CHAPTER 5
RESULTS AND DISCUSSION

The results of the present study entitled **"Sensory evaluation of fructooligosaccharide (FOS) added popular recipes of India and its role in modulating anthropometric indices, gut flora and lipopolysaccharide (LPS) in obese young adults of urban Vadodara"** are presented, discussed and interpreted in this chapter. These results are presented in to four main phases according to the objectives of the study.

Phase I Development and standardisation of fructooligosaccharide (FOS) incorporated popular recipes of India and studying their various organoleptic attributes and overall acceptability.

Phase II Situational analysis: mapping the prevalence of obesity and hypertension in banks employees of urban Vadodara (A cross-sectional design).

Phase III Comparison of grade-I obese subjects with non-obese subjects in terms of anthropometry profile, medical history, family history of diseases, defecation profile, hunger and satiety, psychological depression status, dependency on habits, dietary intakes, biophysical profile, lipemic profile, enotoxemia and gut microbiota (*LAB, bifidobacteria, bacteroides and clostridium*) and to understand the correlations between various parameters.

Phase IV Effect of fructooligosaccharide (FOS) supplementation on anthropometry profile, blood pressure, defecation profile, hunger and satiety, psychological depression, dietary intakes, lipemic parameters, plasma LPS level and gut microbiota (*LAB, bifidobacteria, bacteroides and clostridium*) in obese grade-I adults.

Phase I Development and standardisation of fructooligosaccharide (FOS) incorporated popular recipes of India and studying their various organoleptic attributes and overall acceptability.

Diet has an impact on gut health. Functional foods are indistinguishable compared to conventional foods; and can be consumed as part of the normal diet. Among these, FOS (oligofructose or oligofructan) has come into sight in response to consumer demand for low calorie food. It is one of the functional food ingredient which is not yet well exploited and consumed by Indian population, therefore, FOS added recipes need to be developed and studied for the feasibility of its addition and their acceptability so as to expand the data base of FOS added Indian recipes.

This phase of the research work was undertaken to study the acceptability trials of selected FOS incorporated food products *viz.* ***Lilva kachori, Vegetable parantha, Rawa idli*** and ***Chocolate cake*** at varying levels of addition. The selection of recipes was primarily based on various cooking methods *viz. deep frying, shallow frying, steaming and baking* and secondly on popularity.

For this phase, above selected four food products for FOS addition/substitution were assessed for their organoleptic properties. Since these food products are commonly consumed in India, they were judged as a vehicle for FOS addition/substitution. Filling material of *lilva kachori and vegetable parantha* were added with FOS at three (5g, 10g, and 15g) and two levels (10g and 15g) respectively and in case of *rawa idli* base material (semolina) was added with FOS at three levels (10g, 15g, and 20g). In *chocolate cake* FOS added at three levels (10g, 20g, and 30g). The results presented are an average of triplicate analysis of all samples for their sensory attributes.

The results of this phase are explained into following sections-

Section 5.0 Effect of addition of FOS at varying levels in the filling material of *lilva kachori*

Section 5.1 Effect of addition of FOS at varying levels in the filling material of *vegetable parantha*

Section 5.2 Effect of addition of FOS at varying levels in *rawa idli*

Section 5.3 Effect of addition of FOS in *chocolate cake* at varying levels

Section 5.0 Effect of addition of FOS at varying levels in the filling material of *lilva kachori*

a) Organoleptic evaluation of the *lilva kachori*

The organoleptic scores of *lilva kachori* prepared by adding filling material with varying levels of FOS are presented graphically in Figure 5.1 (a-f) and tabulated in Table 5.1.

i) **Color and Appearance**: The most affected attribute was *color and appearance* with 18.7 percent reduction in the scores as the levels of addition increased ($p<0.001$). However, even upon addition of 5g FOS to the filling material, the burn spots on the surface of kachori increased after frying which were constantly increased as the concentration of FOS amplified.

ii) **Mouthfeel**: There was significant reduction (14.1%) ($p<0.001$) in the scores as the addition of FOS increased. However, at 5g of FOS addition, mouthfeel remained unaltered.

iii) **Texture**: The scores for *texture* of the kachori significantly ($p<0.001$) reduced by 17.4% with increased levels of FOS addition. It became soft and soggy giving undesirable mouthfeel with increased fluidity of the stuffing of kachori. Although, there was no significant difference found between standard and at 5g of addition level and similarly between at 10g and 15g addition levels.

iv) **Taste**: *Taste* of kachori did not show any significant change from standard to upto 5g FOS addition, however, a significant reduction was seen by 16.6% with the increased level of FOS addition upto 15g. Instead of spicy taste of kachori there was an increase in sweetness, which is not preferred normally.

v) **After taste:** The mean scores for after taste were continued to decrease as the levels of FOS addition increased. Mean scores ranged from 7 (at 5g level) to 5.85 (at 15g level) as against 7.11 scored by standard recipe.

vi) **Overall acceptability:** OA scores of lilva kachori were comparable to standard upto 5g of addition of FOS. There after OA scores significantly decreased upto 15g of addition of FOS. The overall reduction in most of the sensory attributes of lilva kachori ranged from 14 percent to 18 percent.

Table 5.1: Effect of varying levels of FOS addition on the organoleptic qualities of *lilva kachori*

Levels of FOS addition		Organoleptic attributes					
		Color and Appearance	Mouthfeel	Texture	Taste	After Taste	Overall Acceptability
Standard	Mean	7.52^{a}	7.09^{a}	7.23^{a}	7.14^{a}	7.11^{a}	7.40^{a}
	±SD	±1.13	±1.14	±0.95	±1.11	±1.17	±1.03
5g	Mean	7.16^{b}	7.11^{a}	6.95^{a}	7.09^{a}	7^{a}	7^{a}
	±SD	±1.14	±1.10	±1.12	±1.03	±1.10	±1.24
10g	Mean	6.76^{c}	6.59^{b}	6.23^{bd}	6.54^{b}	6.28^{b}	6.33^{bd}
	±SD	±1.18	±1.17	±1.12	±1.13	±1.21	±1.05
15g	Mean	6.11^{d}	6.09^{c}	5.97^{cd}	5.95^{c}	5.85^{c}	6.14^{cd}
	±SD	±1.13	±1.031	±1.37	±1.14	±1.11	±1.22
	% decrease	**$18.7^{\#}\downarrow$**	**$14.1^{\#}\downarrow$**	**$17.4^{\#}\downarrow$**	**$16.6^{\#}\downarrow$**	**$17.7^{\#}\downarrow$**	**$17.0^{\#}\downarrow$**
ANOVA		**11.55^{***}**	**7.95^{***}**	**11.06^{***}**	**10.65^{***}**	**11.31^{***}**	**11.01^{***}**

- Note: Mean values represent the average of 25 determinants in triplicates.
- a, b, c – The non-identical letters in any two rows within the column denote a significant difference at a minimum of 5% level.
- NS – The difference between the mean values within the columns is not significant.
- Maximum score for all the organoleptic attributes was 10.
- Level of significance in increasing order- (* $p<0.05$, ** $p<0.01$, *** $p<0.001$)
- #: Percent decrease in standard to 15 g of FOS addition

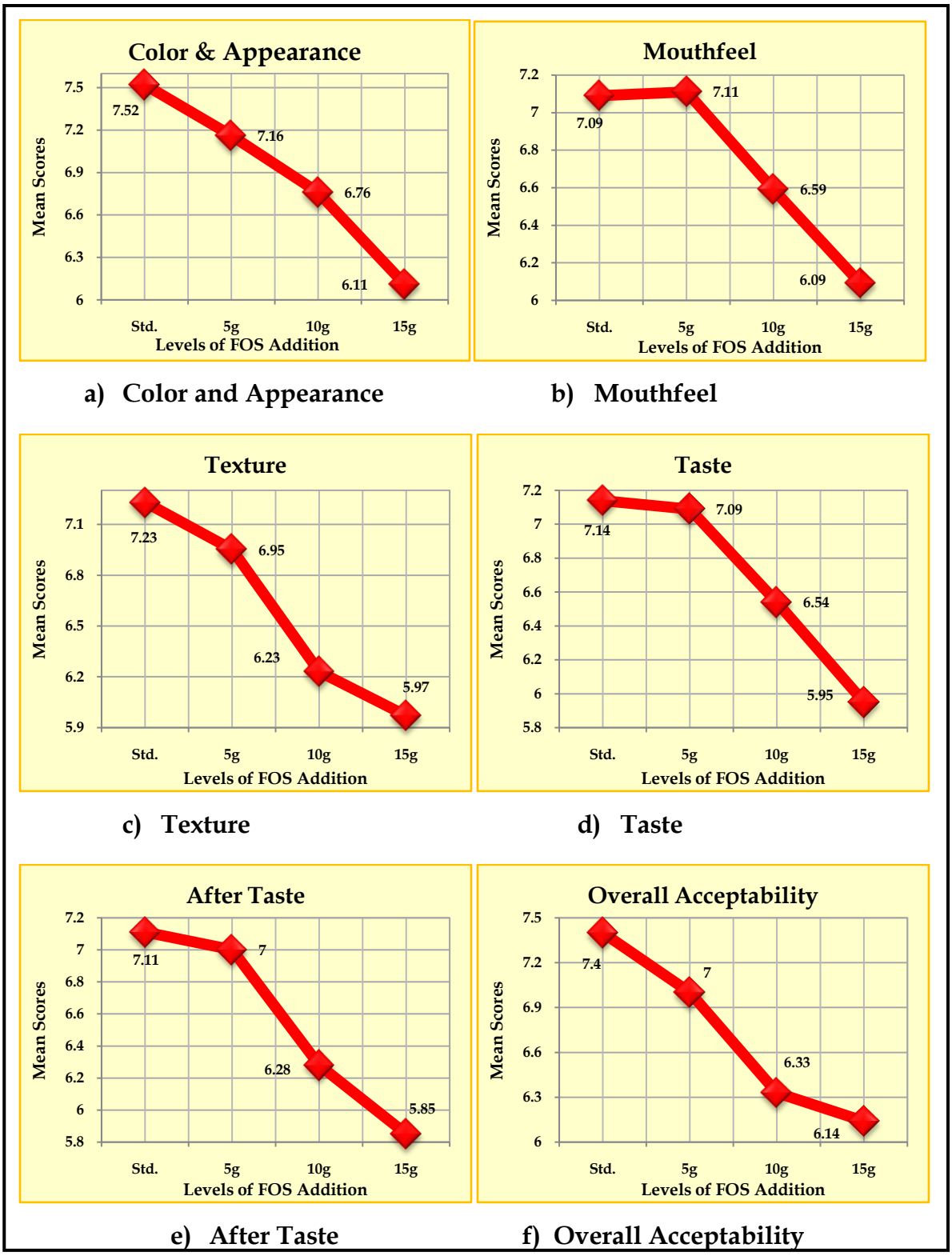


Fig. 5.1 (a-f): Scores for organoleptic attributes of *lilva kachori* added with varying levels of FOS

Section 5.1 Effect of addition of FOS at varying levels in the filling material of *vegetable parantha*

a) Organoleptic evaluation of the *vegetable parantha*

The organoleptic scores of *vegetable parantha* prepared by adding filling material with varying levels of FOS are presented graphically in Figure 5.2 (a-f) and tabulated in Table 5.2.

i) **Color and Appearance**: There was no significant difference in *color and appearance* scores of parantha with increased levels of FOS addition. Although, stickiness and black and brown burn spots on the surface of parantha increased slightly as the concentration of FOS increased resulting in non-significant reduction in scores by 5.5%.

ii) **Mouthfeel**: Mouthfeel remained unaltered as the level of FOS addition increased.

iii) **Texture**: The scores for *texture* of the parantha significantly ($p<0.05$) reduced by 8.6% with increased levels of FOS addition. It became sticky giving undesirable mouthfeel with increased fluidity of the stuffing of parantha.

iv) **Taste**: *Taste* of parantha showed no significant changes at both 10g and 15g FOS addition.

v) **After taste:** The mean scores for after taste were not significantly different as the levels of FOS addition increased from 10g to 15g. Mean scores ranged from 6.90 (at 10g level) to 6.97 (at 15g level) as against 6.90 scored by standard recipe.

vi) **Overall acceptability:** OA Scores were unaffected because there was not much variation in the scores of the various sensory attributes. The overall reduction in most of the sensory attributes of vegetable parantha ranged from 8.6% (texture) to 0.78% (taste).

Table 5.2: Effect of varying levels of FOS addition on the organoleptic qualities of *vegetable parantha*

Levels of FOS addition		Organoleptic attributes: Color and Appearance	Mouthfeel	Texture	Taste	After Taste	Overall Acceptability
Standard	Mean	7.45^{NS}	7.04^{NS}	7.40^{a}	7.09^{NS}	6.90^{NS}	7.14^{NS}
	±SD	±0.94	±0.90	±0.96	±1.07	±1.14	±1.00
10g	Mean	7.21^{NS}	6.95^{NS}	6.85^{b}	7.07^{NS}	6.90^{NS}	6.97^{NS}
	±SD	±1.11	±1.08	±1.33	±1.15	±1.00	±1.13
15g	Mean	7.04^{NS}	6.92^{NS}	6.76^{ab}	7.04^{NS}	6.97^{NS}	7.14^{NS}
	±SD	±1.32	±1.29	±1.39	±1.24	±1.19	±1.24
	% decrease	**5.5$^{\#}$ ↓**	**1.7$^{\#}$ ↓**	**8.6$^{\#}$ ↓**	**0.78$^{\#}$ ↓**	**1.0#↓**	**No change$^{\#}$**
ANOVA		**1.34^{NS}**	**0.13^{NS}**	**3.25***	**0.01 ^{NS}**	**0.05 ^{NS}**	**0.30 ^{NS}**

- Note: Mean values represent the average of 25 determinants in triplicates.
- a, b, c – The non-identical letters in any two rows within the column denote a significant difference at a minimum of 5% level.
- NS – The difference between the mean values within the columns is not significant.
- Maximum score for all the organoleptic attributes was 10.
- Level of significance in increasing order- (* $p<0.05$, ** $p<0.01$, *** $p<0.001$)
- #: Percent decrease in standard to 15 g of FOS addition

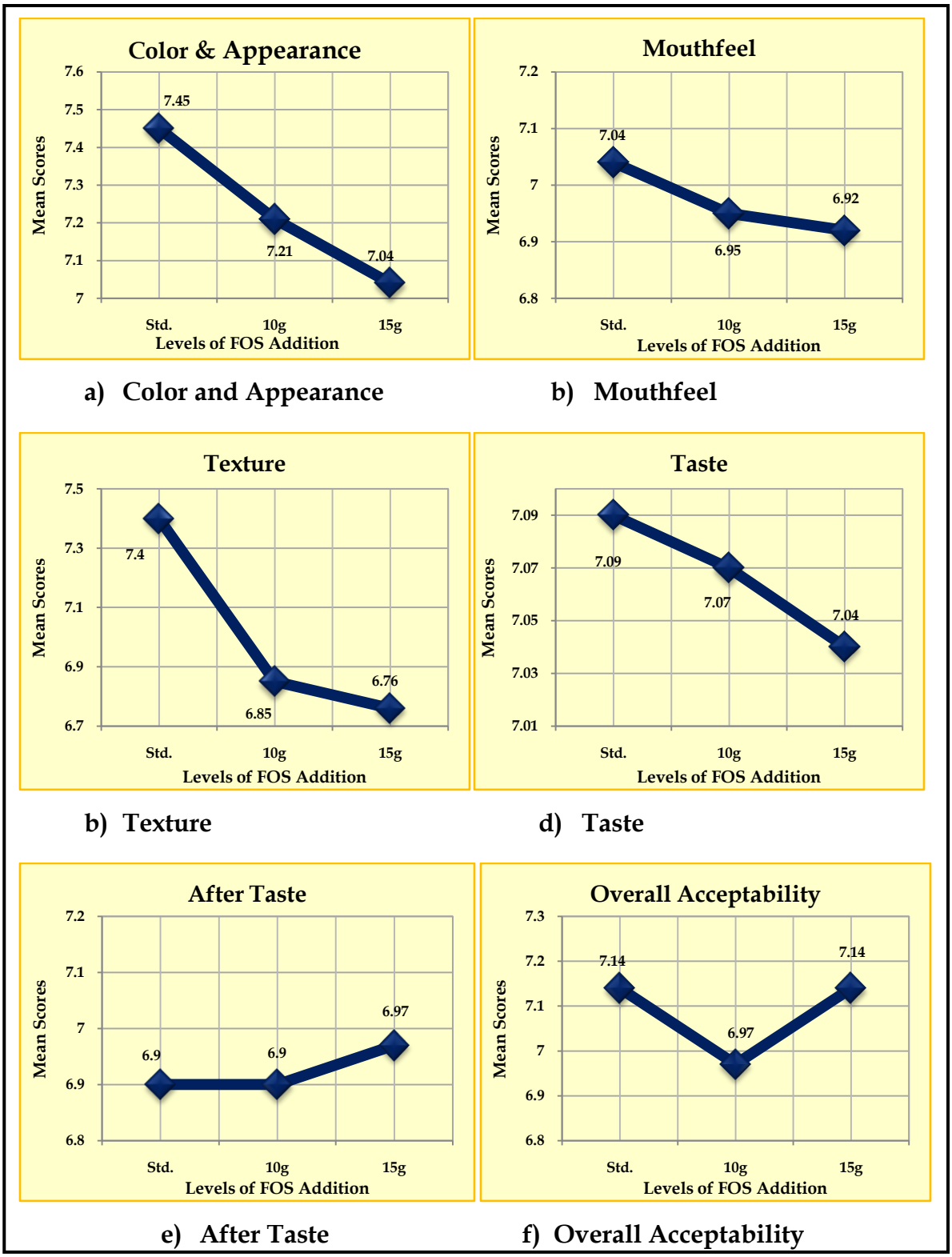


Fig. 5.2 (a-f): Scores for organoleptic attributes of *vegetable parantha* added with varying levels of FOS

Section 5.2 Effect of addition of FOS at varying levels in *rawa idli*

a) Organoleptic evaluation of the *rawa idli*

The organoleptic scores of *rawa idli* prepared by adding idli batter with varying levels of FOS are presented graphically in Figure 5.2.1 (a-f) and tabulated in Table 5.3.

i) **Color and Appearance**: At all the levels of FOS addition the *color and appearance* scores showed no significant changes. However, the scores continued to increase by 5.47% as the levels of FOS addition increased.

ii) **Mouthfeel**: There was non-significant rise in the scores of mouthfeel as the addition of FOS increased. The scores increased by 6.06% at 20 g of FOS addition against standard recipe.

iii) **Texture**: The scores for *texture* of the *rawa idli* significantly ($p<0.01$) improved by 8.95% with increased levels of FOS upto 20g level addition.

iv) **Taste**: *Taste* of rawa idli improved from standard rawa idli by 7.46% although it was non-significant.

v) **After taste:** The mean scores for after taste continued to increase as the levels of FOS addition increased upto 15 g, after that a non-significant reduction was observed by 2.81% at 20g addition of FOS.

vi) **Overall acceptability:** Scores of *rawa idli* significantly improved because of improved scores of the various sensory attributes. The overall scores increased by 8.95% against standard recipe. Fig. 5.2.1 revealed that the most acceptable idli was with 15 g of FOS addition after that all the scores reduced non-significantly.

