a high-fat diet, probably due to the different diets and/or strains of rats used. Dose-dependent decreases in these indices were observed following treatment with lycopene, demonstrating its anti-obesity properties, which is line with the reports of Sluijs et al. [17], Fenni et al. [21], and Wang et al. [28].

Nutritional parameters such as daily feed and energy intake, daily weight gain, and feed efficiency were also monitored. The obese rats (fed Western diet) had a higher feed intake than the normal rats (fed control diet), most likely owing to the Western diet being far more palatable than the control diet [3]. Given the higher caloric content of the Western diet (~4.54 kcal/g) compared to the control diet (~3.76 kcal/g), it is therefore not surprising that the obese rats had higher daily energy intake and weight gain, as well as better feeding efficiency than the normal rats fed with the control diet. Although lycopene did not affect feeding efficiency, it was able to reduce feed and energy intake, as well as weight gain. Although, the underplaying mechanisms remain unclear, the antioxidant properties of lycopene have been suggested as a major mechanism underlying their positive effects [17, 34], and some other studies have reported similar results in subjects treated with different sources of antioxidants [35, 36]. These authors postulated that the antioxidant-rich substances they used inhibited weight gain by reducing calorie intake, stimulating fat breakdown, boosting the growth of friendly gut bacteria, while increasing satiety. Increased satiety may explain the reduced feed intake (and subsequently reduced calorie intake), following treatment with lycopene.

Anthropometric measurements occupy a virtually indispensable position when it comes to the evaluation of nutritional status and associated disorders. This present study draws strength from the surprising observation that few studies have attempted to appraise the effects of proposed anti-obesity nutraceuticals on relevant anthropometrical indices of obesity, along with the paucity of information vis-à-vis the possible effects of lycopene after the development of obesity, as against the preventive approach of most other previous studies. A major limitation of this study is that we did not investigate these effects of lycopene at a more biochemical and molecular level, particularly as it

relates to the specific complications associated with obesity. Indeed, the pathophysiology of obesity extends far beyond the bodily measurements assessed in this present study and future studies may seek to address this limitation, while using a larger sample size.

In conclusion, the data presented here illustrate that oral administration of lycopene evoked reductions in indices of obesity, adiposity, and other anthropometric parameters in obese Wistar rats, as well as occasioning reduced feed and energy intake. We, therefore, posit lycopene supplementation as a viable treatment regime for tackling anthropometrical complications associated with obesity.

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