Research Article

Oxidative stress in relation to obesity in Gujrati and non Gujarati young girls before and after maize diet

Sahi N[1], Verma AK[2], Kaur R[3], Jawanda MK[4]

[1]*Dr Nita Sahi*
Professor, Biochemistry
Pacific University
Udaipur,Rajasthan, India

[2]*Dr Ashok Kumar Verma*
Professor & Head, Biochemistry
RNT Medical College
Udaipur, Rajasthan, India

[3]*Dr Roopinder Kaur*
Professor & Head, Pharmacology
GIDSR, Ferozepur, India

[4]*Dr Manveen Kaur Jawanda*
Professor and Head, Oral and Maxillofacial Pathology
Luxmi Bai Institute of Dental Sciences and Hospital
Patiala, Punjab, India

Received: 19-07-2013
Revised: 20-10-2013
Accepted: 10-11-2013

Correspondence to:
Dr Nita Sahi
09414388987
sahi_mukesh1973@yahoo.co.in

ABSTRACT

Background: *Obesity is the first of the "diseases of civilization" to appear. Its prevalence is escalating at an alarming rate. Environmental and behavioral changes brought about by economic development, modernization and urbanization has been linked to the rise in global obesity. Obesity may induce systemic oxidative stress.*

Objective: *The present scenario suggests that higher oxidative stress is the key factor of obesity and hence a management strategy aiming at control of lipid peroxidation in obesity by use of maize diet is envisaged.*

Material and methods: *This study has been conducted on 1001 Gujarati and non Gujarati girls aged between 18-30 years. They were further distributed according to age, inhabitance, socio economic status, dietary habits, family history and blood pressure. Every subject in each group was asked to replace the wheat chapatti by maize chapatti for 30 days; the girls were examined for oxidative stress parameter MDA before and after maize diet along with the statistical evaluation.*

Results: *There was a positive effect of maize diet on biochemical parameter of all the girls in all the subgroups Malondialdehyde level in total girls before the maize diet was 2.35 ± 0.76nmol/ml which reduced to 1.8 ± 0.46nmol/ml after the diet (P<0.001).*

Conclusions: *The oxidative stress showed improvement in normal, overweight and obese girls, most significantly on overweight and obese girls after taking the diet (P<0.001). Current dietetic practice is to recommend a healthy eating plan of reduced fat, and increased fiber intake.*

Keywords: *Obesity, prevalence, oxidative stress, malondialdehyde (MDA), dietary fiber*

Introduction

Obesity is a complex, highly heritable and heterogeneous group of disorders. Free radicals are implicated in various chronic diseases including obesity. [1] Many recant studies have emphasized a role for reactive oxygen radicals in the development of oxidative stress. Epidemiological data suggest an association of dietary intake of nutrients and fibers which are high in antioxidants and protect against the incidence of obesity. The present study was designed to investigate various risk factors and reactive oxygen species mediated changes as etiological factors in normal, overweight and obese Gujarati and Non Gujarati girls before and after maize diet. Free radicals play an important role in path physiology of obesity and they are destroyed by antioxidant defenses. [2] Aging leads to increase in free radical load increase in oxidative stress, scavenging property decreases, and above all biomarkers of oxidative stress accumulate more with age. [3] High income, food consumption patterns, changes in dietary intake and physical inactivity leads to highest prevalence of obesity in upper socioeconomic class. [4] Shifts to