Table 5.3 : Effect of varying levels of FOS addition on the organoleptic qualities of *rawa idli*

Levels of FOS addition		Organoleptic attributes: Color and Appearance	Mouthfeel	Texture	Taste	After Taste	Overall Acceptability
Standard	Mean	7.3^{NS}	6.6^{NS}	6.7^{a}	6.7^{NS}	6.6^{NS}	6.7^{a}
	±SD	±1.05	±1.39	±1.49	±1.36	±1.39	±1.23
10g	Mean	7.6^{NS}	7.1^{NS}	7.2^{bd}	7.1^{NS}	6.8^{NS}	7.3^{b}
	±SD	±1.16	±1.19	±1.10	±1.01	±1.19	±0.98
15g	Mean	7.7^{NS}	7.2^{NS}	7.7^{c}	7.2^{NS}	7.1^{NS}	7.4^{cbd}
	±SD	±0.96	±1.12	±0.92	±1.23	±1.26	±1.25
20g	Mean	7.7^{NS}	7.0^{NS}	7.3^{d}	7.2^{NS}	6.9^{NS}	7.3^{dbc}
	±SD	±0.71	±1.09	±0.95	±1.18	±1.16	±1.06
	% increase	**$5.47^{\#}\uparrow$**	**$6.06^{\#}\uparrow$**	**$8.95^{\#}\uparrow$**	**$7.46^{\#}\uparrow$**	**$4.54^{\#}\uparrow$**	**$8.95^{\#}\uparrow$**
ANOVA		**1.41^{NS}**	**1.64^{NS}**	**5.32^{**}**	**1.30^{NS}**	**1.11^{NS}**	**3.01^{*}**

- Note: Mean values represent the average of 25 determinants in triplicates.
- a, b, c – The non-identical letters in any two rows within the column denote a significant difference at a minimum of 5% level.
- NS – The difference between the mean values within the columns is not significant.
- Maximum score for all the organoleptic attributes was 10.
- Level of significance in increasing order- (* $p<0.05$, ** $p<0.01$, *** $p<0.001$)
- #: Percent increase in standard to 20 g of FOS addition

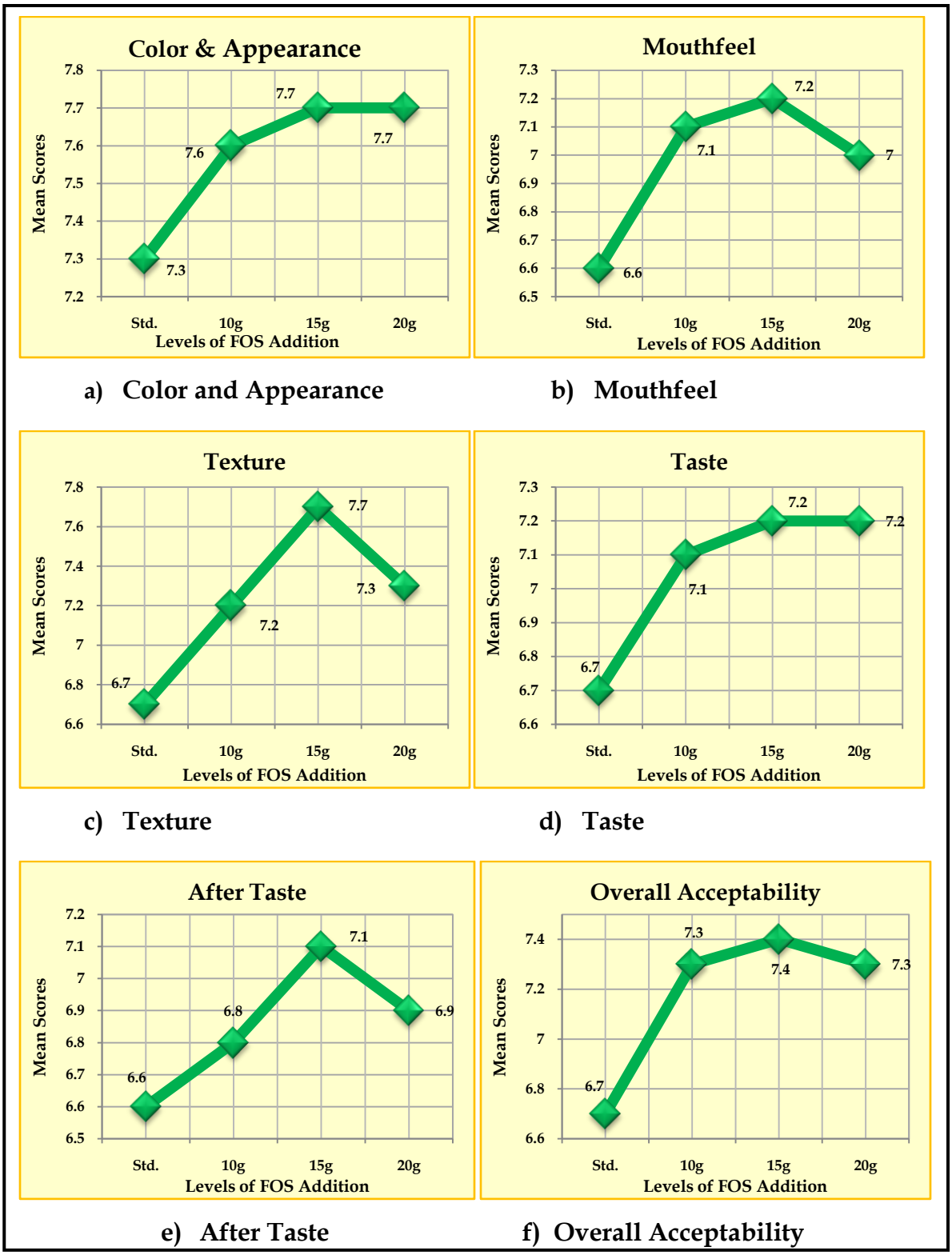


Fig. 5.3 (a-f): Scores for organoleptic attributes of *rawa idli* added with varying levels of FOS

Section 5.3 Effect of addition of FOS in *chocolate cake* at varying levels

a) Organoleptic evaluation of the *chocolate cake*

The organoleptic scores of *chocolate cake,* prepared by adding FOS at varying levels are presented diagrammatically in Figure 5.4 (a-f) and tabulated in Table 5.4.

i) **Color and Appearance**: Upto addition with 20 g of FOS, *color and appearance* scores continued to increase. After that a non-significant reduction in scores was observed by 2.61%. Though, at higher level of substitution *chocolate cake* became darker in color against standard recipe, this change was non-significant.

ii) **Mouthfeel**: In line with color and appearance scores *mouthfeel* scores non-significantly rose in cake added with upto 20g of FOS. Although this improvement was non-significant.

iii) **Texture**: The scores for *texture* of the *chocolate cake* non-significantly improved by 5.92% added with upto 20g of FOS. However, the highest scores was 7.69 at 20g of addition level, after that the scores reduced non-significantly to 7.33 at 30g of addition level.

iv) **Taste**: *Taste* of chocolate cake remained similar to the standard recipe upto 30g of addition of FOS.

v) **After taste:** No significant difference was noticed between all the FOS added *chocolate cake* when compared with standard *cake*.

vi) **Overall acceptability:** OA scores remained unchanged upto 30g addition of FOS as compared to standard recipe. Though, the overall scores reduced by 4.17% in the *cake* added with 30 g FOS against standard recipe. (Fig. 5.3.1)

Table 5.4: Effect of varying levels of FOS addition on the organoleptic qualities of *chocolate cake*

Levels of FOS substitution		Organoleptic attributes					
		Color and Appearance	Mouthfeel	Texture	Taste	After Taste	Overall Acceptability
Standard	Mean	7.90^{NS}	7.33^{NS}	7.26^{NS}	7.24^{NS}	7.00^{NS}	7.43^{NS}
	±SD	±0.76	±1.00	±1.11	±1.21	±1.29	±1.13
10g	Mean	7.98^{NS}	7.55^{NS}	7.50^{NS}	7.45^{NS}	7.19^{NS}	7.60^{NS}
	±SD	±1.18	±1.17	±1.11	±1.13	±1.17	±0.96
20g	Mean	8.02^{NS}	7.67^{NS}	7.69^{NS}	7.57^{NS}	7.43^{NS}	7.74^{NS}
	±SD	±0.95	±1.05	±1.16	±1.09	±1.21	±1.06
30g	Mean	7.81^{NS}	7.10^{NS}	7.33^{NS}	7.05^{NS}	7.00^{NS}	7.12^{NS}
	±SD	±1.27	±1.23	±1.28	±1.25	±1.33	±1.29
	% decrease /increase	**1.13 $^{\#}\downarrow$**	**3.13$^{\#}\downarrow$**	**0.92$^{\#}\uparrow$**	**2.62$^{\#}\downarrow$**	**No change$^{\#}$**	**4.17$^{\#}\downarrow$**
ANOVA		**0.32^{NS}**	**2.12^{NS}**	**1.12^{NS}**	**1.65^{NS}**	**1.11^{NS}**	**2.38^{NS}**

- Note: Mean values represent the average of 25 determinants in triplicates.
- a, b, c – The non-identical letters in any two rows within the column denote a significant difference at a minimum of 5% level.
- NS – The difference between the mean values within the columns is not significant.
- Maximum score for all the organoleptic attributes was 10.
- Level of significance in increasing order- (* $p<0.05$, ** $p<0.01$, *** $p<0.001$)
- #: Percent decrease in standard to 30 g of FOS addition

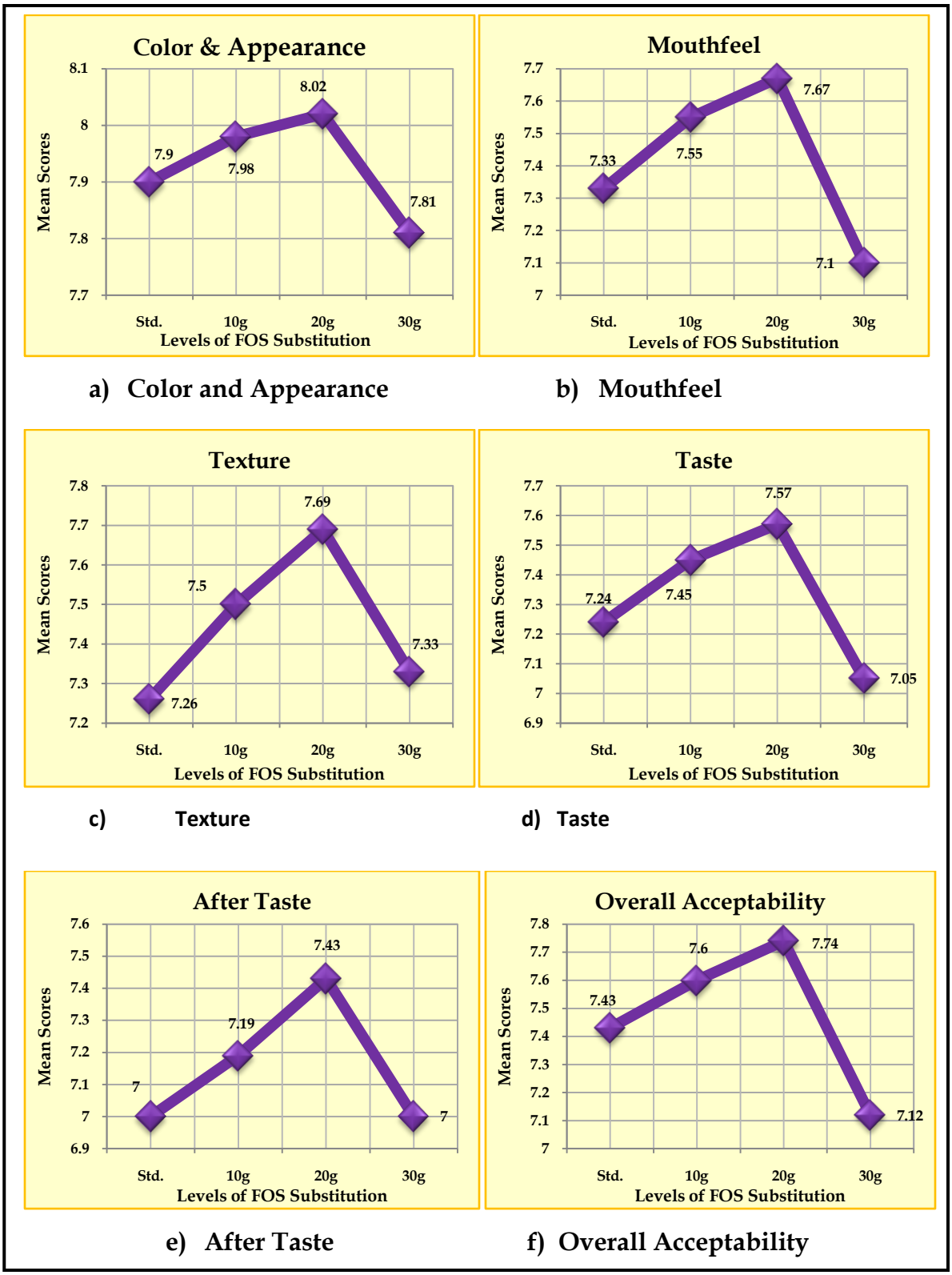


Fig. 5.4 (a-f): Scores for organoleptic attributes of *chocolate cake* added with varying levels of FOS

Results Highlights of Phase I

- ***Lilva kachori*** **was well accepted upto 5g of FOS addition without affecting sensory attributes. At higher levels of FOS incorporation, a significant gradual decrease in all the sensory attributes was exhibited, where color and appearance, texture and aftertaste were greatly affected.**
- **FOS can be incorporated to** ***vegetable paratha*** **upto 15g level without affecting the organoleptic qualities.**
- ***Rawa idli*** **was highly acceptable in terms of all the organoleptic attributes upto 20g of addition of FOS and** ***chocolate cake*** **was highly acceptable upto 30g of addition of FOS.**

DISCUSSION

The main objective of the present phase of the research work was to study the outcome of varying level of FOS addition on organoleptic characteristics of various food products *viz. lilva kachori, vegetable parantha, rawa idli* and *chocolate cake*.

FOS could be added successfully in deep fried food filling material upto 5 g/serving (4 moderate sized) without affecting most of the sensory attributes such as *mouthfeel, texture, taste, aftertaste and overall acceptability*, there after a significant reduction was observed, it can be attributed to reduced scores for most of the sensory attributes. The most affected attribute in lilva kachori was *color and appearance* with 18.7 percent reduction in the scores from standard kachori to 15g of FOS added kachori in terms of burn spots on the surface of kachori that appeared after frying.

In *vegetable parantha*, *color and appearance* scores reduced by 5.5 percent as the level of addition increased. This might be due to non-enzymatic maillard reaction, which was indicated by increased burn spots on the surface of paratha and kachori. Similar results were found in a study wherein, burn spots on the surface of chapatti increased significantly as the level of FOS addition increased from 6% to 20% (Mahendra and Sheth, 2013).

As the level of FOS addition increased in range of 20g-30 g the crumb of *cake* became harder and darker. A study reported increased crumb hardness when inulin was added as powder form rather than gel form (O'Brien et al, 2003). Similarly an enhancement of bread crust darker coloration was also reported for breads prepared in range of 3% to 10% inulin (Hager et al, 2011 and Poinot et al, 2010).

FOS can undergo the maillard browning reaction upon cooking, this property of FOS supported baked goods where have non-desirable effects on fried foods.

Taste and *after taste* scores of *lilva kachori* showed reduction with the increased level of FOS addition. Instead of spicy taste of kachori there was an increase in sweetness, which is not preferred normally. A similar result was observed in a study, wherein, a significant decrease in the scores of aftertaste was perceived for bread with 22 percent level of inulin substitution (Parnami and Sheth, 2010).

Taste scores of *veg. parantha* reduced with increased level of FOS addition, this could be because of increased sweetness of paratha. Physico-chemical characteristics of oligofructose showed it has moderately sweet in taste and has a sweetness of about 35% in comparison with sucrose (A Frank, 2002).

Taste and after taste of chocolate cake remained similar to the standard recipe upto 30g of addition of FOS. An eminent scientist reported that oligofructose addition upto 2 to 25% could be an excellent sugar and fat replacer, and is increases the moisture retention, and improves the texture and enhanced overall functionality of baked goods (A Franck, 2002).

FOS has low sweetness intensity since they are only about one-third as sweet as sucrose (NeoSugar Group, 1984). This property makes FOS a good sugar replacer in sweet food products where in savory products FOS is not very pleasing (Yun, 1996).

The *texture* of the kachori became soft and soggy giving undesirable mouthfeel with increased fluidity of the stuffing of kachori. FOS increases retention of moisture. Similar observations have been found in a study where the investigators have reported that oligofructose contributes humectancy to soft baked goods (N Kaur and Gupta, 2002).

Reduction in *mouthfeel* scores was observed in *veg. parantha* as the levels of FOS incorporation increased. This may be attributed to increased stickiness of the parantha. A significant reduction (7.4 to 8.6 percent) in the *texture* scores was observed as the level of FOS addition increased ($p<.05$) upto 10 g. The resultant

parantha felt sticky and were difficult to break. Similar results were obtained from a study where an increase in crumb hardness of bread was observed when inulin was added at 3 percent to 5 percent levels (O'Brien, et. al., 2003).

Usually kachori and parantha are supposed to be crusty and crispy but addition of FOS in filling material increased the moisture retention. Some technical modification like addition of fillers (corn starch, semolina or bread crumbs) to the filling material or addition of optimum amount of liquid could be done.

Though, there are number of studies conducted on sensory attributes of FOS added food products by processing method of baking, freezing, frying etc. But there is scarcity of studies on effect of FOS added steamed products. Therefore, the present study focused on steamed products like FOS incorporated *rawa idli* and was studied for its organoleptic characteristics.

FOS incorporation upto 20 g enhanced overall sensory qualities of *rawa idli*. All the sensory attributes excelled to the standard where texture and overall acceptability enhanced significantly ($p<0.05$). Water absorption power decreased as the level of FOS addition increased. Due to FOS addition thinning of batter was also observed. This may be because of synergistic effect of FOS. Parallel results were showed in a study conducted on *vegetable chilla* where in difficulty in flipping of *vegetable chilla* on shallow frying was observed due to the thinning of the batter at 15 g of FOS addition (Aparna et. al., 2013). The texture of rawa idli improved significantly ($p<0.01$), the panel members reported an increase in the softness of the idli which is contradictory to a study undertook in which texture scores were reduced by 3.75 percent in *dhokla* as little in hardness and decrease in softness (A Mahendra, 2013).

The water retention property of FOS increased the softness of idli and cake which is a desired attribute in both the food product. In kachori and parantha, stickiness increased due to moisture retention.

All the organoleptic attributes for *chocolate cake* remained unaffected as the level of FOS incorporation increased, this might be because of improved texture, after taste, taste and mouthfeel and overall acceptability. Numerous researches explain the analogous results for increased organoleptic attributes of cookies, ice-creams, meat sausages after FOS incorporation (Handa C et al 2011; Ting-ning lin and Gruen I 2012; Freitas Folly GA 2013).

Concluding Remarks

FOS can be incorporated in all the four food products studied. However, rawa idli and chocolate cake remained the most acceptable products even at the higher levels (20g and 30g respectively) of FOS addition. Lilva kachori and vegetable parantha were acceptable upto 5g and 15g of FOS addition respectively. FOS can be a very good alternative for sugar and fat replacement in baked and sweet food items where in savory food item some technical modification needed to be done.