urbanization, non manual labor, high calorie food, sedentary life style and stress leads to three times prevalence of obesity in urban. Non Vegetarians girls consumed high fatty diets, high calorie diets and high glycemic index food along with less use of dietary fiber. [5] Hyperlipidemic diets have double bonds in fatty acids which are vulnerable to oxidation leading to increased lipid peroxidation. Obesity leads to hypertension as secretion of adrenalin is increased, increase in cardiac output, blood volume and arterial resistance is also present, high fat feeding-decrease arachidonic acid path leading to increased peroxidation and production of F2 isoprostanes, inhibition of anti atherogenic vasodilator- Nitric oxide, and increase in vasoconstrictors- superoxide anions, endothelin, linoleic acid, arachidonic acid leads to formation of increased thromboxanes and leukotrienes, [6] Insulin resistance leads to impaired glucose use which results in storage of carbohydrate and fat, and inflammation of tissues increases cytokines production, leptin cause sympathetic activation to kidney resulting in increase arterial pressure. [7] DF modulate glucose response, prevents against carbohydrate induced hypercholesterolemia and hypertriglyceridemia, has sustained energy release-part of energy released in small intestine as glucose (source of energy for brain),part is released in large intestine as SCFA (source in muscles and fat tissue), increases glucose uptake in cells, stimulate action of insulin, slows absorption of carbohydrates by causing delay in gastric emptying, increases glucagon secretion, stimulate gluconeogenesis, divert acetyl Co A to form glucose not cholesterol, protect against atherosclerosis, [8] favourably alters lipid metabolism, decreases TG, LDL, BP, increases HDL, antioxidants, [9] protect against chronic diseases, affect properties like volume, bulk, viscosity, in intestinal lumen, which alters metabolic path of hepatic cholesterol and lipoprotein metabolism [10] and lowers LDL-C -increases fat oxidation, decreases fat storage, fat cell size and its synthesis, change sequence in which body burns food-brings fat on top list, [11] restore normal intestinal functions. [12]

Material and Methods

The present study encompasses anthropometry study as well as clinical study with Maize diet in 1001 Gujarati as well as Non Gujarati girls aged between 18 to 30 years. The practical work was carried out in the Department of Biochemistry, RNT Medical College, Udaipur. The study protocol was approved before the commencement of the study by the Institutional Ethics Committee and all the girls gave their written informed consent.

The girls were residing in different institutional hostels. Actually these girls had come from different districts of Gujarat, Rajasthan mainly and also from other States like Maharashtra, Uttar Pradesh, Madhya Pradesh etc. for study purpose, so they were selected for this study. Interview schedule was developed to collect the general information regarding age, dietary habits, socioeconomic status, suffering from any illness, or taking any treatment, caste, religion etc. The subjects with any clinical or biochemical evidence of liver, kidney or endocrine disease and those on treatment that effect the lipid metabolism were excluded from the study. The categorization of obese subjects were made on the basis of body mass index (BMI) [13] or waist hip ratio (WHR) [14, 15] or standard chart of desirable weight in relation to height, published by

Metropolitan Life Insurance Company (Bray, 1978) [16] as well as on the basis of skin fold thickness. Normal subjects of identical age group with that of respective obese group acted as control. Measurement of different Anthropometric parameters like age, height, weight was done. The most widely used clinical tool for measurement of obesity is the Body Mass Index (BMI) i.e. wt. in Kg / m^2 height

BMI was accurately calculated using SI units Recently the Ministry's Consensus for the prevention and management of obesity and metabolic syndrome for the country has declared that the country's new diagnostic cut off for the body mass index is 23kg/m^2 as opposed to 25kg/m^2 globally [Health Ministry, Diabetes Foundation of India, All India Institute of Medical Sciences, Indian Council of Medical Research, The National Institute of Nutrition, 11/26/2008]. According to them, a person with a body mass index of 23kg/m^2 will now be considered as overweight and below that as one with normal BMI – unlike the cut off limit of 25kg/m^2 earlier. Those with BMI of 25kg/m^2 will be clinically termed obese as opposed to 30kg/m^2 at the international level, and those with BMI of 32.5kg/m^2 will require bariatric surgery to estimate excess flab. Every subject blood pressure was measured with a standardized protocol with an aneroid sphygmomanometer.