FOS (oligofructose or oligofructan) has come into light in response to consumer demand for low calorie food. It is one of the functional food ingredient which is not yet well exploited and consumed by Indian population, therefore, more FOS added recipes need to be developed and studied for the feasibility of its addition and their acceptability so as to expand the data base of FOS added Indian recipes.

Looking at the scenario of use of FOS in managing of obesity, type 2 diabetes and other NCDs, and for the better gut health and immunity all the four products studied may be recommended for consumption by this population group.

Phase II Situational analysis: mapping the prevalence of obesity and hypertension in banks employees of urban Vadodara (A cross-sectional design).

Obesity has reached epidemic proportions globally, with more than 1.9 billion overweight adults with at least 600 million of them clinically obese and is a major contributor to the global burden of chronic disease and disability. Often coexisting in developing countries with under-nutrition, obesity is a complex condition, with serious social and psychological dimensions, affecting virtually all ages and socioeconomic group (WHO, 2016).

Globalization puts junk food and fast food within easy reach of a population often hard-pressed to find time to cook healthy meals, and with more than enough money to buy a greasy lunch at a nearby restaurant. In India, these factors have contributed to the rise of bad eating habits. Lack of exercise amongst a growing urban middle class, and their effects are startlingly visible. Overweight/obesity may not be considered as a specific disease but it is certainly the mother of important degenerative diseases of adult life such as hypertension diabetes and CVD. Prevention and control of this problem must, therefore, claim priority attention.

Therefore in the view of this, the present phase was planned to map the prevalence of obesity and hypertension in banks employees of urban Vadodara (A cross-sectional design).

For achieving the desired objectives, a total of 10 different banks from different areas of Vadodara city were conveniently selected based on the permission obtained from the administration department to organize the health screening camp. A total of five hundred and ninety five (595) bank employees irrespective of age and gender were screened for their anthropometric measurements, body fat percentage, basal metabolic rate and blood pressure.

The methodology to collect the above mentioned information is elaborated in Material and Methods chapter and results are presented in sections 5.4.1 to 5.4.5.

The results of this phase are presented into following sections-

Section 5.4.1	Distribution of subjects according to gender and age
Section 5.4.2	Classification of subjects according to BMI
Section 5.4.3	Anthropometric and biophysical profile of the subjects
Section 5.4.4	Prevalence of abdominal obesity and central obesity in bank employees
Section 5.4.5	Distribution of subjects according to percent body fat
Section 5.4.6	Prevalence of hypertension in the bank employees

Section 5.4.1 Distribution of subjects according to gender and age

The results as shown in Table 5.5 reveals that, out of five hundred and ninety five subjects screened, 75.79% were males and 24.20% were females. Out of these 70.50% of the males and 63.19% females were in the age group of 26-35 years respectively. Only 56 males and 8 females were of above 35 years of age.

Section 5.4.2 Classification of subjects according to BMI

According to Asia pacific BMI cut offs only 30.3 percent of males and 38.88 percent of female had normal BMI. Overall 7.2% of bank employees were underweight. A total of 7.05 percent subjects fell into obesity-grade II category. A total 37.25 percent male and 22.91 percent female were in obesity-grade I category and 19.49 percent bank employees were overweight (Table 5.6).

Table 5.5 : Distribution of the subjects according to the gender and age

Categories of age	Number of subjects (N=595)
Males	**451** (75.79)
21 - 25 yrs	77 (17.07)
26 - 30 yrs	187 (41.46)
31 - 35 yrs	131 (29.04)
> 35 yrs	56 (12.41)
Females	**144** (24.20)
21 - 25 yrs	45 (31.25)
26 - 30 yrs	66 (45.83)
31 - 35 yrs	25 (17.36)
>35 yrs	8 (5.55)

Note: numbers in parenthesis indicate percentage

Table 5.6 : Classification of subjects according to BMI

Classification of BMI (Kg / m^2)	Male (n=451)	Female (n=144)	Total subjects (N=595)
Underweight (<18.5)	29 (6.4)	14 (9.7)	43 (7.2)
Normal (18.5 - 22.9)	137 (30.3)	56 (38.88)	193 (32.43)
Overweight (23.0 - 24.9)	91 (20.17)	25 (17.36)	116 (19.49)
Obesity-grade I (25.0 - 29.9)	168 (37.25)	33 (22.91)	201 (33.78)
Obesity-grade II (≥30.0)	26 (5.76)	16 (11.11)	42 (7.05)
Total prevalence of obesity	194 (43.01)	49 (34.02)	243 (40.84)

Note: numbers in parenthesis indicate percentage

Section 5.4.3 Anthropometric and biophysical profile of the subjects

The mean weight of the male participants was 70.65±13.12 and for female subjects it was 58.23±12.07 kg. (Table 5.7). Mean waist circumference for male was 87.67 cm and mean hip circumference was 96.38 cm. Mean BP of the male bank employees was approx. 131/79 mmHg and for females it was 116/71 mmHg.

Table 5.7: Anthropometric and biophysical profile of subjects subjected to screening

Parameters	Males (n=451) (Mean ± SD)	Females (n=144) (Mean ± SD)
Height (cms)	170.08±6.91	156.20±6.86
Weight (kg)	70.65±13.12	58.23±12.07
BMI (Kg / m²)		
Underweight (<18.5)	17.34±1.00	17.56±0.70
Normal (18.5 - 22.9)	21.17±1.32	20.91±1.30
Overweight (23.0 - 24.9)	23.98±0.49	23.78±0.62
Obesity-grade I (25.0 - 29.9)	26.95±1.37	27.06±1.43
Obesity-grade II (≥ 30.0)	32.55±2.46	33.31±2.46
WC (cm)	87.67±9.69	77.29±11.08
HC (cm)	96.38±8.77	94.38±9.05
Waist Hip Ratio	0.90±0.05	0.81±0.06
Body Fat (%)	25.86±5.62	32.73±6.21
Systolic BP (mmHg)	131.25±14.18	116.09±18.97
Diastolic BP (mmHg)	79.81±10.40	71.48±10.56

Section 5.4.4 Prevalence of abdominal obesity and central obesity in bank employees

According to cut offs for waist circumference and waist hip ratio given by WHO 43.45% of males and 38.19% of female subjects showed the presence of abdominal obesity and 44.12% males and 30.55% females were at risk of developing central obesity. (Table 5.8)

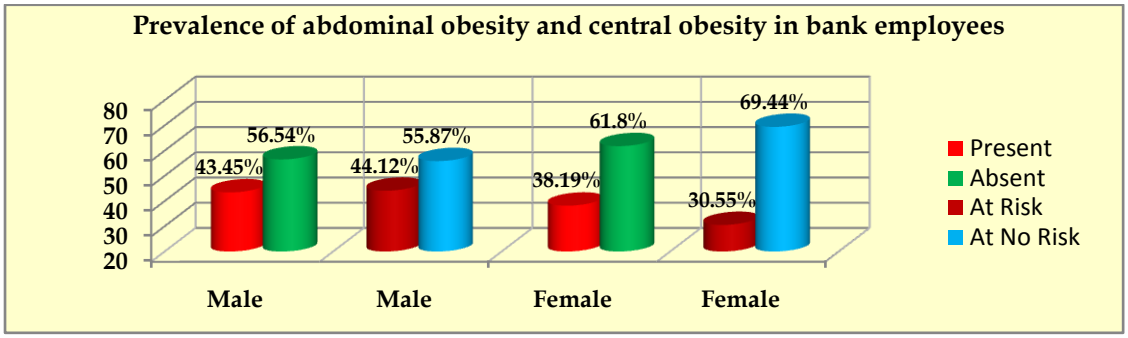


Figure 5.5: Prevalence of abdominal obesity and central obesity in bank employees

Table 5.8: Prevalence of abdominal obesity based on high waist circumference and high waist hip ratio in bank employees

Abdominal Obesity		Male (n = 451)	Femal (n=144)	Total (N=595)
(WC)	Present	196 (43.45)	55 (38.19)	251(42.18)
	Absent	255 (56.54)	89 (61.80)	344 (57.81)
(WHR)	At risk	199 (44.12)	44 (30.55)	243 (40.84)
	At no risk	252 (55.87)	100 (69.44)	352 (59.15)

Note: numbers in parenthesis indicate percentage

Section 5.4.5 Distribution of subjects according to percent body fat

The result of this subsection are presented graphically in Figure 5.6 and tabulated in Table 5.9. According to body fat percent levels 61.41% males and 52.7% of females had ≥25% of fat mass and females had ≥32% of fat mass in their body respectively. Only 6.8 % of total subjects were fit according to body fat percent levels.

Table 5.9: Distribution of the subjects according to percent body fat

Level of fitness	Male (n=451)	Female (n=144)	Total (N=595)
Athletes (Male: 6%-13%; Female; 14%-20%)	14 (3.10)	5 (3.4)	19 (3.19)
Fitness (Male: 14%-17%; Female; 21%-24%)	28 (6.2)	13 (9.02)	41 (6.8)
Acceptable (Male: 18%-24%; Female; 25%-31%)	132 (29.26)	50 (34.7)	182 (30.58)
Obese (Male: ≥25%; Female; ≥32%)	277 (61.41)	76 (52.7)	353 (59.32)

Note: numbers in parenthesis indicate percentage
Source: ACE (2009), 'what are the guidelines for percentage of body fat loss'American Council on Exercise (ACE).

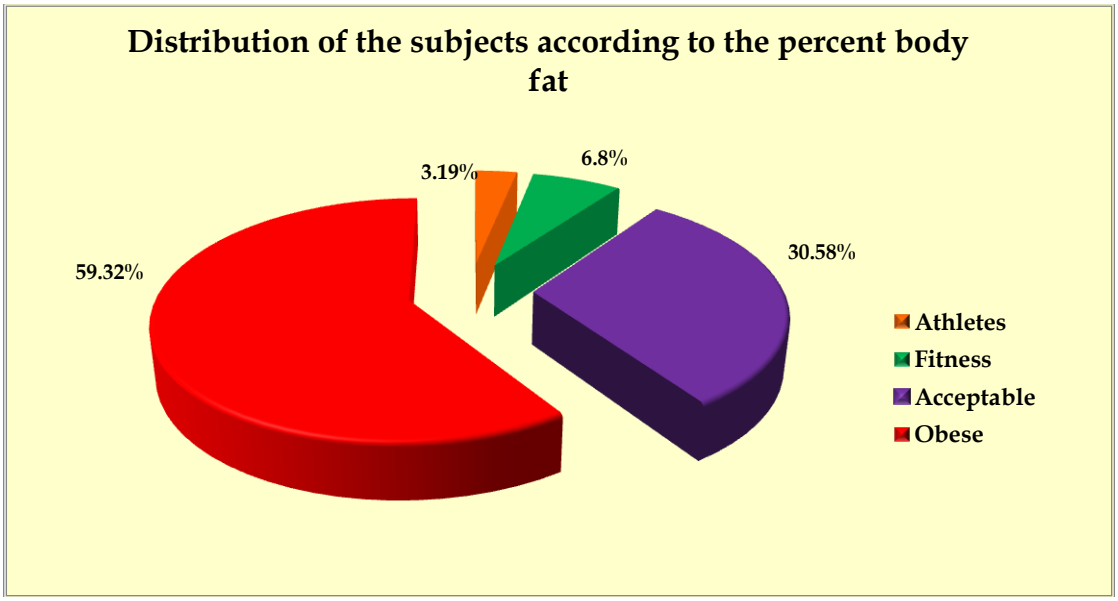


Figure 5.6: Distribution of the subjects according to the percent body fat

Section 5.4.6 Prevalence of hypertension in the bank employees

Table 5.4.5 reveals that approximately more than 54% of subjects were pre-hypertensive and 24% of subjects had moderate hypertension. Male were more hypertensive than females. (Table 5.10)

Table 5.10: Prevalence of hypertension in the bank employees

Hypertension Classification (mmHg)	Male (n=451)	Female (n=144)	Total (n=595)
Normal (SBP <120 and DBP <80)	92 (20.39)	99 (68.75)	191 (32.10)
Pre-hypertension (SBP <139 or DBP <89)	287 (63.63)	38 (26.38)	325 (54.62)
Moderate hypertension (SBP 140–160 or DBP 90–110)	130 (28.82)	10 (6.94)	140 (23.52)
Severe hypertension (SBP >160 or DBP > 110)	14 (3.10)	3 (2.08)	17 (2.85)

Note: numbers in parenthesis indicate percentage
Source: "Understanding blood pressure readings", American Heart Association, January 2011

Results Highlights

- **Out of five hundred and ninety five subjects screened, 75.79% were males and 24.20% were females.**
- **The prevalence of obesity was observed to be 40.83% (BMI ≥25) and 19.29% for overweight (BMI 23–24.9).**
- **Approximately more than 54% of subjects were pre-hypertensive with more males (63.63%) than females (26.38%) and 24% of subjects had moderate hypertension.**

DISCUSSION

Obesity is getting bigger promptly in India and Gujarat ranks 10th for males and 7th for females in the prevalence of overweight and obesity (Ramachandran A, et.al, 2010, NFHS-3). It affects every section of the globe and is the most overlooked public health problem of today. According to the National Family Health Survey (NFHS-3), the percentage of overweight or obesity increased from 11% in NFHS- 2 to 15% in NFHS-3.

In the present phase both male and female subjects screened were predominantly overweight or obese (61%). Obesity grade –I was more distinct in males (43%) as compared to females (34%). However, 11% of females belonged to obesity grade – II as compared to males (6%). Similar results are also mentioned in report of National Family and Health Survey -3 (NHFS 3), where in percentage of overweight and obese women increased from 11% in NFHS-2 to 15% in NFHS-3. Even in states like Punjab (30%), Kerala (28%) and Delhi (26%), percentage of overweight and obesity amongst women is highest (Unnikrishnan AG., et.al., 2012).

According to the National Family Health Survey 4 (NFHS-4), the percentage of women aged 15-49 years who are overweight or obese increased from 12.6% in NFHS- 3 to 20.7% in NFHS-4 and the percentage of men aged 15-49 years who are overweight or obese increased from 9.3% in NFHS- 3 to 18.6% in NFHS-4. In urban settings 26.3 % men and 31.3% of women were obese stated NHFS-4 (MOHFW, 2016). This may be due to lesser physical activity in the urban areas. Furthermore, overweight and obesity are both higher for women than men. The prevalence of overweight or obese in women is highest in Chandigarh (41.5%), followed by Delhi (34.9%), Kerala (32.4%) and Punjab (31.3%), all of which are relatively richer states (MOHFW, 2016). The prevalence of underweight and overweight among men shows similar variations by age, education, and prosperity index.

The prevalence of overweight and obesity in Gujarat in women aged 15-49 years was 34.5% in urban area and 15.4% in rural area. For men prevalence

was 25.9% in urban area and 14.4% for rural area. Overall prevalence of overweight and obesity was 34.5% for women and 25.9% for men in Gujarat in the year 2015-2016 (MOHFW, 2016).

Abdominal obesity, defined as increased waist circumference is one of the components of the collection of metabolic abnormalities collectively called as the metabolic syndrome (MS). The latest definition of MS by the International Diabetes Federation (IDF) has included abdominal obesity as one of the essential components (Alberti, 2005). Most of the subjects had abdominal obesity indicating a high risk for the development of NCD's. Asian Indians have increased visceral fat and central obesity and this is referred to as the Asian Indian phenotype (Joshi SR 2003; Snehlata et al 2003; Shetty 2012). It has been reported in several studies that visceral fat is associated with abdominal obesity and type 2 diabetes mellitus (Blaha et. al., 2008).

For several years it has been recognized that South Asian have certain unique clinical and biochemical characteristics that are collectively referred to as the Asian-Indian Phenotype. Despite relatively lower rates of obesity as defined by BMI they tend to have larger WC and WHR and thus have a superior extent of central obesity. Further Indians also tend to have excess body fat (Mishra and Shrivastava, 2013). Analogous to these findings 61.41% males and 52.7% of females had ≥25% and ≥32% of fat mass in their body respectively.

Hypertension is a modern day's wave and it is an increasingly significant medical and public health issue. Indian studies have revealed that the prevalence of hypertension has increased by 30 times among the urban population over a period of 55 years and about ten times among the rural population over a period of 36 years (Gupta R, 1997). In present study the average systolic/diastolic blood pressure of the subjects was 128/74 mg/dl however, approximately more than 46% of subjects were pre-hypertensive and 15% of subjects had moderate hypertension. These results were well supported the study where in 1493 bank employees studied for their

hypertension profile and overall prevalence of hypertension found to be 30.5% and 34.5% were pre-hypertensive (Mohmmedirfan HM et, al, 2012). Another study conducted in which prevalence of hypertension in the urban and rural population was found to be 40.8% and 17.9% respectively (Midha T, 2015).

Concluding Remarks

The prevalence of obesity and hypertension was high in staff of the selected banks.

Phase III **Comparison of grade-I obese subjects with non-obese subjects in terms of anthropometry profile, medical history, family history of diseases, defecation profile, hunger and satiety, psychological depression status, dependency on habits, dietary intakes, biophysical profile, atherogenic profile, endotoxemia and gut microbiota (*LAB, bifidobacteria, bacteroides and clostridium*) and understand the correlations between various parameters.**

Obesity and overweight are defined as abnormal or excessive fat accumulation in the body that may weaken one's health. The worldwide prevalence of obesity has more than doubled between 1980 and 2014 (WHO, 2016). Worldwide, at least 2.8 million people die each year because of being overweight or obese and approximately 2.3% of global DALYs (Disability-adjusted life years) are caused by obesity. Obesity leads to adverse metabolic effects on blood pressure, cholesterol, triglycerides and insulin resistance (GHO, WHO, 2016).

Recent researches draw attention to the role of gut microbiota in obesity and other degenerative diseases. The human gut microbiome also changes with the host to play an important part in several metabolic functions. Several studies supported the difference between lean and obese gut flora and the role of gut microbiota in energy harvesting and storage from ingested food and expenditure of energy (John K. DiBaise et. al., 2012)

Therefore an attempt was made to see the difference between non-obese and obese individuals with regards to their anthropometry profile, medical history, family history of diseases, defecation profile, hunger and satiety, psychological depression status, dependency on habits, dietary intakes, biophysical profile, lipemic profile, LPS and gut microbiota (*LAB, bifidobacteria, bacteroides and clostridium*) and understanding the correlations amongst these parameters.

For achieving the desired objectives, a total of 200 subjects were enrolled (100 non-obese and 100 obese) and screened based on inclusion and exclusion criteria. Informed consent was obtained from them. The methodology used to collect the

above mentioned information is elaborated in Material and Methods chapter and results of this phase are presented in sections 5.5.1 to 5.5.17

Section 5.5.1 Background information of obese and non-obese young adults

Section 5.5.2 Anthropometric profile of obese and non-obese young adults

Section 5.5.3 Prevalence of abdominal obesity based on waist circumference and waist hip ratio in obese and non-obese young adults

Section 5.5.4 Percent prevalence of obesity in subjects according to percent body fat

Section 5.5.5 Blood pressure of obese and non-obese young adults

Section 5.5.6 Family history of diseases of obese and non-obese young adults

Section 5.5.7 Personal medical history of obese and non-obese young adults

Section 5.5.8 Defecation profile of obese and non-obese young adults

Section 5.5.9 Personal habits of obese and non-obese young adults

Section 5.5.10 Physical activity pattern of obese and non-obese young adults

Section 5.5.11 Psychological depression profile of obese and non-obese young adults

Section 5.5.12 Hunger and satiety pattern of obese and non-obese young adults

Section 5.5.13 Frequency of consumption of food and dietary intakes of obese and non-obese young adults

Section 5.5.14 Atherogenic profile of obese and non-obese young adults

Section 5.5.15 Endotoxemia in obese and non-obese young adults

Section 5.5.16 Colonization of beneficial and potentially harmful bacteria in the gut of obese and non-obese young adults

Section 5.5.17 Relationships amongst BMI, direct and indirect determinants of obesity

Section 5.5.1 Background information of obese and non-obese young adults

The background information of the total subjects enrolled revealed that more number of subjects belonged to non-obese category of younger age group than older age group. There were more males (162) than females (38) in both the categories with majority of subjects being Hindu. 95% of obese and 91% of non-obese subjects were at least graduates or had higher degree in education. Most subjects came from nuclear family and belonged to upper middle class. The monthly per capita income of majority of participants was greater than Rs 28,114 per month for both the obese and non-obese subjects (Table 5.11)

Table 5.11: Background information of non-obese and obese young adults under study

Parameters	Non-obese (n=100)	Obese (n=100)
Age		
25-30 yrs.	76 (76)	50 (50)
31-35yrs.	24 (24)	50 (50)
Sex		
Male	76 (76)	86 (86)
Female	24 (24)	14 (14)
Religion		
Hindu	96 (96)	95 (95)
Muslim/Christian/Others	4 (4)	5 (5)
Type of family		
Joint	28 (28)	21 (21)
Nuclear	48 (48)	56 (56)
Extended nuclear	24 (24)	23 (23)
Education		
Profession or honors'/graduate or post graduate	91 (91)	95 (95)
Intermediate/ post high school diploma/high school certificate/middle school certificate	9 (9)	5 (5)
Occupation		
Profession/semi-profession	72 (72)	90 (90)
Clerical, shop-owner, farmer/skilled worker/semi-skilled worker	18 (18)	10 (10)
Family Income Per Month		
≥ 28114	67 (67)	66 (66)
≤ 28114	33 (33)	34 (34)
Socioeconomic Class		
Upper class	5 (5)	6 (6)
Upper middle class	95 (95)	94 (94)

Note: Figures in parenthesis represent percent of subjects.

Section 5.5.2 Anthropometric profile of obese and non-obese young adults

Anthropometric profile depicted in Table 5.12 revealed that the mean BMI of obese subjects was 27.6 kg/m^2 and for non-obese it was 21 kg/m^2. Waist circumference and hip circumference were higher in obese subjects. Obese male subjects had ≥25% body fat and obese female subjects had ≥32% body fat.

Table 5.12: Mean values for anthropometric parameters of non-obese and obese young adults

Parameters		Non-obese (n=100)	Obese (n=100)	Mean difference	't' value	*p-value*
Height (cms) Mean±SD	**Male**	169.97 ± 6.30	170.52 ± 6.49	0.552	0.54^{NS}	**0.585**
	Female	156.05 ± 6.78	156.62 ± 6.35	0.574	0.25^{NS}	**0.798**
Weight (kg) Mean±SD	**Male**	61.14 ± 5.96	80.57 ± 8.44	19.43	16.71***	**0.000**
	Female	50.27 ± 3.84	67.22 ± 6.93	16.95	9.73***	**0.000**
BMI (kg/m^2) Mean±SD	**Male**	21.14 ± 1.30	27.62 ± 1.55	6.48	28.56***	**0.000**
	Female	20.66 ± 1.26	27.34 ± 1.45	6.67	14.86***	**0.000**
	Total subjects	21±1.3	27.6 ± 1.54	6.55	32.54***	**0.000**
WC(cms) Mean±SD	**Male**	80.93 ± 5.88	96.74 ± 7.40	15.81	14.91***	**0.000**
	Female	71.70 ± 7.93	94.28 ± 6.49	22.57	9.01***	**0.000**
HC (cms) Mean±SD	**Male**	90.89 ± 5.16	102.90 ± 6.32	12.01	13.13***	**0.000**
	Female	88.41 ± 4.49	106.07 ± 5.92	17.65	10.38***	**0.000**
WHR Mean±SD	**Male**	0.88 ± 0.05	0.93 ± 0.04	0.049	6.78***	**0.000**
	Female	0.80 ± 0.06	0.89 ± 0.06	0.081	3.53**	**0.001**
Percent body fat Mean±SD	**Male**	22.05 ± 3.05	30.48 ± 3.81	8.43	15.40***	**0.000**
	Female	28.82 ± 2.81	38.56 ± 3.80	9.73	9.02***	**0.000**

Note: NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed

Section 5.5.3: Percent prevalence of abdominal obesity based on waist circumference and waist hip ratio in obese and non-obese young adults

As shown in Table 5.13 prevalence of abdominal obesity was significantly high ($p<0.000$) in obese subjects (84%) compared to non-obese subjects (9%). Also obese subjects were 53.08 times at a higher risk of developing abdominal obesity. Obese subjects also showed high prevalence of central obesity.

Table 5.13: Prevalence of abdominal obesity based on high waist circumference and high waist hip ratio in obese and non-obese young adults

Parameters		Obese male (n=86)	Non-obese male (n=76)	Obese female (n=14)	Non-obese female (n=24)	Total obese (n=100)	Total non-obese (n=100)
WC	Present	71 (82.55)	5 (6.57)	13 (92.85)	4 (16.66)	84 (84)	9 (9)
	Absent	15 (17.44)	71 (93.42)	1 (7.69)	20 (83.33)	16 (16)	91 (91)
$\chi 2$ Value (p-value)		92.94*** (0.000)		20.21*** (0.000)		112.48*** (0.000)	
OR-CI		67.21 CI (23.18-194.83)		65.00 CI (6.51-648.26)		53.08 CI (22.26-126.55)	
WHR	At risk	71 (82.55)	22 (28.94)	10 (71.42)	5 (20.83)	81 (81)	27 (27)
	At no risk	15 (17.44)	54 (71.05)	4 (28.57)	19 (79.16)	19 (19)	73 (73)
$\chi 2$ Value (p-value)		47.13*** (0.000)		9.22** (0.002)		58.40*** (0.000)	
OR-CI		11.61 CI (5.51-24.48)		9.5 CI (2.07-43.50)		11.52 11.53 CI (5.91-22.45)	

- Figures in parenthesis represent percent of subjects
- NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***. 2 tailed.
- Waist circumference cut-offs: Male <90- No; >90-Yes; Females <80-No; >80-Yes;
- Source: Consensus Statement for Diagnosis of Obesity, Abdominal Obesity and the Metabolic Syndrome for Asian Indians and Recommendations for Physical Activity, Medical and Surgical Management; JAPI 2009; Vol. 57; p – 163– 170.
- Waist hip ratio cut-offs: Male <0.9- No; >0.9-Yes; Females <0.85-No; >0.85-Yes;
- Source: Waist Circumference and Waist–Hip Ratio: Report of a WHO Expert Consultation Geneva, 8–11 December 2008.

Section 5.5.4: Percent prevalence of obesity in the subjects according to percent body fat

100% obese female subjects had >32% body fat where as only 12.50% non-obese females had >32% body fat. 94.18% obese male subjects had greater than 25% of body fat (Table 5.14)

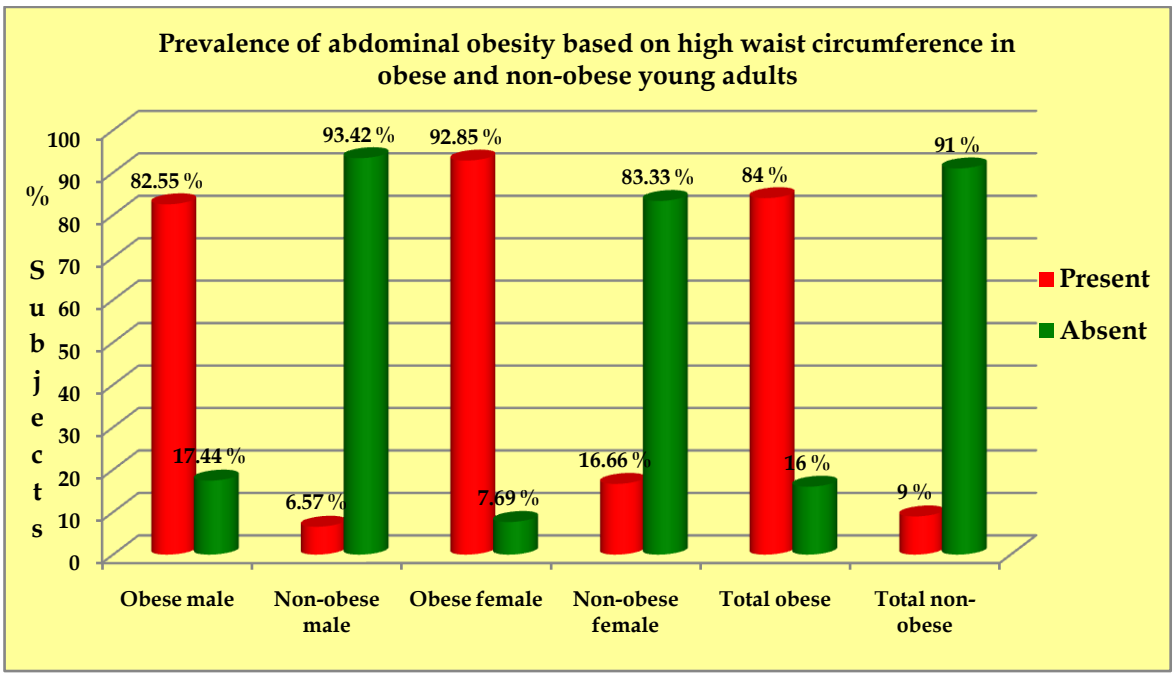


Fig. 5.7: Prevalence of abdominal obesity based on high waist circumference in obese and non-obese young adults

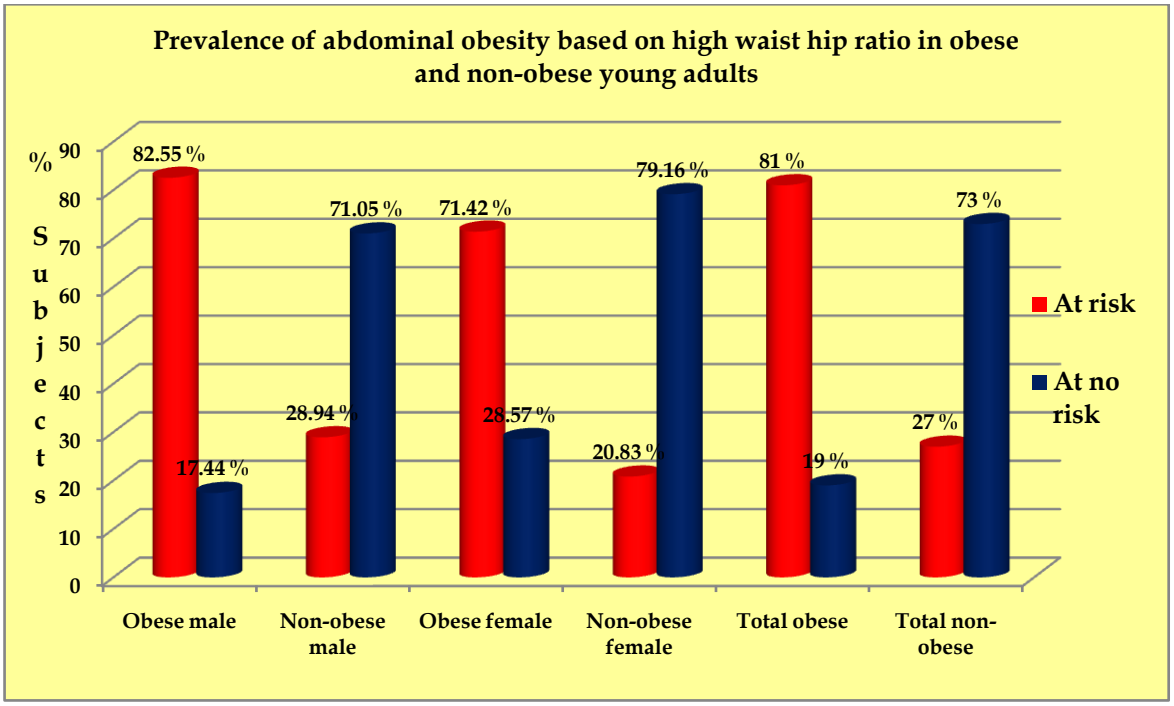


Fig. 5.8: Prevalence of abdominal obesity based on high waist circumference and high waist hip ratio in obese and non-obese young adults

Table 5.14: Percent prevalence of obesity in subjects according to percent body fat

Percent body fat	Obese male (n=86)	Non-obese male (n=76)	Obese female (n=14)	Non-obese female (n=24)	Total obese (n=100)	Total non-obese (n=100)
Fitness	0	11 (14.47)	0	2 (8.33)	0	13 (13)
Acceptable	5 (5.81)	55 (72.36)	0	19 (79.16)	5 (5)	74 (74)
Obese	81 (94.18)	10 (13.15)	14 (100)	3 (12.5)	95 (95)	13 (13)
χ2 Value (p-value)	**106.93*** (0.000)**		**22.66*** (0.000)**		**134.66*** (0.000)**	
OR-CI	106.92 CI (34.82-328.23)				127.15 CI (43.54-371.31)	

- Figures in parenthesis represent percent of subjects.
- NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed.
- Cut-offs: Male- fitness 14-17%; acceptable 18-24%; obese >25% fat.
- Females- fitness 21-24%; acceptable 25-31%; obese >32% fat.
- Source: ACE (2009) what are the guidelines for percentage of body fat loss? American Council on Exercise (ACE).

Section 5.5.5 Hypertension profile of obese and non-obese young adults

(Table 5.15.1 and 5.15.2) Although 77% of obese and 64% of non-obese subjects were pre-hypertensives, their diastolic blood pressure was within the normal range in both obese and non-obese subjects. It was the systolic blood pressure that crossed the normal limits of 120mmHg. Obese were 1.88 times at a higher risk of developing hypertension. A significant difference (p<0.01) was observed in systolic blood pressure of obese and non-obese females. Mean values for blood pressure were significantly (p<0.01) high in obese subjects (127.08/79.90) when compared to non-obese subjects (122.88/76.37)

Table 5.15.1: Prevalence of hypertension in obese and non-obese young adults

Hypertension	Non-obese (n=100)	Obese (n=100)	χ^2 value	*p-value*	OR
Normal	36 (36)	23 (23)	**4.04***	**0.044**	**1.88 CI (1.01-3.49)**
Pre-hypertensive	64 (64)	77 (77)			

- Figures in parenthesis represent percent of subjects
- NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed.
- Source: "Understanding blood pressure readings", American Heart Association, January 2011

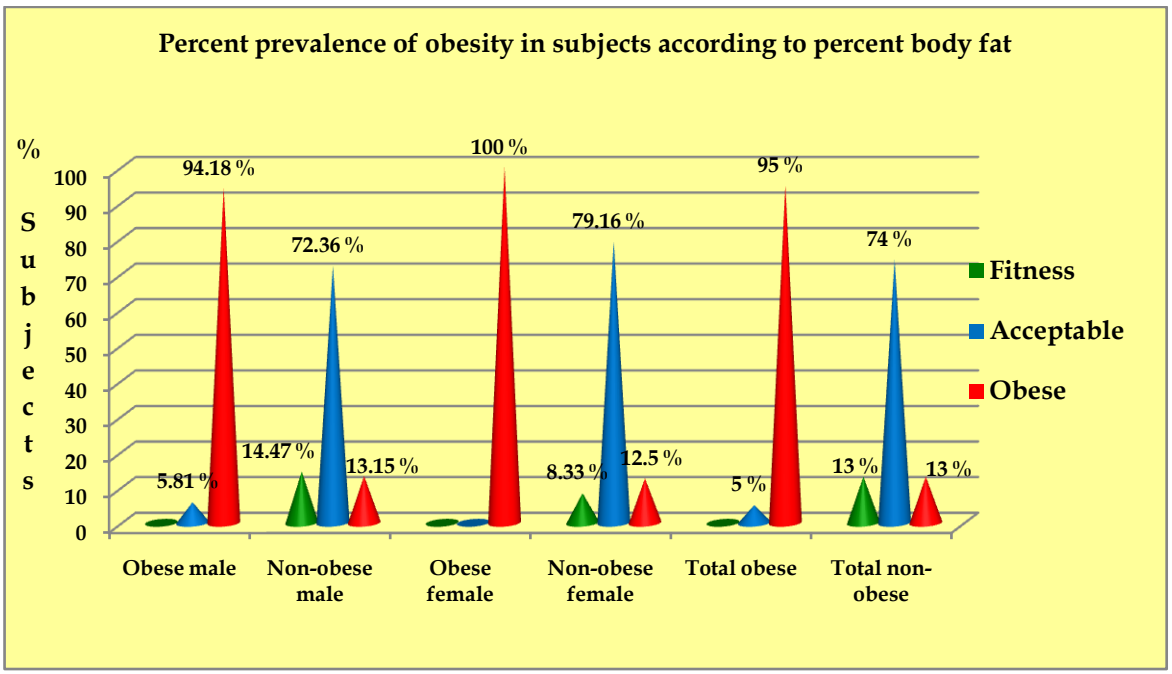


Fig. 5.9: Percent prevalence of obesity in subjects according to percent body fat

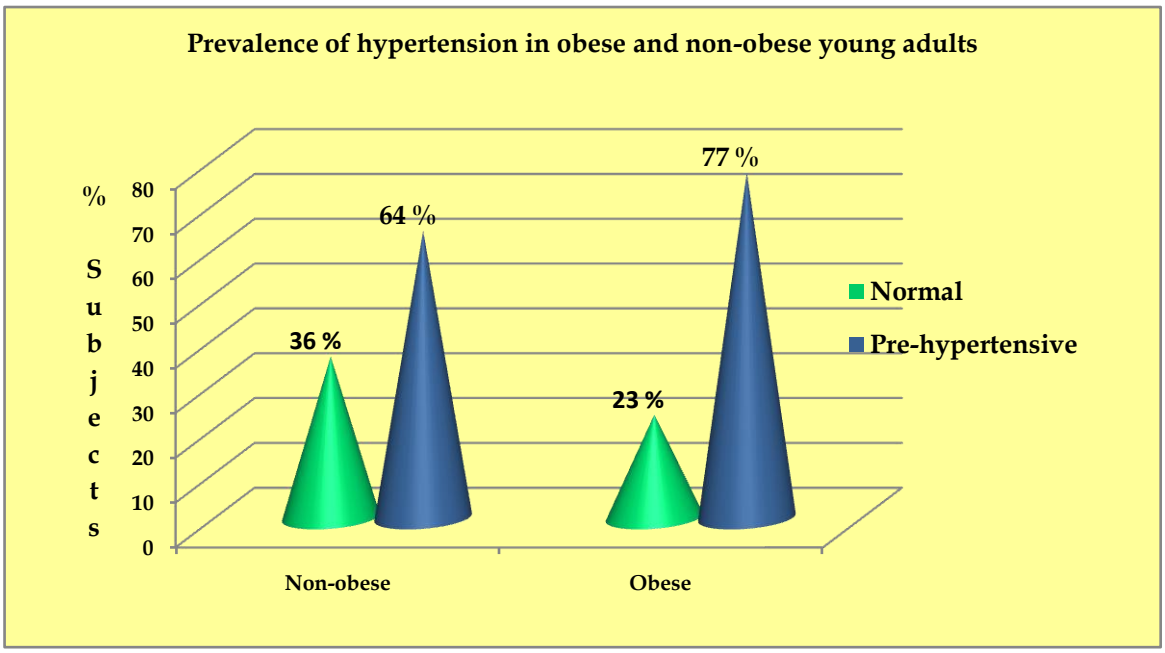


Fig. 5.10: Prevalence of hypertension in obese and non-obese young adults

Table 5.15.2: Mean values of blood pressure of non-obese and obese young adults

Parameters		Non-obese (n=100)	Obese (n=100)	Mean diff.	't' Value	*p-value*
Systolic blood pressure (mmHg) Mean±SD	**Male**	125.72±12.59	127.79±8.17	**2.06**	**1.25^{NS}**	**0.212**
	Female	113.87±10.04	122.71±8.73	**8.83**	**2.74****	**0.009**
	Total subjects	122.88±13.01	127.08±8.39	**4.20**	**2.71****	**0.007**
Diastolic blood Pressure (mmHg) Mean±SD	**Male**	77.72±9.59	80.44±6.88	**2.71**	**2.08***	**0.038**
	Female	72.08±8.54	77.00±6.58	**4.91**	**1.85^{NS}**	**0.072**
	Total subjects	76.37±9.62	79.90±6.91	**3.59**	**3.02****	**0.003**

Note: NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed.