Waist Circumference was measured in centimeters at the midpoint between the bottom of the ribs and the top of the iliac crest Women with a waist circumference less than 80cm is considered as normal while with 80 – 87.9 cm were classified as overweight, and women with waist circumference >88 cm were classified as obese (Park 2005) [17]. Recently, the Health Ministry has declared that cut off's for waist circumference will now be 90cm for Indian men as opposed to 102cm globally, and 80cm for Indian women as opposed to 88cm at the international level [17]. Hip Circumference was measured at the largest posterior extension of the buttocks. Waist Hip Ratio was determined by dividing WC by HC. WHR= WC/HC Women with a WHR less than 0.80 was categorized as normal while between 0.80 – 0.84 were classified as overweight and women with a WHR >0.85 were classified as obese. [17] For measurement of Skin fold Thickness [17, 26] all measurements were taken, with the subject seated on a stool, on the right side of the body. The sites selected were biceps, triceps, sub-scapular, supra iliac. At these four sites, the skin fold was pinched up firmly between the thumb and forefinger and pulled away slightly from the underlying tissues before applying the calipers for the measurements. The average of the four sites was taken as skin fold thickness in centimeter.

The instrument used was the Harpenden skin fold caliper. (British Indicators Ltd. St. Albans, Herts.); which exerts a constant pressure at varying openings of the jaws. Total body fat percent was calculated using the following formula as reported by YMCA formula (Young men Christian association). [26] It uses only body weight and waist (at naval) measurements to calculate body fat percentage. [18]

$$\text{Body fat \%} = \frac{-76.76+4.15 \times \text{Waist} - 0.082 \times \text{Weight} \times 100}{\text{Weight}}$$

Body Fat was calculated by multiplying body weight (kg) with body fat percentage. [19] Lean Body Mass (LBM) was obtained by subtracting the body fat (kg) from total body weight. [20]

The venous blood samples were collected by the Standard techniques. Blood was collected in the morning after minimum of 12 hour of overnight fasting.

5 – 6 ml of blood from anticubetal vein was withdrawn in a perfectly clean dry syringe and was transferred to a clean dry centrifuge tube slowly by the side of the tube after removing needle to avoid haemolysis. All the blood samples were taken in the recumbent position. The blood was allowed to clot at room temperature for 30 minutes. The serum was separated by centrifugation at 3000 revolutions per minute (rpm) for 15 minutes. Samples with signs of haemolysis were discarded. Analytical Grade Chemicals and standards were used. The serum was preserved in refrigerator at 4^0 C. MDA was estimated in serum. The present study encompasses clinical study with Maize diet in 1001 Gujarati as well as Non Gujarati girls aged between 18 to 30 years before and after maize diet. Normal subjects of identical age group with that of respective obese group acted as control. The subjects were divided into two groups:

Control group: Possessing normal body weight with healthy body mass index between 18 -25 kg/m^2.

Study Group: Possessing overweight /obesity having body mass index between 25 to 30 kg/m^2.

Every subject in each group was asked to replace the wheat chapatti by maize chapatti and no change was made in the rest of the ingredients. They were asked to take the maize diet for thirty days. Physical and biochemical parameters were determined before and after consumption of the maize diet for thirty days, and, the effect of maize fibers was studied. The difference in the parameters was evaluated when the two communities Gujarati and Non Gujarati were compared with each other before as well as after the maize diet. The study evaluated oxidative stress in obesity and the effect of maize diet on the parameter. The parameter selected to evaluate the oxidative stress was malondialdehyde, an oxidative stress marker; along with the statistical analysis with t and p values.

Thiobarbituric acid reactive substance (TBARS) [1]

Procedure:

To 0.8 ml serum, 1.2 ml of TCA-TBA HCl reagent was added, mixed and kept in boiling water bath for 10 minutes. After cooling, 2 ml of freshly prepared NaOH solution was added so as to eliminate centrifugation. The absorbance of pink color obtained was measured at 535 nm against blank which contained distilled water in place of serum.

Calculation

Molar extinction of TBARS at 535 nm 1.56X 10-5/M/Cm

$$\frac{V \times \Delta OD \text{ at } 535 \text{ nm}}{0.156} \qquad \frac{V \times \Delta OD \text{ at } 535 \text{ nm}}{0.156}$$

= 25.6 x ΔOD at 535 n moles /serum

Normal range = 2.0-3.0 n mole/ml serum

Statistical Analysis

Data was analyzed statistically by using student 't' test with the help of SPSS software version 19.

Results

Table 1(a) shows MDA in total girls (1001), Gujarati girl (526) and non Gujarati girls (475) before and after maize diet. Total girls, Gujarati and NonGujarati girls were statistically evaluated when compared before and after consumption of maize, and also comparison was made for Gujarati v/s NonGujarati before (BMD) as

well as after the maize diet (AMD). As per ICMR recommendations the normal range of malondialdehyde is 1.05-3.2 n moles/ml In the present study, MDA level in total girls before the maize diet was 2.35 ± 0.76nmol/ml which reduced to 1.8 ± 0.46nmol/ml after the diet (P<0.001). Results were almost similar for Gujarati and Non Gujarati girls.

According to age: There is no definite criterion for dividing the different age groups but presently the subjects were divided as less than 20 years and more than 20 years. Malondialdehyde followed an increasing trend with increasing age. The parameter showed significant changes when comparison was done before and after maize diet (P<0.001). When comparison was between Gujarati and Non Gujarati of age <20 group, statistically significant change was observed for malondialdehyde (P<0.05) after the maize diet.

According to Inhabitance: Girls of urban category showed higher values for oxidative stress than those of rural category. Malondialdehyde for both the categories were statistically significant (P<0.001).

According to socio economics class: Keeping in view that obesity is a disorder mainly of affluent class; the girls were divided as lower, middle and upper socio economic class. The oxidative stress for lower socio economic class was less as compared to that in upper socioeconomic class. When fiber diet was considered, the parameter was statistically significant in all three classes (P<0.001).

According to diet: As diet plays an imp role in development of obesity, subjects were divided as vegetarian and non vegetarian. (Table 2a, 2b) The mean values of MDA between the two groups varied and lower values were obtained for vegetarians girls. The effect of maize was almost similar for both the groups in form of significance as (P<0.001).

According to family history of disease: Data obtained for family history of hypertension and that of diabetes were almost similar but family history of coronary Artery Disease had very high values of malondialdehyde in comparison to data of girls who were without any family history malondialdehyde (2.17 ± 0.72nmol/ml v/s 3.15 ± 0.37nmol/ml). However statistically, similar results were seen for the effect of maize diet on the parameter (P < 0.001). (Table 2a, 2b)

According to Blood Pressure: Systolic blood pressure < 120mmHg and diastolic blood pressure < 80mmHg categories showed normal oxidative stress but the high normal category showed high values. SBP < 120mmHg v/s SBP (121-130mmHg) showed mean value of malondialdehyde as 1.81 ± 0.54nmol/ml v/s 2.82 ± 0.58nmol/ml. However effect of maize diet on all categories was almost same. Malondialdehyde was highly significant (P < 0.001) for all categories except high normal diastolic blood pressure category where it was (P<0.05). (Table 2a, 2b)

Table:1(a) Oxidative stress (MDA) (nmol/ml) in Total, Gujrati and Non-Gujrati Girls before and after Maize diet in relation with inhabitance, socioeconomic class, and dietary habits