Section 5.5.6 Family history of disease and personal medical history of obese and non-obese young adults

As seen in Table 5.16.1 and 5.16.2 family history of NCD's was more common in obese than non-obese subjects. Most of obese subjects had strong family history where only 15% of non-obese had strong family history. There was a significant association (χ^2 Value-26.61***) between family history of disease with BMI of the subjects. Subjects with severe family history of co-morbidities were at 5.13 time's higher risk of developing obesity [RR-5.13; CI (2.69-9.77)].

Table 5.16.1: Family history of diseases amongst non-obese and obese young adults

Family History of NCD's	Non-obese (n=100)	Obese (n=100)	χ^2 value	*p-value*	Relative risk
Mild family history (0-2)	53 (53)	18 (18)	**26.61*****	**0.000**	**5.13 CI (2.69-9.77)**
Moderate family history (3-4)	32 (32)	23 (23)			
Strong family history (5-6)	15 (15)	59 (59)			

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, p < 0.05: *, p < 0.001: ***; 2 tailed.

Table 5.16.2: Family History of diseases amongst non-obese and obese young adults

Type of Disease		Non-obese (n=100)	Obese (n=100)
Obesity	Both Parents	7 (7)	27 (27)
	Single Parent	35 (35)	44 (44)
	Brother	3 (3)	21 (21)
	Sister	1 (1)	17 (17)
	Grand Parents	10 (10)	24 (24)
	Other Relation	27 (27)	50 (50)
Hypertension	Both Parents	7 (7)	15 (15)
	Single Parent	36 (36)	43 (43)
	Brother	2 (2)	3 (3)
	Sister	2 (2)	3 (3)
	Grand Parents	18 (18)	21 (21)
	Other Relation	12 (12)	23 (23)
Diabetes Mellitus	Both Parents	2 (2)	4 (4)
	Single Parent	23 (23)	34 (34)
	Brother	1 (1)	2 (2)
	Sister	0	1 (1)
	Grand Parents	23 (23)	29 (29)
	Other Relation	25 (25)	30 (30)
CVD's	Both Parents	2 (2)	1 (1)
	Single Parent	12 (12)	18 (18)
	Brother	0	1 (1)
	Sister	0	0
	Grand Parents	10 (10)	18 (18)
	Other Relation	11 (11)	10 (10)

Note: Figures in parenthesis represent percent of subjects.

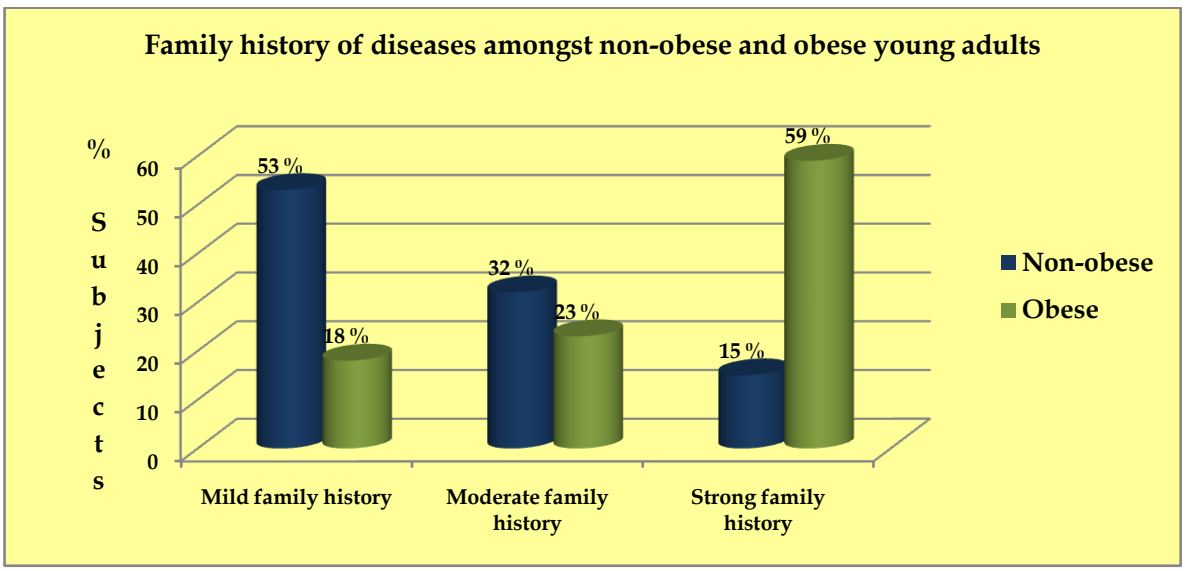


Fig. 5.11: Family History of diseases amongst non-obese and obese young adults

Section 5.5.7 Personal medical history of obese and non-obese young adults

In study groups more non-obese subjects (52%) had gastrointestinal disorders like heartburn and acidity compared to obese subjects (29%). Medical history revealed that with regards to dental problems 74% of non-obese had cavities, dry mouth, bleeding/swollen gums and bad breathe whereas 61% obese subjects had such problems. Locomotor disorders like knee joint pain and back pain was more prevalent in obese subjects (51%) whereas less non-obese subjects (10%) reported similar kind of problems (Fig. 5.12).

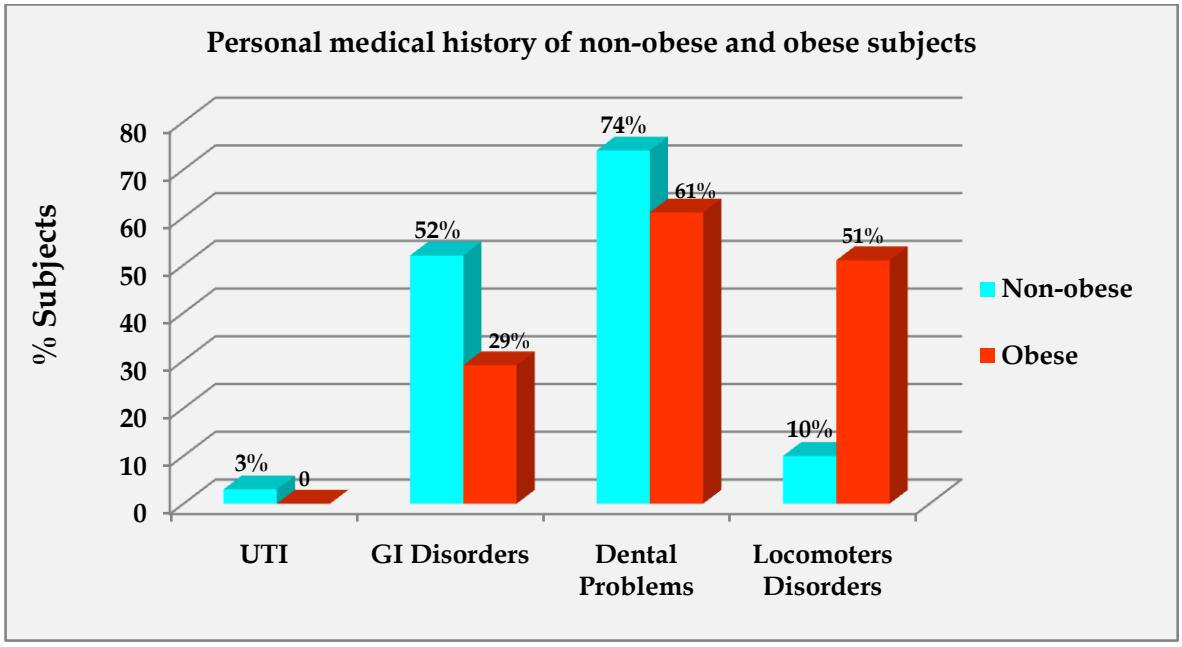


Figure 5.12: Personal medical history of non-obese and obese subjects

Section 5.5.8. Defecation profile of obese and non-obese young adults

(Table 5.17.2) There was a difference in defecation profile of non-obese and obese subjects. As per subjects perception 27% non-obese subjects and 38% obese subjects reported the presence of constipation. (Table 5.17.1) There was a non-significant association found between BMI and defecation profile of the subjects [χ^2 Value-2.99^{NS}; OR-1.84; CI (0.91-3.70)] but after administering the tool to know the detailed defecation profile of the subject results revealed that 26% of obese and 16% of non-obese had constipation.

Table 5.17.1: Defecation profile of non-obese and obese young adults

Defecation profile	Non-obese (n=100)	Obese (n=100)	χ^2 value	*p-value*	OR
Constipated	16 (16)	26 (26)	**2.99^{NS}**	**0.083**	**1.84**
No constipation	84 (84)	74 (74)			**CI (0.91-3.70)**

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, p < 0.05: *, p < 0.001: ***; 2 tailed.

Table 5.17.2: Defecation profile of non-obese and obese young adults

Defecation Profile	Normal (n=100)	Obese (n=100)
Constipation (As subjects perception)		
Present	27 (27)	38 (38)
Absent	73 (73)	62 (62)
Frequency (times / day)		
1	72 (72)	62 (62)
2-3	28 (28)	38 (38)
Quantity of stool		
Small	17 (17)	23 (23)
Middle/large	83 (83)	77 (77)
Hardness of stool		
Very hard/hard	13 (13)	39 (39)
Medium/soft	87 (87)	61 (61)
Color of stool		
Blackish/ middle	80 (80)	94 (94)
Yellowish	20 (20)	6 (6)
Odor of stool		
Strong	7(7)	25 (25)
Medium/Weak	93 (93)	75 (75)
Feeling after defecation		
Bad	7 (7)	21 (21)
Fine/ very Fine	93 (93)	79 (79)
Regular use of laxatives		
No	90 (90)	83 (83)
Yes	10 (10)	17 (17)
Constipated	16 (16)	26 (26)
Normal defecation	84 (84)	74 (74)
Degree of constipation		
Severe	3 (3)	1 (1)
Moderate	10 (10)	1 (1)
Mild	4 (4)	24 (24)
No constipation	84 (84)	74 (74)

Note: Figures in parenthesis represent percent of subjects.

Section 5.5.9 Personal habit profile of obese and non-obese young adults

Obese subjects consumed more alcohol (60%), cigarette (29%), tea (67%), and coffee (21%) compared to the non-obese subjects. Significant association was seen between BMI and intake of alcohol (χ^2-10.53**), cigarette (χ^2-4.04*) tea (χ^2-20.38***), and coffee (χ^2-6.78**) (Table 5.18.1). As Shown in table 5.18.2, 63% of obese subjects were found in severe to extreme category of dependency on habits compared to 36% of non-obese subjects. No association [χ^2 Value-3.70^{NS}; OR-2.15; CI (0.97-4.73)] was observed between the BMI and dependency on habits.

Table 5.18.1: Personal habit profile of non-obese and obese young adults

Dependency	Frequency of consumption	Non-obese (n = 100)	Obese (n = 100)	χ^2 value	*p-value*	OR
Alcohol	Frequently	11 (11)	20 (20)	**10.53****	**0.001**	**2.55 CI (1.44-4.51)**
	Less frequently	26 (26)	40 (40)			
	Never	63 (63)	40 (40)			
Cigarette	Frequently	14 (14)	26 (26)	**4.04***	**0.044**	**1.99 CI (1.01-3.92)**
	Less frequently	3 (3)	3 (3)			
	Never	83 (83)	71 (71)			
Tobacco powder/paste	Frequently	7 (7)	10 (10)	**0.57^{NS}**	**0.448**	**1.47 CI (0.53-4.04)**
	Never	93 (93)	90 (90)			
Tea	Frequently	34 (34)	63 (63)	**20.38*****	**0.000**	**3.77 CI (2.09-6.77)**
	Less frequently	1 (1)	4 (4)			
	Never	65 (65)	33 (33)			
Coffee	Frequently	1 (1)	11 (11)	**6.78****	**0.009**	**3.05 CI (1.28-7.28)**
	Less frequently	7 (7)	10 (10)			
	Never	92 (92)	79 (79)			
Aerated Drinks	Frequently	76 (76)	61 (61)	**1.22^{NS}**	**0.268**	**0.60 CI (0.25-.47)**
	Less frequently	15 (15)	25 (25)			
	Never	9 (9)	14 (14)			

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed.

Table 5.18.2: Personal habits of non-obese and obese young adults

Degree of dependency	Non-obese (n=100)	Obese (n=100)	χ^2 value	*p-value*	OR
Mild dependency	21 (21)	11 (11)	**3.70^{NS}**	**0.054**	**2.15 CI (0.97-4.73)**
Moderate dependency	43 (43)	26 (26)			
Severe dependency	36 (36)	57 (57)			
Extreme dependency	0	6 (6)			

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, p < 0.05: *; 2 tailed.

Section 5.5.10 Physical activity level of obese and non-obese young adults

Table 5.19 depicts almost equal percentage of non-obese and obese subjects belonged to sedentary and moderate level of physical activity and no significant association was observed between BMI and physical activity level of non-obese and obese subjects.

Table 5.19: Association of BMI with physical activity level in non-obese and obese young adults

Physical activity level (MET minutes)	Non-obese (n = 100)	Obese (n= 100)	χ^2 value	*p-value*	OR
Low (<60 mins)	50 (50)	53 (53)	**0.17^{NS}**	**0.672**	**1.12 CI (0.64-1.96)**
Moderate 1 (≥60mins for ≥3 days)	4 (4)	11 (11)			
Moderate 2 (≥150 mins for ≥5days)	34 (34)	35 (35)			
Moderate 3 (≥600 mins for ≥5days)	8 (8)	0			
High 1 (≥1500mins for ≥3days)	4 (4)	1 (1)			

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, p < 0.05: *, p < 0.001: ***; 2 tailed.

Section 5.5.11 Psychological depression profile of obese and non-obese young adults

As shown in table 5.20, 20% of non-obese subjects suffered from borderline clinical depression to severe depression as compared to 8% obese subjects. No significant association was observed between BMI and varying degree of depression.

Section 5.5.12 Hunger and satiety pattern of obese and non-obese young adults

Table 5.21 describes that no significant difference was observed in the mean hunger scores of non-obese and obese subjects. The intensity of hunger pattern was same in both the groups. However, the satiety was significantly delayed ($p<0.001$) in obese subjects at the specific meal time of breakfast, lunch, evening and dinner compared to non-obese subjects. The non-obese subjects consumed less quantity of food and had early satiety compared to obese individuals.

Table 5.20: Percent prevalence of psychological depression in non-obese and obese young adults

Psychological depression level	Non-obese male (n=76)	Obese male (n=86)	Non-obese female (n=24)	Obese female (n=14)	Non-obese (n=100)	Obese (n=100)
Normal	46 (60.52)	56 (65.11)	11 (45.83)	5 (35.71)	57 (57)	61 (61)
Mild mood disturbance	15 (19.73)	26 (30.23)	8 (33.33)	5 (35.71)	23 (23)	31 (31)
Borderline clinical	9 (11.84)	0	4(16.66)	2(14.28)	13 (13)	2 (2)
Moderate depression	4 (5.26)	4 (4.65)	0	1 (7.14)	4 (4)	5 (5)
Severe depression	2 (2.63)	0	1 (4.16)	1 (7.14)	3 (3)	1 (1)
(χ^2) value (p-value) OR (CI)	**0.36^{NS} (0.547) 0.82 (0.43-1.55)**		**0.36^{NS} (0.547) 1.52 (0.39-5.91)**		**0.32^{NS} (0.566) 0.84 (0.48-1.48)**	

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, p < 0.05: *, p < 0.01: **; 2 tailed.

Table 5.21: Mean hunger and satiety scores of non-obese and obese young adults at various meal timings

	Meal	Non-obese (n=100) Mean ± SD	Obese (n=100) Mean ± SD	Students "t" test	*p-value*
Hunger scores	**Breakfast**	4.09 ± 0.78	3.98 ± 0.88	**0.93^{NS}**	**0.353**
	Lunch	3.51 ± 0.68	3.49 ± 0.88	**0.17^{NS}**	**0.858**
	Evening	4.08 ± 0.90	4.18 ± 0.88	**0.79^{NS}**	**0.430**
	Dinner	3.49 ± 0.78	3.37 ± 1.05	**0.91^{NS}**	**0.361**
	Total mean score	3.87 ± 0.54	3.86 ± 0.66	**0.11^{NS}**	**0.908**
Satiety scores	**Breakfast**	6.16 ± 0.64	6.38 ± 0.82	**2.09***	**0.037**
	Lunch	6.39 ± 0.70	6.94 ± 0.87	**4.88*****	**0.000**
	Evening	5.67 ± 0.99	6.05 ± 0.65	**3.18****	**0.002**
	Dinner	6.71 ± 0.82	7.30 ± 1.00	**4.56*****	**0.000**
	Total mean score	6.39 ± 0.60	6.79 ± 0.64	**4.55*****	**0.000**

Note: NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed; Hunger scores 1 – 5, where 1= Famished, starving 2= Headache, weak, cranky, low energy , 3= Want to eat now, stomach growls and feels empty, 4= Hungry - but could wait to eat, starting to feel empty but not there yet, 5= Not hungry, not full: Satiety scores 5 –10, where 5= Not hungry, not full, 6 = Feeling satisfied, stomach feels full and comfortable, 7 = Feeling full, definitely don't need more food, 8 = uncomfortably full, 9 = Stuffed, very uncomfortable, 10 = Bursting, painfully full

Section 5.5.13 Frequency of consumption of food and dietary intakes of obese and non-obese young adults

Table 5.22.1 represents frequency of food consumption of non-obese and obese subjects. Consumption of fibrous fruits was reported more by non-obese subjects as compared to obese subjects.

Table 5.22.1: Frequency of food consumption of non-obese and obese young adults

Food groups		Non-obese (n=100)	Obese (n=100)
Cereals	**Frequently**	100 (100)	100 (100)
Millets	**Frequently**	1 (1)	2 (2)
	Less frequently	87 (87)	87 (87)
	Never	12 (12)	11 (11)
Pulses and legumes	**Frequently**	91 (91)	86 (86)
	Less frequently	9 (9)	14 (14)
Vegetables	**Frequently**	99 (99)	95 (95)
	Less frequently	1 (1)	5 (5)
Nuts and oil seeds	**Frequently**	77 (77)	84 (84)
	Less frequently	23 (23)	16 (16)
Condiments and spices	**Frequently**	75 (75)	73 (73)
	Less frequently	25 (25)	23 (23)
Days/week	**≤ 3 Days**	83 (83)	87 (87)
	≥ 4 Days	17 (17)	13 (13)
Fruits - high fiber (>5 g %)	**Frequently**	19 (19)	10 (10)
	Less frequently	81 (81)	90 (90)
Fruits – moderate fiber (4.99 -2 g %)	**Frequently**	15 (15)	10 (10)
	Less frequently	85 (85)	90 (90)
Fruits – low fiber (1.99 -0.5 g %)	**Frequently**	22 (22)	20 (20)
	Less frequently	78 (78)	80 (80)

Note: Figures in parenthesis represent percent of subjects.
Frequently: daily;2-3 times in a week ;once in a week, Less Frequently: fortnightly; monthly; rarely.

Table 5.22.2 reveals significantly higher intakes of all the macro nutrients in obese male as compared to non-obese male ($p<0.01$), whereas, energy and fat intake was significantly higher in obese females than non-obese female subjects ($p<0.05$). Obese males also had significantly higher total dietary fiber intakes as compared to non-obese males ($p<0.01$). Sodium intakes were also found to be significantly higher in both obese males and females than non-obese males and females.

Table 5.22.2: Mean intake of nutrients as per 24 hr dietary recall of non-obese and obese young adults

Nutrients	Non-obese Male# (n=76)	Obese male# (n=86)	't' test	Non-obese Female# (n=24)	Obese Female# (n=14)	't' test
Energy (Kcal)	2173.7±777.9	2793.4±576	**5.80*** **	1974.5±665.0	2403.1±533.6	**2.05***
CHO (g)	311.2±147.0	370.6±76.6	**3.27****	252.83±75.28	298.3±68.9	**1.85**^{NS}
Protein (g)	61.9±20.1	84.8±24.8	**6.37*** **	53.41±15.36	59.1±9.9	**1.24**^{NS}
Fat (g)	77.0±32.4	103.0±39.2	**4.55*** **	76.41±35.34	104.9±29.8	**2.53***
Crude fibre (g)	7.79±2.92	9.54±2.59	**4.02*** **	6.59±2.25	6.05±1.60	**0.79**^{NS}
Insoluble dietary fibre (g)	13.79±5.53	15.48±5.86	**1.93**^{NS}	12.82±3.62	11.62±4.33	**0.91**^{NS}
Soluble dietary fibre (g)	4.55±2.00	5.44±1.64	**3.10****	4.34±1.21	4.14±1.53	**0.45**^{NS}
Total dietary fibre (g)	18.13±7.55	21.50±7.73	**2.80****	17.41±4.55	16.64±5.86	**0.45**^{NS}
Sodium (mg)	196.7±94.6	316.0±197.6	**4.79*** **	222.5±185.7	408.2±228.6	**2.73****
Total MUFA (mg)	10.35±11.95	12.46±12.15	**1.11**^{NS}	9.65±10.17	19.13±12.24	**2.56***
Total PUFA (mg)	9.32±10.95	16.31±16.06	**3.19****	6.69±7.68	19.18±11.53	**4.0*** **
Total saturates (mg)	15.41±6.24	15.41±14.64	**0.01**^{NS}	11.03±13.72	23.66±14.02	**2.71****

Note: NS = non-significant, $p < 0.05$: *, $p < 0.01$: **, $p < 0.001$: ***; 2 tailed; # both the male and female subjects falls into sedentary and moderately physical activity.

Section 5.5.14 Atherogenic profile of obese and non-obese young adults

The outcomes of the lipid profile revealed that the mean serum cholesterol of obese was significantly higher than non-obese but both the groups were within the normal range. Mean serum TG levels of obese subject were 194.61 mg/dl which exceeded the normal values where in non-obese it was slightly higher (160.22 mg/dl). HDL cholesterol was at lower side in both the groups. Both the groups were at risk as the TC/HDL ratio was higher than 4. (Table 5.23)

Table 5.23: Atherogenic profile of non-obese and obese young adults

Parameters (mg/dl)	Non-obese (n=40)	Obese (n=72)	Mean diff.	't' value	p-value
Total cholesterol	147.16±33.21	182.57±30.47	**35.41**	**5.70*** **	**0.000**
Serum TG	160.22±57.30	194.61±67.53	**34.38**	**2.72****	**0.008**
HDL	35.60±7.03	36.58±7.59	**0.98**	**0.67^{NS}**	**0.501**
LDL	79.01±33.83	107.15±32.01	**28.13**	**4.36*** **	**0.000**
VLDL	32.54±10.69	38.83±13.56	**6.29**	**2.52***	**0.013**
TC/HDL Ratio	4.21±1.02	5.20±1.42	**0.99**	**3.88*** **	**0.000**
LDL/HDL Ratio	2.29±0.96	3.11±1.32	**0.82**	**3.45****	**0.001**

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, $p < 0.05$: *, $p < 0.01$: **; 2 tailed

Section 5.5.15 Endotoxemia in obese and non-obese young adults

An attempt was made to study the prevalence of obese and non-obese subjects. Metabolic endotoxemia (as indicated by high LPS values) was more prominent in obese (47.06%) as compared to non-obese subjects (35.29%). A non significant association was found between BMI and endotoxemia (Table 5.24.1). The mean value for LPS was found to be significantly high by 8.52 pg/ml in obese subjects as compared to non-obese subjects (Table 5.24.2).

Table 5.24.1 Prevalence of metabolic endotoxemia in obese and non-obese young adults

Endotoxemia	Non-obese (n=34)	Obese (n=68)	χ^2 value	*p-value*	OR
Endotoxemia	22 (64.71)	36 (52.94)	**1.26^{NS}**	**0.260**	**1.62 CI (0.69-3.81)**
Metabolic endotoxemia	12 (35.29)	32 (47.06)			

Note: Figures in parenthesis represent percent of subjects; NS = non-significant; 2 tailed
Endotoxemia (≤20 pg/ml); metabolic endotoxemia (≥20 pg/ml). Source; (Berg RD, 1996).

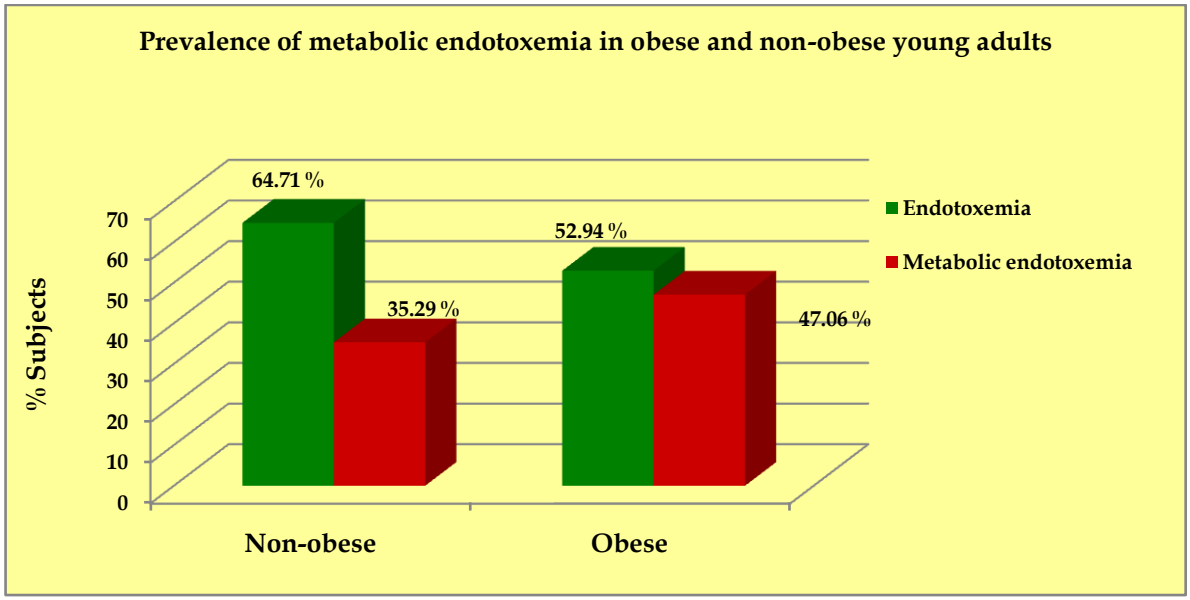


Fig. 5.13: Prevalence of metabolic endotoxemia in obese and non-obese young adults

Table 5.24.2: Plasma LPS levels of non-obese and obese young adults

	Non-obese (n=34)	Obese (n=68)	Mean diff.	't' value	*p-value*
Plasma LPS (pg/ml)	15.02±8.17	23.55±18.89	**8.52**	**2.51***	**0.014**

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, p < 0.05: *; 2 tailed

Section 5.5.16 Gut profile of obese and non-obese young adults

The fecal microbial load of non-obese and obese subjects revealed that the gut of the non-obese subjects was colonized more with the friendly bacteria like *lactic acid*

bacteria (11.89 vs 10.86) and *bifidobacteria* (12.26 vs 12.07) as compared to obese subjects. However, the gut of the obese subjects was colonized with higher counts of *bacteroides* (12.44 vs 13.76) and *clostridium* (11.73 vs 11.76) as compared to non-obese subjects which are potential pathogens. (Table 5.25)

Table 5.25: Gut profile of non-obese and obese young adults

Gut flora $\log_{10}$ Values (CFU /g)	Non-obese (n=100) Mean±SD	Obese (n=81) Mean±SD	Mean diff.	't' value	p-value
Fecal *bifidobacteria*	12.26±1.87	12.07±1.10	**0.19**	**0.83^{NS}**	**0.406**
Fecal *lactic acid bacteria*	11.89±1.52	10.86±1.13	**1.03**	**5.04*****	**0.000**
Fecal *bacteroides*	12.44±1.46	13.76±0.89	**1.32**	**7.09*****	**0.000**
Fecal *clostridium*	11.73±1.76	11.76±0.37	**0.02**	**0.12^{NS}**	**0.898**

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, $p < 0.05$: *, $p < 0.01$: **; 2 tailed.

Section 5.5.17 Relationships amongst BMI, direct and indirect determinants of obesity

Sub section 5.5.17.1 Correlation amongst anthropometric parameters, blood pressure, defecation status and lifestyle factors in non-obese and obese subjects

Table 5.26 summarizes the correlation amongst anthropometric parameters blood pressure and lifestyle factors. Age was positively corelated with anthropometric parameters and blood pressure. Weight, BMI, WC, WHR, percent body fat and diastolic BP were found to be significantly positively associated with family history of diseases. Defecation status was negatively associated with BMI, waist circumference and percent body fat which means as BMI, waist circumference and percent body fat incresed constipation increased. A positive significant association was also seen amongst alcohol intake, cigarette smoking, tea and coffee intake and weight, BMI, WC and WHR. Subjects who were more dependent on personal habits had more weight and BMI as these were positively corelated with each other.

Sub section 5.5.17.2 Correlation amongst anthropometric parameters and dietary intakes in non-obese and obese subjects

As seen in Table 5.27 all the anthropometric parameters were significantly positively associated with intake of macronutrients and total PUFA intake. Percent body fat positively corelated with energy and fat intake and total PUFA intake. Sodium intake was also found to be positively correlated with all the anthropometric paremeters and percent body fat. Total dietary fiber was seen to be positively correlated with all anthropometric parameters. Total MUFA intake was positively correlated with waist circumference.

Table 5.26: Correlation amongst anthropometric parameters, blood pressure and lifestyle factors in non-obese and obese subjects

Life style factors	Weight	BMI	WC	WHR	% body fat	Systolic BP	Diastolic BP
Age	0.281**	0.317**	0.314**	0.352**	0.148*	0.271**	NS
Systolic BP	0.382**	0.251**	0.377**	0.376**	NS	NS	0.676**
Diastolic BP	0.320**	0.240**	0.304**	0.289**	NS	0.676**	NS
Family history of diseases	0.318**	0.418**	0.402**	0.317**	0.339**	NS	0.182**
Defecation status	NS	-0.151*	-0.201**	NS	-0.177*	NS	NS
Alcohol intake	0.326**	0.251**	0.322**	0.297**	NS	0.188**	0.162*
Cigarette smoking	0.226**	0.189**	0.251**	0.237**	NS	NS	NS
Tea intake	0.386**	0.323**	0.391**	0.268**	0.143*	NS	NS
Coffee intake	0.187**	0.216**	0.192**	NS	0.212**	NS	NS
Aerated drinks intake	NS	-0.174*	NS	NS	-0.161*	-0.139*	NS
Dependency on habits	0.360**	0.279**	0.370**	0.326**	NS	0.148*	0.181*
Physical activity level	NS	NS	-0.141*	NS	NS	NS	NS

Note: NS: Non-significant; $p < 0.05$: *, $p < 0.01$: **

Table 5.27: Correlations amongst anthropometric parameters and dietary intakes in non-obese and obese subjects

Dietary intakes	Weight	BMI	WC	WHR	Percent body fat
CHO intake	0.337**	0.260**	0.247**	0.149*	NS
Energy intake	0.411**	0.381**	0.395**	0.231**	0.151*
Fat intake	0.296**	0.328**	0.388**	0.213**	0.217**
Protein intake	0.465**	0.398**	0.432**	0.341**	NS
Sodium intake	0.228**	0.362**	0.380**	0.245**	0.307**
Total dietary fibre	0.243**	0.202**	0.231**	0.219**	NS
Total MUFA	NS	NS	0.219**	NS	NS
Total PUFA	0.255**	0.302**	0.362**	0.229**	0.176*

Note: NS: Non-significant; $p < 0.05$: *, $p < 0.01$: **

Sub section 5.5.17.3 Correlation amongst anthropometric parameters, lifestyle factors, atherogenic profile, gut flora and endotoxemia in non-obese and obese subjects

(Table 5.28) Age was significantly positively associated with total cholesterol, serum TG, VLDL, bacteriodes and LPS and negatively correlated with *Lactic acid bacteria*. A significant positive correlation was seen between weight and BMI with atherogenic profile, LPS and *Bacteriodes* and a negative corelation was seen with *Lactic acid bacteria*. Systolic blood pressure was negatively correlated with *Clostridium.* Family history of diseases was positively correlated with atherogenic profile, LPS and *Bacteriodes*. Defecation status was positively correlated with *Bifidobacteria* and Lactic acid *bacteria* while alcohol intake was negatively corelated with these. Physical activity level and depression status negatively correlated with *Bacteriodes*. Hunger scores negatively correlated with LPS and satiety scores negatively correlated with *Bifidobacteria* and *Clostridium*.

Sub section 5.5.17.4 Correlation amongst dietary intake, atherogenic profile, gut flora and endotoxemia in non-obese and obese subjects

Energy intake, CHO intake, fat intake, fatty acid intakes negatively associated with *Bifidobacteria*. *Clostridium* was negatively correlated with CHO, total MUFA and total saturates intake and *Lactic acid bacteria* was negatively correlate with fat intake. Bacteriodes was positively correlated with fat, protein, sodium and total PUFA intake. A positive correlation was found between total cholesterol, serum TG and VLDL with energy, protein and sodium intake. LPS is significantly positively associated with protein intake and total dietary fiber. (Table 5.29)

Table 5.28: Relationship amongst anthropometric parameters, life style factors, atherogenic profile, gut profile and LPS

Life style factors	Total C	Serum TG	LDL	VLDL	TC/HDL	LDL/HDL	*Bifidobacteria*	*Lactic acid bacteria*	*Bacteroides*	*Clostridium*	LPS
Age	0.233*	0.246**	NS	0.227*	NS	NS	NS	-0.169*	0.209**	NS	0.226*
Weight	0.253**	0.197*	NS	0.186*	NS	NS	NS	-0.326**	0.334**	NS	0.270**
BMI	0.405**	0.257**	0.315**	0.232*	0.284**	0.242*	NS	-0.346**	0.441**	NS	0.297**
WC	0.291**	0.257**	0.203*	0.247**	0.235*	0.193*	NS	-0.287**	0.399**	NS	0.287**
WHR	NS	0.224*	NS	0.228*	NS	NS	NS	-0.162*	0.303**	NS	0.266**
Percent body fat	0.362**	0.188*	0.307**	NS	0.258**	0.207*	NS	-0.220*	0.306**	NS	NS
Systolic blood pressure	NS	NS	NS	NS	NS	NS	NS	NS	NS	-0.163*	NS
Family history of diseases	0.398**	0.215*	0.326**	0.222*	0.296**	0.246*	NS	NS	0.259**	NS	0.229*
Defecation status	NS	-0.219*	NS	-0.221*	NS	NS	0.191**	0.186*	NS	NS	NS
Alcohol	NS	NS	NS	NS	NS	NS	-0.231**	-0.190*	NS	NS	NS
Tea	0.213*	NS	NS	NS	0.245**	NS	NS	NS	0.188*	NS	NS
Personal habits	0.300**	0.193*	0.247**	0.191*	0.268**	0.209*	NS	NS	0.235**	NS	NS
Physical activity	NS	NS	NS	NS	NS	NS	NS	NS	-0.168*	NS	NS
Depression	NS	NS	NS	NS	NS	NS	NS	NS	-0.213**	NS	NS
Hunger mean scores	NS	0.198*	NS	0.193*	NS	NS	NS	NS	NS	NS	-0.210*
Satiety mean scores	NS	NS	NS	NS	NS	NS	-0.154*	NS	NS	-0.217**	NS

Note: NS: Non-significant; $p < 0.05$: *, $p < 0.01$: **

Table 5.29: Relationship amongst dietary intakes, atherogenic profile, gut profile and endotoxemia

Dietary intakes	Total cholesterol	Serum TG	HDL	VLDL	*Bifidobacteria*	*Lactic acid bacteria*	*Bacteroides*	*Clostridium*	LPS
CHO intake	NS	NS	NS	NS	-0.221**	NS	NS	-0.156*	NS
Energy intake	0.246**	0.224*	NS	0.234*	-0.159*	NS	NS	NS	NS
Fat intake	NS	0.292**	0.189*	0.304**	-0.157*	-0.161*	0.185*	NS	NS
Protein intake	0.263**	0.199*	NS	0.215*	NS	NS	0.210*	NS	0.346**
Sodium intake	0.279**	0.267**	NS	0.249**	NS	NS	0.293**	NS	NS
Total dietary fibre	NS	0.230*	NS	0.237*	NS	NS	NS	NS	0.314**
Total MUFA	NS	0.207**	0.191*	0.204*	-0.220**	NS	NS	-0.165*	NS
Total PUFA	NS	0.335**	0.278**	0.354**	-0.186*	NS	0.199**	NS	NS
Total saturates	NS	NS	NS	NS	-0.166*	NS	NS	-0.171*	NS

Note: NS: Non-significant; $p < 0.05$: *, $p < 0.01$: **

Sub section 5.5.17.5 Correlation amongst atherogenic profile, gut flora and endotoxemia in non-obese and obese subjects

Total cholesterol, LDL, TC/HDL and LDL/HDL ratio was negatively associated with *Lactic acid bacteria* and positively correlated with *Bacteriodes*. A positive correlation was found between *Bifidobacteria* with *LAB, Bacteriodes* and *Clostridium*. Clostridium was positively associated wih *LAB* and *Bacteriodes*. LPS negatively associated with LAB and clostridium where positively associated with bacteroides. (Table 5.30)

Sub section 5.5.17.6 Relationship of BMI with direct and indirect determinants of obesity

To further assess the relationship between BMI with direct and indirect determinants of obesity linear multiple regression analysis was performed. Table 5.31 reveals the strong predictors of obesity in young adults of urban Vadodara. Enter method was selected for analysis and was the best fit model at $p<0.001$ level of significance. LPS, sodium intake, carbohydrates intake, tea intake, alcohol intake, family history of disease, protein intake, *LAB, bacteroides*, total PUFA intake, fat intake and energy intake were found to be the predictor of obesity to the accuracy of 53.1%. Upon further analysis factors that contributed to obesity ranked in the order of contribution were intake of fat ($\beta=0.452$) followed by intake of energy ($\beta=0.344$), *LAB* ($\beta=0.312$), *Bacteroides* ($\beta=0.257$), intake of sodium ($\beta=0.243$) and intake of tea ($\beta=0.231$) were found to be the significant contributors.