Parameters	Total Girls B vs A (n=1001)	Gujrati Girls B vs A (n=526)	Non-Gujrati Girls B vs A (n=475)	Guj vs NonGuj Girls BMD (526vs475)	Guj vs Non-Guj. Girls AMD (526vs475)
MDA (nmol/ml)	19.59*	13.17*	14.35*	0.83	0.69
Rural	Total Girls B vs A (n=242/1001)	Guj.Girls B vs A (n=69/242)	Non Gujrati Girls B vs A (n=173/242)	Guj. vs Non Guj BMD (69vs173)	Guj. vs Non Guj AMD (69vs173)
MDA (nmol/ml)	9.77*	4.9*	8.47*	0.1	0.78
Urban	Total Girls B vs A (n=759/1001)	Guj.Girls B vs A (n=457/759)	Non Guj.Girls B vs A (n=302/759)	Guj vs NonGuj BMD (457vs302)	Guj vs NonGuj AMD (457vs302)
MDA (nmol/ml)	17.01*	12.35*	11.81*	0.74	0
Lower class	Total Girls B vs A (n=194/1001)	Guj. Girls B vs A (n=64/194)	Non Guj. Girls B vs A (n=130/194)	Guj vs NonGuj BMD (64vs130)	Guj vs Non Guj AMD (64vs130)
MDA (nmol/ml)	8.34*	4.32*	7.18*	0.96	0.43
Middle class	Total Girls B vs A (n=447/1001)	Guj. Girls B vs A (n=183/447)	Non Guj Girls B vs A (n=264/447)	Guj vs NonGuj BMD (183vs264)	Guj vs NonGuj AMD (183vs264)
MDA (nmol/ml)	13.22*	8.04*	10.54*	0.41	1.34
Upper class	Total Girls B vs A (n=360/1001)	Guj Girls B vs A (n=279/360)	Non Guj Girls B vs A (n=81/360)	Guj vs NonGuj BMD (279vs81)	Guj vsNonGuj AMD (279vs81)
MDA (nmol/ml)	11.84*	9.7*	7.15*	2.05*	-0.57
Vegetarian	Total Girls B vs A (n=721/1001)	Guj Girls B vs A (n=404/721)	Non Guj. Girls B vs A (n=317/721)	Guj vsNonGuj BMD (404vs317)	Guj vsNonGuj AMD (404vs317)
MDA (nmol/ml)	16.21*	11.39*	11.64*	-0.18	1.45

Table: 2(a) Oxidative stress in Total, Gujrati and Non-Gujrati girls before and after Maize diet in relation with diet, family history, and blood pressure

Non vegetarian	Total Girls (n=280/1001)		Gujrati Girls (n= 122/280)		Non Gujrati Girls (n= 158/280)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	2.39±0.76	1.81±0.44	2.34±0.76	1.79±0.46	2.43±0.76	1.82±0.43
FH of BP	Total Girls (n=100/1001)		Gujrati Girls (n=29/100)		Non Gujrati Girls (n= 71/100)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	2.91±0.53	2.1±0.3	3.01±0.52	2.17±0.29	2.87±0.53	2.07±0.3
FH-CAD	Total Girls (n=68/1001)		Gujrati Girls (n= 22/68)		Non Gujrati Girls (n=46/68)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	3.15±0.37	2.22±0.21	3.27±0.12	2.29±0.11	3.09±0.43	2.18±0.24
FH-DM	Total Girls (n= 51/1001)		Gujrati Girls (n= 28/51)		Non Gujrati Girls (n=23/51)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	2.91±0.58	2.11±0.32	2.99±0.47	2.16±0.23	2.81±0.69	2.05±0.39
SBP<120	Total Girls (n=466/1001)		Gujrati Girls (n= 253/466)		Non Gujrati Girls (n=213/466)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	1.81±0.54	1.49±0.37	1.77±0.52	1.48±0.38	1.86±0.56	1.5±0.36
DBP<80	Total Girls (n=543/1001)		Gujrati Girls (n=289/543)		Non Gujrati Girls (n= 254/543)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	1.92±0.62	1.56±0.41	1.92±0.64	1.57±0.44	1.92±0.6	1.55±0.38
SBP-121-139	Total Girls (n=535/1001)		Gujrati Girls (n=273/535)		Non Gujrati Girls (n=262/535)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	2.82±0.58	2.07±0.33	2.85±0.55	2.12±0.31	2.79±0.62	2.03±0.34
DBP-81-90	Total Girls (n= 458/1001)		Gujrati Girls (n=237/458)		Non Gujrati Girls (n=221/458)	
	Before	After	Before	After	Before	After
MDA (nmol/ml)	2.86±0.56	2.09±0.32	2.83±0.57	2.1±0.33	2.88±0.55	2.07±0.3