Table 5.30: Relationship amongst atherogenic profile, gut profile and endotoxemia

	Serum TG	HDL	LDL	VLDL	TC/HDL	LDL/HDL	*Lactic acid bacteria*	*Bacteroides*	*Clostridium*	LPS
Total C	0.295**	NS	0.894**	0.288**	0.697**	0.673**	-0.281**	0.198*	NS	NS
Serum TG	NS	0.202*	NS	0.989**	NS	NS	NS	NS	NS	NS
HDL	NS	NS	-0.233*	0.235*	-0.631**	-0.535**	NS	NS	NS	NS
LDL	NS	NS	NS	NS	0.822**	0.862**	-0.257**	0.191*	NS	NS
VLDL	NS	NS	NS	NS	NS	-0.185	NS	NS	NS	NS
TC/HDL	NS	NS	NS	NS	NS	0.941**	-0.201*	0.228*	NS	NS
LDL/HDL	NS	NS	NS	NS	NS	NS	-0.200*	0.198*	NS	NS
Bifidobacteria	NS	NS	NS	NS	NS	NS	0.544**	0.331**	0.394**	NS
Lactic acid bacteria	NS	NS	NS	NS	NS	NS	NS	NS	0.209**	-0.236*
Bacteroides	NS	NS	NS	NS	NS	NS	NS	NS	0.248**	0.222*
Clostridium	NS	NS	NS	NS	NS	NS	NS	NS	NS	-0.205*

Note: NS: Non-significant; $p < 0.05$: *, $p < 0.01$: **

Table 5.31: Model summary of relationship of BMI with various parameters

Model Summary				
Model	**R**	**R Square**	**Adjusted R Square**	**Std. Error of the Estimate**
1	0.767[a]	0.588	**0.531**	2.33
a. Predictors: (Constant) LPS, sodium intake, carbohydrates intake, tea intake, alcohol intake, family history of disease, protein intake, *LAB, bacteroides*, total PUFA intake, fat intake, energy intake				

ANOVA[b]						
Model		**Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
1	**Regression**	675.226	12	56.269	**10.334**	**0.000[a]**
	Residual	473.715	87	5.445		
	Total	1148.940	99			
a. Predictors: (Constant), LPS, sodium intake, carbohydrates intake, tea intake, alcohol intake, family history of diseases, protein intake, LAB, bacteroides, total PUFA intake, fat intake, energy intake						
b. Dependent Variable: BMI						

Coefficients[a]						
Model		**Unstandardized Coefficients**		**Standardized Coefficients**	**t**	**Sig.**
		B	**Std. Error**	**Beta**		
1	**(Constant)**	19.265	4.243		4.541	0.000
	Family history of disease	0.503	0.320	0.126	1.570	0.120
	Alcohol intake	0.166	0.135	0.096	1.231	0.222
	Tea intake	0.236	0.076	0.231**	3.096	0.003
	CHO intake	0.001	0.002	0.045	.541	0.590
	Energy intake	0.002	0.001	0.344*	2.053	0.043
	Fat intake	-0.040	0.015	-0.452**	-2.758	0.007
	Protein intake	0.009	0.017	0.055	.505	0.615
	Sodium intake	0.004	0.002	0.243**	2.646	0.010
	Total PUFA intake	0.005	0.029	0.021	0.175	0.862
	LAB	-0.775	0.198	-0.312***	-3.907	0.000
	Bacteroides	0.708	0.236	0.257**	3.005	0.003
	LPS	0.025	0.018	0.105	1.401	0.165
a. Dependent Variable: BMI						

Results Highlights of Phase III

- There were more males (162) than females (38) in both the categories with majority of subjects being Hindu.
- Age was positively corelated with anthropometric parameters and blood pressure.
- Prevalence of abdominal obesity was significantly high ($p<0.000$) in obese subjects when compared to non-obese.
- Obese were 1.88 times at a higher risk of developing hypertension.
- Subjects with severe family history of co-morbidities were at 5.13 time's higher risk of developing obesity [OR-5.13; CI (2.69-9.77)] and anthropometric perameters, percent body fat and diastolic BP was positively associated with family history of diseases.
- 26% of obese and 16% of non-obese had constipation and overall defecation status was negatively associated with BMI, WC and percent body fat.
- Significant association was seen between BMI and intake of alcohol (χ^2-10.53**), cigarette smoking (χ^2-4.04*), intake of tea (χ^2-20.38***), and intake of coffee (χ^2-6.78**).
- Total MUFA intake was positively correlated with WC.
- The intensity of hunger pattern was same in both the groups. The non-obese subjects consumed less quantity of food and had early satiety compared to obese individuals.
- The mean cholesterol of the subjects was within the normal range. Both the groups were at risk as the TC/HDL ratio was higher than 4. Age was positively associated with TC, STG and VLDL.
- TC, LDL, TC/HDL and LDL/HDL ratio was negatively associated with lactic acid bacteria and positively associated with *bacteriodes.*

Continued….

Results Highlights of Phase III

- The LPS was found to be significantly high by 8.52 pg/ml in obese subjects as compared to non-obese subjects. Age and family history of diseases was positively corelated with LPS. Hunger scores were negatively corelated with LPS.
- The gut of the non-obese subjects was colonized more with the friendly bacteria and the gut of the obese subjects was colonized with higher counts of *bacteroides* and *clostridium* which are potential pathogens. Satiety scores were negalively corelated with *bifidobacteria* and *clostridium. Lactic acid bacteria* were negatively corelated with fat intake. LPS was negatively associated with *LAB* and *clostridium*.
- Factors that contributed to obesity ranked in the order of contribution were intake of fat (β=0.452) followed by intake of energy (β=0.344), *LAB* (β=0.312), *Bacteroides* (β=0.257), intake of sodium (β=0.243) and intake of tea (β=0.231) were found to be the significant contributors.

DISCUSSION

Present phase of the study was an attempt made to see the difference between non-obese and obese individuals with regards to their anthropometry profile, medical history, family history of diseases, defecation profile, hunger and satiety, psychological depression status, dependency on habits, dietary intakes, biophysical profile, lipemic profile, LPS and gut microbiota (*LAB, bifidobacteria, bacteroides and clostridium*) and understanding the correlations amongst these parameters.

Results said that obese subjects had comparatively higher waist circumference, waist hip ratio and percent body fat to non-obese subjects. It can be compared with the researches; discovered that Asian Indians exhibit unique features of obesity; excess body fat, abdominal adiposity, increased subcutaneous and intra-abdominal fat, and deposition of fat in ectopic sites (liver, muscle, etc.) (Mishra A et. al., 2009). Prevalence of abdominal obesity was significantly high ($p<0.000$) in obese subjects (84%) compared to non-obese subjects (9%). 81% Obese subjects also had waist-hip-ratio more than normal cut-offs. Asian Indians have more abdominal fat deposition than their European and Pacific Island counterparts. They also have significantly higher ratio of abdominal fat to thigh (Rush E, Plank L, Chandu V, et al., 2004).

6.57% of non-obese male and 16.66% of non-obese female subjects had abdominal obesity, despite the fact they had normal BMI, in line with the study in which 30.9% of men and 32.8% of women in industrial population in India were reported to have abdominal obesity with normal BMI (Reddy et al., 2006).

Results showed 100% obese female subjects had >32% body fat where as only 12.50% non-obese females had >32% body fat. 94.18% obese male subjects had greater than 25% of body fat. Several investigators have shown that body fat is higher in Asians, particularly south Asians, compared with white Caucasians for the similar level of BMI (Mishra A and Vikram NK, 2004).

Studies reaveled that south asians appear to be metabolically obese, though BMI levels may fall into the category of non-obese. This phenomenon is partially explained by excess body fat, high intraabdominal and subcutaneous fat, and ectopic fat deposition in various organs and body sites, which may contribute to insulin resistance, dyslipidemia, hyperglycemia, and excess procoagulant factors in south asians (Mishra A and Bhardwaj S, 2014).

A recent study indicated the overall prevalence of hypertension in India was 29.8% in which 35.8% hypertensive residing in west urban india (Anchala, R et al., 2014). In another study; showed that the prevalence of hypertension was significantly higher in individuals more than 35 years as compared to those less than 35 years (Jugal K, Neeru G et al., 2016), which is similar to this study where all the subjects were less than 35 years of age in which 77% of obese and 64% of non-obese subjects were pre-hypertensives. Obese were 1.88 times at a higher risk of developing hypertension. The prevalence of hypertension was found to be consistently increased with increasing BMI, weight, WC and WHR. This is a well established fact obesity increases the risk of development of hypertension.

In present study family history of NCD's was more common in obese than non-obese subjects. Subjects with severe family history of co-morbidities were at 5.13 time's higher risk of developing obesity. Family history helps to identify people at high risk of obesity and related diseases like diabetes, and CVD's. The role of heredity and genes in the development of obesity is recently come into light. A study conducted in Gambia on 5389 adult's subjects stated that subjects with a family history of hypertension had a higher diastolic BP and BMI, higher cholesterol and uric acid concentrations, and an increased risk of obesity. Those with a family history of obesity had a higher BMI and were at increased risk of obesity. Individuals with a family history of diabetes had a higher BMI and higher concentrations of glucose, cholesterol, triglycerides and uric acid, and their risk of obesity and diabetes was increased. Subjects with a family history of stroke had a higher BMI, as well as

higher cholesterol, triglyceride and uric acid concentrations; affirmed a study conducted by team of scientists (Van der Sande et al., 2001). In line with this in the present study revealed that family history of NCD's had a significant positive correlation with atherogenic profile of the subjects, more the heredity; higher the TC, TG, LDL, VLDL, TC/HDL ratio and LDL/HDL ratio.

Results revealed that 16% of non-obese and 26% of obese subjects had constipation. Similarly, a significant negative relationship was observed between BMI, WC and percent body fat with constipation clearly indicated a connection between obesity and constipation. A study conducted on city inhabitants and farmers had the similar results in which bowel habit was studied in 966 obese patients, revealed the significant difference in constipation frequency i.e. 8.3% in obese patients and 1.5% in normal-weighting, according to weekly bowel actions criterion (Pecora P et.al., 1981). In line with this, a cross-section household survey had done on Tehran to find the prevalence of gastrointestinal symptoms and disorders and their related factors on 18,180 adults, found 38.9% of subjects who had functional constipation, had a BMI more than 25 kg/m^2 (Pourhoseingholi MA et. al., 2009). The etiology of obesity and constipation is not clear but a comprehensible association between obesity and constipation could be bring into being with these studies however further studies to find out the underlying cause required.

20% obese subjects frequently consume alcohol where only 11% non-obese subjects frequently consume alcohol and a positive association was found between alcohol consumption and BMI. Available evidence suggested that the association between alcohol and obesity is non-linear, differing in relation to patterns and levels of drinking (French MT et. al., 2010). The possible reason suggested by Yehomans; energy from alcohol appears to be additive to energy from other sources, increasing the energy density of the meal. Further than adding energy to a meal, alcohol may actually stimulate food intake (Yehomans MR, 2010). Another study suggested that alcohol intake may be a risk factor for obesity in some individuals, possibly based on a huge number of

factors like gender, frequency of drinking, amount of alcohol, drinking pattern, types of alcohol and tendency of weight gain (Traversy G and Chaput JP, 2015).

There were more frequent smokers in obese subjects (26%) than non-obese subjects (14%), a significant positive association was found between smoking with weight, BMI, WC and WHR. The possible mechanism connecting obesity and smoking partly comprehended and it was also contradictory. Some studies suggested that heavy smokers (i.e., those smoking a greater number of cigarettes/d) have greater body weight than do light smokers (Bamia C et.al., 2004; John U et.al., 2005; Chilero A et.al., 2007) and that there is a clustering of smoking, obesity, and lower socioeconomic status, at least in developed countries (Wild SH and Byrne CD, 2006). Finally, there is increasing evidence that smoking affects body fat distribution and that it is associated with central obesity and insulin resistance (Eliasson B, 2003 and Houston TK et.al., 2006).

On the other hand, contradictory evidences suggested that nicotine acutely increases energy expenditure (EE) (Hofstetter A et.al., 1986) and could reduce appetite, which likely explains why smokers tend to have lower body weight than do non-smokers and why smoking cessation is frequently followed by weight gain (Williamson DF et.al., 1991; Ward KD, Klesges RC and Vander Weg MW, 2001). Moreover, a belief popular among both smokers and nonsmokers is that smoking is an efficient way to control body weight (Potter BK et. al., 2004). The number of cigarettes smoked rises, BMI, obesity levels and blood pressures decrease (Gumus, 2013). Another study on 499,504 middle aged adults in UK revealed that current smokers were less likely to be obese than never smokers (adjusted OR 0.83 95% CI 0.81-0.86) and former smokers were more likely to be obese than both current smokers (adjusted OR 1.33 95% CI 1.30-1.37) and never smokers (adjusted OR 1.14 95% CI 1.12-1.15). Among smokers, the risk of obesity increased with the amount smoked and former heavy smokers were more likely to be obese than former light smokers (adjusted OR 1.60, 95% 1.56-1.64, $p<0.001$). Risk of obesity fell with time from quitting. After 30 years, former smokers still had higher risk of obesity than

current smokers but the same risk as never smokers (Shadrach D, Daniel FM and Jill P, 2015).

Tea and coffee intake were positively associated with weight and BMI, 63% of obese and 34% of non-obese were having tea more frequently. Studies suggested that caffeine has a anti-obesity effect, it increases the metabolic rate and fat oxidation; which is contradictory to these results (Zheng G et. al., 2004; Westerterp-Plantenga, Diepvens, Joosen, Bérubé-Parent, and Tremblay, 2006; Kristel D et.al., 2007). However, in present study subjects were having tea and coffee along with sugar and milk, which added extra calories to daily diet; this could be the reason of positive association of BMI and intake of tea and coffee.

Several studies have established association between depression and obesity (Nina S et. al., 2016). A meta-analysis on 17 cross sectional studies revealed a significant positive association between depression and obesity in the general population, which appeared to be more noticeable among women (De Wit lenore et. al., 2010). Unlike present study revealed 20% of non-obese subjects were suffered from borderline to severe depression as compared to 8% obese subjects and also no significant association was found between obesity and depression.

In the present study results demonstrated that obese subjects had delayed satiety; means they consumed more food as compared to non-obese subjects and also a significant difference was noted in energy and fat intake between obese and non-obese individuals. Above all, all the anthropometric parameters positively associated with the intake of macronutrients, intake of sodium, total dietary fibers, MUFA and PUFA. Studies suggested the possible mechanism of hunger and satiety regulation in obese individuals. A hormone called leptin is made by adipose cells contributing in regulation of energy balance through hunger inhibition (Zhang F et.al., 1997). Ghrelin in contrast is a fast-acting hormone, apparently playing a role in meal initiation (Klok MD, Jakobsdottir S and Drent ML, 2007). In obesity, a decreased sensitivity to leptin occurs, resulting in an inability to detect satiety despite high energy stores (Brennan AM and Mantzoros CS, 2006).

Cardiovascular diseases (CVDs), the leading cause of morbidity and mortality in the western World, are now emerging public health challenges in developing countries (WHO, 2002). The association between dyslipidemia, obesity and hypertension is well established (Nguyen NT et.al., 2008; Brown CD et.al., 2008) and all have been found to be major risk factors for the development of CVD, a leading cause of visits to physicians (Gordon H, 2000) and cause of death (Kadiri S et.al., 1999). In the present study a significant difference in obese and non-obese subjects in all lipid profile parameters were seen except HDL cholesterol. Obese subjects have higher values of TC, TG and LDL than non-obese. Similar results was seen in a study conducted at Abakaliki, South Eastern Nigeria where both overweight and obese subjects had significantly higher plasma lipids when compared with individuals that were either underweight or normal weight and also the obese subjects had significantly higher TG in comparison with overweight subjects (Ugwuja E, Ogbonna N, Nwibo, A and Onimawo I 2013).

Obesity was earliest expressed as a low-grade inflammatory condition more than a decade ago (Heilbronn and Campbell, 2010). Low-grade inflammation is an attribute of obesity in which adipose tissue liberates various inflammatory mediators. Although the source of these mediators within adipose tissue is not apparent, however infiltrating macrophages seem to be especially important, though adipocytes themselves play a role. Obese people have higher circulating concentrations of many inflammatory markers than lean individuals and these are supposed to play as causative agents in insulin resistance and other metabolic disturbances (Calder PC et al., 2013). Two eminent scientists and their team looked for an inflammatory factor contributing in the onset of insulin resistance, obesity, and diabetes; recognized bacterial lipopolysaccharide (LPS) as a triggering factor (Cani PD, Amar J el.al., 2007). The gut microbiota is also involved in obesity-induced inflammation via LPS-related endotoxemia that induces cytokine secretion and insulin resistance (Pereira SS and Alvarej L JI, 2014). In line with these findings present study revealed that obese had significantly higher circulating plasma

LPS levels by 8.52 pg/ml and also LPS levels was positively associated with weight, BMI, WC, WHR and percent body fat. In 2010 Cani and team observed metabolic endotoxemia could modulate gut microbiota (Cani PD and Delzenne NM, 2010).

Diet-induced obesity strongly altered gut microbiota composition with reduced Bifidobacterium spp. and Bacteroides-related bacteria, Eubacterium rectale-Clostridium coccoides group content (Cani PD, Amar J et.al., 2007: Cani PD, Neyrinck AM et.al., 2007). Backhed and Fredrik said that obesity is associated with decreased microbial diversity in the human gut with lower levels of *Bacteroidetes* (Backhed and Fredrik, 2009) is contradictory to present study where obese had more *Bacteroides* than non-obese, however non-obese gut was colonized with more *Bifidobacteria* and *Lactic acid bacteria*. A reciprocal association was seen between *Lactic acid bacteria* with age, weight, BMI, WC, WHR, percent body fat, alcohol intake, TC, LDL, TC/HDL, and LDL/HDL; clearly indicating obese subjects had lower numbers of *LAB* when compared to non-obese; unlike *Bacteroides* which was positively associated with all of above parameters. Both of these bacteria found to be a significant contributor in development of obesity upon further analysis.

From above discussed paragraph it can be concluded that gut microbiota and obesity complexly associated which needs to be followed by a line of investigation in order to understand this liasoning.

Concluding Remarks

Present phase of the study assures that there were statistically significant differences in obese and non-obese subjects with respect to gut microbiota, LPS, heredity, atherogenic indices, defecation, diet, hunger-satiety regulation, smoking and drinking alcohol.