Table: 2(b) Oxidative stress in Total, Gujrati and Non-Gujrati Girls before and after Maize diet in relation with diet, family history, and blood pressure

Non vegetarian	Total Girls B vs A (n=280/1001)	Guj Girls B vs A (n=122/280)	Non Guj Girls B vs A (n=158/280)	Guj vs NonGuj BMD (122vs158)	Guj vs NonGuj AMD (122vs158)
MDA (nmol/ml)	11.05*	6.87*	8.69*	-0.98	-0.56
FH of BP	Total Girls B vs A (n=100/1001)	Guj. Girls B vs A (n=29/100)	Non Guj. Girls B vs A (n=71/100)	Gu vs Non Guj BMD (29vs71)	Guj vs Non Guj AMD (29vs71)
MDA (nmol/ml)	13.28*	7.58*	11.06*	1.21	1.55
FH-CAD	Total Girls B vs A (n=68/1001)	Guj. Girls B vs A (n=22/68)	Non Guj. Girls B vs A (n=46/68)	Guj vs Non Guj BMD (22vs46)	Guj vs Non Guj AMD (22vs46)
MDA (nmol/ml)	18.1*	28.17*	12.63*	2.63*	2.59*
FH-DM	Total Girls B vs A (n=51/1001)	Guj. Girls B vs A (n=28/51)	Non Guj. Girls B vs A (n=23/51)	Guj vs Non Guj BMD (28vs23)	Guj vs Non Guj AMD (28vs23)
MDA (nmol/ml)	8.62*	8.48*	4.63*	1.06	1.19
SBP<120	Total Girls B vs A (n=466/1001)	Guj Girls B vs A (n=253/466)	Non Guj Girls B vs A (n=213/466)	Guj vs Non Guj BMD (253vs213)	Guj vs Non Guj AMD (253vs213)
MDA (nmol/ml)	10.46*	7.05*	7.8*	-1.79	-0.58
DBP<80	Total Girls B vs A (n=543/1001)	Guj. Girls B vs A (n=289/543)	Non Guj Girls B vs A (n=254/543)	Guj vs Non Guj BMD (289vs254)	Guj vs Non Guj AMD (289vs254)
MDA (nmol/ml)	11.16*	7.54*	8.34*	0	0.57
SBP-121-139	Total Girls B vs A (n=535/1001)	Guj Girls B vs A (n=273/535)	Non Guj Girls B vs A (n=262/535)	Guj vs Non Guj BMD (273vs262)	Guj vs Non Guj AMD (273vs262)
MDA (nmol/ml)	25.77*	19.28*	17.43*	1.18	3.20*
DBP-81-90	Total Girls B vs A (n=458/1001)	Guj Girls B vs A (n=237/458)	Non Guj Girls B vs A (n=221/458)	Guj vs Non Guj BMD (237vs221)	Guj vs Non Guj AMD (237vs221)
MDA (nmol/ml)	25.69*	17.14*	19.36*	-0.96	1.02

Discussion

According to Reddy KK et al [2] (1997) severe obesity is associated with lipid per oxidation. Obesity increases the mechanical and metabolic loads on the myocardium, thus increasing myocardial oxygen consumption. [21] A negative consequence of this is the production of reactive oxygen species such as superoxide, hydroxyl radical, and hydrogen peroxides from the increased mitochondrial respiration. If the production of these oxygen species exceeds the antioxidant capacity of the cell, oxidative stress resulting in lipid per oxidation may occur. The second mechanism by which obesity can independently cause lipid per oxidation is by progressive and cumulative cell injury resulting from pressure of large body mass. Cell injury causes the release of cytokines specially tumor necrosis factor alpha, which generates reactive oxygen species from the tissues which in turn cause lipid per oxidation. A third possible mechanism is through diet. Nutritional

obesity which is the predominant form in our study subjects implies the consumption of hyperlipidemic diets which may be involved in oxygen metabolism. Double bonds in the fatty acid molecules are vulnerable to oxidation reactions and consequently may cause lipid per oxidation. [3, 22]

Studies indicate that serum lipid peroxide levels tend to increase with age. During aging, oxidative stress and free radical load increase and their scavenging activity get decreased. Almost all biomarkers of oxidative stress have been found to accumulate with age. P-MDA showed higher values although in acceptable range for girls with age more than 20 years in comparison to that in age category of less than 20 years (2.39±0.77 nmol/ml v/s 2.31±0.74 nmol/ml); The increase in levels of MDA indicates that lipid per oxidation was increased because of obesity. This is in accordance to various studies. [2, 3, 5] The causes of increase lipids and lipid peroxides were due to alteration of functions of erythrocyte membrane. This inhibits the activity of antioxidant enzymes leading to accumulation of radicals which causes maximum lipid per oxidation and tissue damage in obesity. P-MDA values were directly related to socioeconomic status, the lower class had minimum oxidative stress (2.29±0.75 nmol/ml). The middle and upper class had high oxidative stress (2.36±0.75 nmol/ml). According to inhabitance rural girls had reasonably acceptable value of MDA (2.31±0.73 nmol/ml) while urban girls had higher level of the same (2.36±0.77 nmol/ml). MDA was also directly proportional to blood pressure. As the blood pressure increased MDA level also increased. (1.81±0.54 nmol/ml v/s 2.82±0.58 nmol/ml) The study was also in accordance to report by Bjorntop et al. [6] Reddy et al [2] who reported that significant increase in plasma lipid peroxides, free radical generation, MDA levels were obtained in urban inhabitants compared to rural. Indirectly, indication is that the changes of LDL oxidation were higher in obese state and could be one of the etiological factors in obesity. [23] These presumptions are supported well by reports of other workers like Olusi et al. [3] Obesity induces systemic oxidative stress and increase oxidative stress in accumulated fat is one of the underlying causes of dysregulation of adipocytokines and development of metabolic syndrome and plays critical role in pathogenesis of various diseases. [7, 8, 24] Thus our study, in general, correlates with most of the available reports.

Oxidative stress marker-MDA was well within acceptable range as per ATP III guidelines in total, Gujarati and Non Gujarati girls. MDA increased with age. In total girls the average level of MDA for age<20 years was 2.31n mole/ml of serum while for age >20 years it was 2.39n mole/ml of serum. The same observation was seen in Gujarati girls (2.27v/s 2.37). Oxidative stress was maximum for middle and upper socio economic class when compared with lower class; Rural inhabitants showed less oxidative stress as compared to urban inhabitants (2.31nmol/ml of serum v/s 2.36nmol/ml of serum). Non vegetarian category showed high oxidative stress (2.39nmol/ml of serum v/s 2.33nmol/ml of serum) Oxidative stress was maximum for girls with family history of CAD along with total cholesterol as compared to the respective values for the other family history of diseases. The increasing trend of MDA was FH CAD>FH HT = FH DM > without FH; FH CAD>FH HT > FH DM > without FH and that of TAA was FH CAD <FH HT < FH DM < without FH. Effect of maize diet was quite evident and almost equal (P<0.001) on the parameter in total, Gujarati and Non Gujarati girls. The

oxidative stress showed improvement in normal, overweight and obese girls, most significantly on overweight and obese girls after taking the diet although the MDA levels were high but in acceptable range before the diet was taken. MDA levels were significantly reduced after the diet (P<0.001) When matched for age, MDA was significantly decreased for both the groups after the diet. When the two groups were compared with each other MDA was statistically significant (P<0.001). Effect of maize was seen for all socio economic classes. Highly significant changes were observed for almost all parameters in rural and urban categories for oxidative stress when matched for diet (P<0.001). For the vegetarian group of girls MDA level improved (P<0.001). MDA was significantly changed in the comparative analysis in family history of hypertension and coronary artery disease before as well as after the diet. [10] The girls of high normal category of blood pressure were more significantly affected by the diet which was evident by their improved MDA profile. [26]

Referances

1. Buege JA, Aust SD. The thiobarbituric acid assay. Methods Enzymol 1978;52:306.
2. Reddy KK, Ramamurty R, Papa Rao. Free radical and antioxidant status in urban and rural Tirupati men. Asia Pacific J Clin nutr 1997;6:296-311.
3. Olusi SO. Obesity is an independent risk factor for plasma lipid peroxidation and depletion of erythrocyte cytoprotective enzymes in humans. Obesity 2002;26:1159-64.
4. Sharma JP, Rastogi V, Niaz MA. Socioeconomic status and obesity. Eur Heart J 1997;18:588-95.
5. Peiris AN, Thakur RK. Relationship of regional fat distribution and obesity. South Med J 1991;84:961–5.
6. Bjorntorp P. Classification of obese patients and complications related to the distribution of surplus fat. Nutrition 1990;6:131–7.
7. Singh SP, Sikri G, Garg MK. Body mass index and obesity. Tailoring cut off for an Asian Indian male population. MJAFI 2008;64:350-3.
8. Sparks JD, Sparks CE. Lipoprotein synthesis and secretion. Biochimica Biophysica Acta 1994;1215:9-32.
9. Trowell, Tsai AW. Cardiovascular risk factors. Arch Intern Med 1972;162:1182–9.
10. Tungland BC, Meyer D. Nondigestible oligo and polysaccharides, their role in human health. Food Science 2002;173-89.
11. Van Itallie TB. Dietary fiber and obesity. American Journal of Clinical Nutrition 1978;31:S43-52.
12. Zannad F, Gille B, Grentzinger A. Weight reduction. Am Heart J 2002;144:508–15.
13. Arcaro G, Zamboni M, Rossi L, Turcato E, Covi G, Armellini F, Lechi A et al. Body fat distribution. Int J Obes Relat Metab Disord 1999;23:936–42.
14. Ardern CI, Janssen I, Ross R, Katzmarzyk PT. Development of waist circumferences thresholds within BMI categories. Obes Res 2004;12:1094.
15. Barzilai N, Gupta G. Pathophysiology of fat distribution. Ann N Y Acad Sci 1999; 892:58-72.
16. Bray GA. Nutrient balance and obesity. Med Clin North Am 1989;73.
17. Park YW, Zhu S, Palaniappan L, Heshka S, Carnethon MR, Heymsfield SB. The metabolic syndrome. Arch Intern Med 2003;163:427–36.
18. Pradeep Kumar, Vikas K, Kosambia JK. Prevalence of hypertension among the employs of south Gujarat. Indian Journal of Community Medicine 2002;27:19-22.

19. Perry AC, Applegate EB, Allison ML, Miller PC, Signorile JF. Relation between anthropometric measurements of fat distribution in overweight and obese females. Am J Clin Nutr 1997;66:829-36.
20. Pi-Sunyer FX, Aronne LJ, Heshmati HM. Weight measurements. JAMA 2006;295: 761-75.
21. Simin Liu. Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle aged women. Am J Clin Nutr 2003;78:920-7.
22. Slavin J. Dietary fiber and body weight. Nutrition 2009;21:411-18.
23. Voors AW, Webber LS, Frerichs RR, Berenson GS. Body height and body mass. Am J Epidemiol 1997;106:101–8.
24. Sharma JP, Rastogi V, Niaz M. Socioeconomic status and obesity. Eur Heart J 1997;18:588-95.
25. Durnin JV, Rahaman MM. The assessment of the amount of fat in the human body from measurements of skin fold thickness. Br J Nutr 1967;21:681.
26. Nora E, Straznicky, Christopher J, Vicki E, Wilkliams J. Obesity 2008;34:580.

Cite this article as: Sahi N, Verma AK, Kaur R, Jawanda MK. Oxidative stress in relation to obesity in Gujrati and non Gujarati young girls before and after maize diet. Int J Med and Dent Sci 2014; 3(1): 314-324.

Source of Support: Nil
Conflict of Interest: No