Obese were at higher risk of developing metabolic syndrome when compared to non-obese.

In the perspective to this scenario, obesity needs to be addressed immediately. It can be achieved by modulating gut microbiota by means of prebiotics and probiotics, which may bring out positive changes in obesity status.

Phase IV **Effect of fructooligosaccharide (FOS) supplementation on anthropometry profile, blood pressure, defecation profile, hunger and satiety, psychological depression, dietary intakes, lipemic parameters, plasma LPS level and gut microbiota (*LAB, bifidobacteria, bacteroides and clostridium*) in obese grade-I adults.**

Obesity is a chronic disease and has negative effects on all the systems of the body. Obesity greatly raises the risk of having other co-morbidities. Hence, obesity needs to be treated timely. Now a days prebiotic and probiotic are coming up with improved health including obesity.

Fructooligosaccharides is one of the promising prebiotic food which have a positive health implications. The prebiotic approach dictates that non-viable food components are specifically fermented in the colon by indigenous bacteria thought to be of positive value, e.g. *bifidobacteria, lactobacilli*. Various data have shown that fructo-oligosaccharides (FOS) are specifically fermented by bifidobacteria. (Gibson GR, 1980).

With these considerations, the present study was designed to observe the effect of FOS supplementation on obese individual for their anthropometric indices, blood pressure, defecation profile, hunger and satiety scores, depression status, atherogenic profile, plasma LPS level and gut microbiota (*LAB, bifidobacteria, bacteroides and clostridium*).

For achieving the desired objectives, a total of 116 obese subjects were enrolled from 10 different private banks of urban Vadodara and randomly divided in two groups i.e. experimental and placebo groups which received FOS (20 g) and dextrose (20 g) respectively for 90 days. Post intervention the sample size remained as 51 in experimental group and 32 in placebo group, after considering the dropouts due to various reasons.

The results of this phase of the study are presented in following sub sections:

5.6.1 Anthropometric profile of obese subjects before and after intervention with FOS

5.6.2 Blood pressure of obese subjects before and after intervention with FOS
5.6.3 Defecation profile of obese subjects before and after intervention with FOS
5.6.4 Hunger scores of obese subjects before and after intervention with FOS
5.6.5 Satiety scores of obese subjects before and after intervention with FOS
5.6.6 Depression status of obese subjects before and after intervention with FOS
5.6.7 Dietary intakes of obese subjects before and after intervention with FOS
5.6.8 Atherogenic indices of obese subjects before and after intervention with FOS
5.6.9 Endotoxemia in obese subjects before and after intervention with FOS
5.6.10 Gut microflora counts in terms of *Bifidobacteria, LAB, Bacteroides and Clostridium* of obese subjects before and after intervention with FOS

Section 5.6.1 Anthropometric profile of obese subjects before and after intervention with FOS

Both the placebo and control group was statistically same before the intervention. Experimental group showed significant reduction in weight (1.44%) and BMI (1.32%) after intervention with FOS for 90 days. A significant reduction was also seen in waist circumference by 2.18%, WHR by 2.15% and percent body fat by 2.92% after intervention. (Table 5.32)

Section 5.6.2 Blood pressure of obese subjects before and after intervention with FOS

As shown in Table 5.33, both the placebo and experimental group was similar before the intervention. Systolic blood pressure values significantly reduced by 1.79% ($p<0.001$), where diastolic blood pressure values also reduced non-significantly in experimental group subjects after intervention. (Fig. 5.14)

Table 5.32: Anthropometric profile of obese young adults before and after intervention

Parameters		Placebo group (n=32)	Experiment group (n=51)	Student 't' Test (p-value)
Weight (kg) (Mean±SD)	Pre intervention	76.92±11.73	80.34±8.56	1.53^{NS} (0.130)
	Post intervention	76.71±11.39	79.18±8.66	1.11^{NS} (0.268)
	Paired 't' Test	**0.71^{NS} (0.481)**	**3.87*** (0.000)**	
	% decrease	**0.27%↓**	**1.44%↓**	
BMI(kg/m^2) (Mean±SD)	Pre intervention	27.23±1.55	27.86±1.52	1.83^{NS} (0.070)
	Post intervention	27.17±1.53	27.49±1.65	0.89^{NS} (0.375)
	Paired 't' Test	**0.55^{NS} (0.581)**	**3.32** (0.002)**	
	% decrease	**0.22%↓**	**1.32%↓**	
WC (cms) (Mean±SD)	Pre intervention	95.38±7.45	97.12±7.49	1.03^{NS} (0.305)
	Post intervention	94.30±6.82	95.00±7.65	0.42^{NS} (0.675)
	Paired 't' Test	**2.23* (0.033)**	**5.74*** (0.000)**	
	% decrease	**1.13%↓**	**2.18%↓**	
HC (cms) (Mean±SD)	Pre intervention	102.41±7.28	103.82±5.84	0.97^{NS} (0.332)
	Post intervention	101.64±7.58	103.43±5.69	1.22^{NS} (0.225)
	Paired 't' Test	**2.25* (0.031)**	**1.94^{NS} (0.0.58)**	
	% decrease	**0.75%↓**	**0.37%↓**	
WHR (Mean±SD)	Pre intervention	0.92±0.04	0.93±0.04	0.31^{NS} (0.753)
	Post intervention	0.92±0.04	0.91±0.05	0.98^{NS} (0.330)
	Paired 't' Test	**0.19^{NS} (0.849)**	**4.01*** (0.000)**	
	% decrease	**---**	**2.15%↓**	
Percent body fat (Mean±SD)	Pre intervention	31.46±5.06	31.79±3.86	0.33^{NS} (0.740)
	Post intervention	31.63±5.04	30.86±3.67	0.79^{NS} (0.428)
	Paired 't' Test	**0.41^{NS} (0.685)**	**2.80** (0.007)**	
	%increase/decrease	**0.54%↑**	**2.92%↓**	

Note: NS = non-significant, $p < 0.05$: *, $p < 0.01$: **, $p < 0.001$: ***; 2 tailed

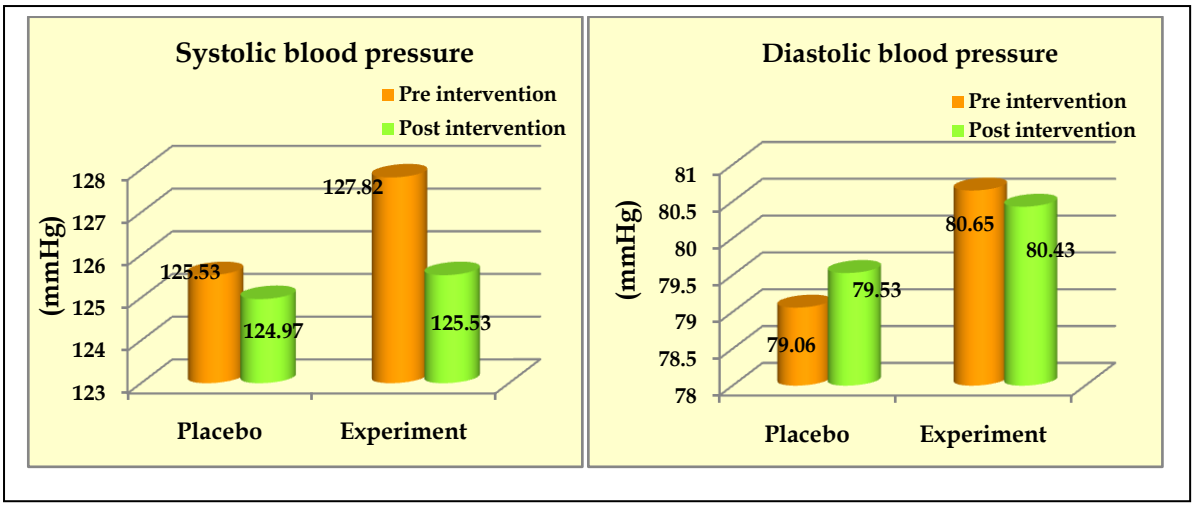


Fig. 5.14: Blood Pressure of obese subjects before and after intervention with FOS

Table 5.33: Blood pressure of obese subjects before and after intervention with FOS

Blood pressure (mmHg)		Placebo group (n=32)	Experiment group (n=51)	Student 't' Test (p-value)
Systolic blood pressure (Mean±SD)	Pre intervention	125.53±8.76	127.82±8.70	1.16^{NS} (0.248)
	Post intervention	124.97±8.26	125.53±6.94	0.33^{NS} (0.740)
	Paired 't' Test	**0.69^{NS} (0.490)**	**4.35^{***} (0.000)**	
	% decrease	**0.44%↓**	**1.79%↓**	
Diastolic blood pressure (Mean±SD)	Pre intervention	79.06±7.35	80.65±7.46	0.94^{NS} (0.347)
	Post intervention	79.53±5.73	80.43±6.14	0.66^{NS} (0.507)
	Paired 't' Test	**0.72^{NS} (0.476)**	**0.45^{NS} (0.650)**	
	% increase/decrease	**0.59%↑**	**0.27%↓**	

Note: NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed

Section 5.6.3 Defecation profile of obese subjects before and after intervention with FOS

Defecation profile of both the placebo and control group was statistically same before the intervention. This significantly improved after intervention with FOS in experimental group subjects. As per subjects perception 88.24% subjects reported absence of constipation after intervention in experimental group. Frequency of passing the stool in a day increased significantly from one time to two-three times in a day and fecal output also increased significantly in experimental group subjects post intervention indicating better cleaning of

stomach. Hardness and odor of stool reduced significantly ($p<0.001$) after intervention in experimental group subjects. Feeling after defecation significantly improved from bad to fine post intervention in experimental group. Degree of constipation reduced significantly ($p<0.001$) from moderate to absence of constipation after intervention in experimental group subjects. Overall defecation profile improved significantly however placebo group remained unaffected post intervention (Table 5.34.1 and 5.34.2).

Table 5.34.1: Defecation profile of obese subjects before and after intervention with FOS

Defecation Profile		Placebo Group (n=32)	Experiment Group (n=51)	χ^2 Value
Constipation (As Per Subjects Perception)	**Pre Intervention**			
	Present	13(40.62)	21(41.18)	**0.00^{NS} (0.960)**
	Absent	19(59.38)	30(58.82)	
	Post intervention			
	Present	11(34.38)	6(11.76)	**6.09* (0.013)**
	Absent	21(65.62)	45(88.24)	
	χ^2 Value	**0.26^{NS} (0.608)**	**11.22*** (0.000)**	
Frequency (times / day)	**Pre Intervention**			
	1	22(68.75)	29(56.86)	**1.15^{NS} (0.281)**
	2-3	10(31.25)	22(43.14)	
	Post intervention			
	1	17(53.12)	12(23.53)	**7.48** (0.006)**
	2-3	15(46.88)	39(76.47)	
	χ^2 Value	**1.61^{NS} (0.203)**	**11.67*** (0.000)**	
Quantity of Stool	**Pre Intervention**			
	Small	10(31.25)	11(21.57)	**0.96^{NS} (0.326)**
	Middle to large	22(68.75)	40(78.43)	
	Post intervention			
	Small	7(21.87)	3(5.88)	**4.68* (0.030)**
	Middle to large	25(78.13)	48(94.12)	
	χ^2 Value	**0.70^{NS} (0.399)**	**5.24* (0.021)**	
Hardness of stool	**Pre Intervention**			
	Very hard to hard	13(40.63)	24(47.05)	**0.32^{NS} (0.568)**
	Medium to soft	19(59.38)	27(52.95)	
	Post intervention			
	Very hard to hard	13(40.62)	7(13.72)	**7.68** (0.005)**
	Medium to soft	19(59.38)	44(86.28)	
	χ^2 Value	**0.00^{NS} (1.00)**	**13.26*** (0.000)**	

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, $p < 0.05$: *, $p < 0.01$: **, $p < 0.001$: ***

Table 5.34.2: Defecation profile of obese subjects before and after intervention with FOS

Defecation Profile		Placebo Group (n=32)	Experiment Group (n=51)	χ^2 Value
Color of Stool	**Pre Intervention**			
	Blackish to middle	28(87.50)	49(96.08)	**2.13^{NS} (0.144)**
	Yellowish	4(12.50)	2(3.92)	
	Post intervention			
	Blackish to middle	30(93.75)	49(96.08)	**0.22^{NS} (0.631)**
	Yellowish	2(6.25)	2(3.92)	
	χ^2 Value	**0.72^{NS} (0.394)**	**0.00^{NS} (1.00)**	
Odor of Stool	**Pre Intervention**			
	Strong	10(31.25)	11(21.56)	**0.96^{NS} (0.326)**
	Medium to weak	22(68.75)	40(78.44)	
	Post intervention			
	Strong	8(25)	0(0)	**13.94*** (0.000)**
	Medium to weak	24(75)	51(100)	
	χ^2 Value	**0.30^{NS} (0.581)**	**12.20*** (0.000)**	
Feeling after defecation	**Pre Intervention**			
	Bad	6(18.75)	13(25.49)	**0.50^{NS} (0.479)**
	Fine	26(81.25)	38(74.51)	
	Post intervention			
	Bad	5(15.62)	1(1.96)	**5.40* (0.020)**
	Fine	27(84.38)	50(98.04)	
	χ^2 Value	**0.10^{NS} (0.742)**	**11.80*** (0.000)**	
Regular use of Laxatives	**Pre Intervention**			
	Yes	6(18.75)	10(19.60)	**0.00^{NS} (0.923)**
	No	26(81.25)	41(80.40)	
	Post intervention			
	Yes	6(18.75)	5(9.80)	**1.35^{NS} (0.244)**
	No	26(81.25)	46(90.20)	
	χ^2 Value	**0.00^{NS} (1.00)**	**1.93^{NS} (0.164)**	
Defecation Profile	**Pre Intervention**			
	Constipated	9(28.12)	14(27.45)	**0.00^{NS} (0.947)**
	Normal defecation	23(71.87)	37(72.55)	
	Post intervention			
	Constipated	1(3.12)	0(0)	**1.58^{NS} (0.206)**
	Normal defecation	31(96.88)	51(100)	
	χ^2 Value	**7.46** (0.006)**	**16.06*** (0.000)**	
Degree of constipation	**Pre Intervention**			
	Moderate	0(0)	1(1.96)	**0.00^{NS} (0.947)**
	Mild	9(28.12)	13(25.49)	
	No constipation	23(96.88)	37(72.55)	
	Post intervention			
	Mild	1(3.12)	1(0)	**0.12^{NS} (0.727)**
	No constipation	31(96.88)	51(100)	
	χ^2 Value	**7.46** (0.006)**	**13.35*** (0.000)**	

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, $p < 0.05$: *, $p < 0.01$: **, $p < 0.001$: ***

Section 5.6.4 Hunger scores of obese subjects before and after intervention with FOS

Table 5.35 depicts significant reduction in the appetite by 12.83% and 15.12% during lunch and dinner time respectively in experimental group subjects after intervention. Although overall a non-significant increase in appetite in terms of the total mean hunger scores was found to be 0.75% and 3% in experimental and placebo group subjects respectively after intervention.

Table 5.35: Hunger scores of obese subjects before and after intervention with FOS

Meal time		Placebo group (n=32)	Experiment group (n=51)	Students 't' Test (p-value)
Breakfast	Pre intervention	4.00±0.67	3.86±1.04	0.66^{NS} (0.509)
	Post intervention	3.91±0.81	4.02±0.81	0.61^{NS} (0.539)
	Paired 't' Test	**0.90^{NS} (0.374)**	**1.09^{NS} (0.281)**	
	% decrease/increase	**2.25%↓**	**4.14%↑**	
Lunch	Pre intervention	3.72±0.77	3.35±0.95	1.82^{NS} (0.072)
	Post intervention	3.69±0.59	3.78±0.78	0.60^{NS} (0.550)
	Paired 't' Test	**0.19^{NS} (0.845)**	**3.00** (0.004)**	
	% decrease/increase	**0.80%↓**	**12.83%↑**	
Evening	Pre intervention	4.00±1.04	4.27±0.80	1.34^{NS} (0.182)
	Post intervention	4.16±1.08	4.35±0.77	0.96^{NS} (0.336)
	Paired 't' Test	**1.22^{NS} (0.231)**	**0.64^{NS} (0.522)**	
	% increase	**4.0%↑**	**1.87%↑**	
Dinner	Pre intervention	3.50±1.07	3.24±1.10	1.07^{NS} (0.287)
	Post intervention	3.78±0.94	3.73±0.77	0.29^{NS} (0.770)
	Paired 't' Test	**1.55^{NS} (0.130)**	**3.03** (0.004)**	
	% increase	**8.0%↑**	**15.12%↑**	
Mean hunger scores	Pre intervention	4.00±0.56	4.00±0.72	0.13^{NS} (0.890)
	Post intervention	3.88±0.47	3.97±0.56	0.73^{NS} (0.466)
	Paired 't' Test	**1.11^{NS} (0.274)**	**2.77^{NS} (0.783)**	
	% decrease	**3.0%↓**	**0.75%↓**	

Note: Figures in parenthesis represent percent of subjects; NS = non-significant, $p < 0.05$: *, $p < 0.01$: **, $p < 0.001$: *** Hunger scores 1 – 5, where 1= Famished, starving 2= Headache, weak, cranky, low energy , 3= Want to eat now, stomach growls and feels empty, 4= Hungry - but could wait to eat, starting to feel empty but not there yet, 5= Not hungry, not full

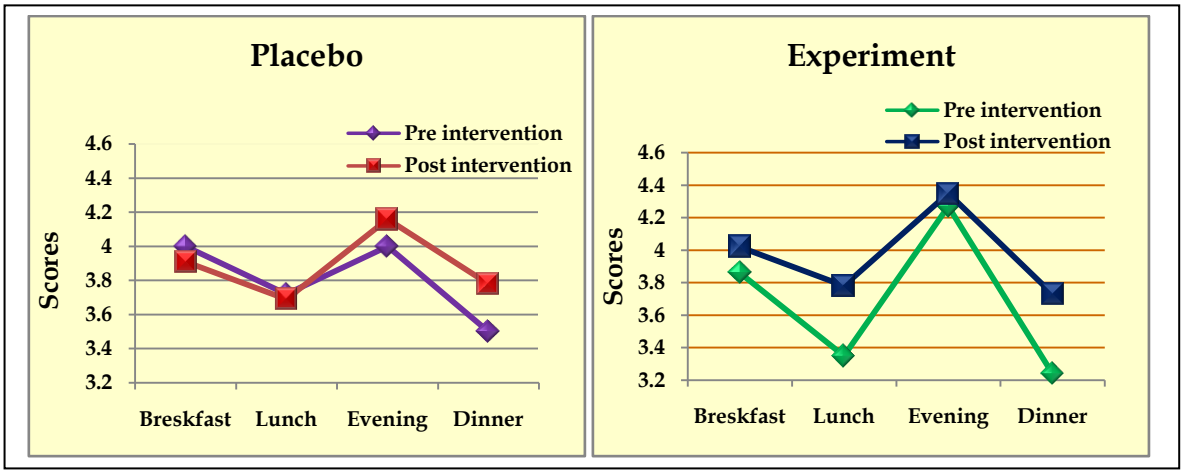


Fig. 5.15: Hunger scores of obese subjects before and after intervention with FOS

Section 5.6.5 Satiety scores of obese subjects before and after intervention with FOS

A significant improvement was seen in achieving early satiety during most of the meal times. Highest reduction in satiety scores was observed at the lunch time by 8.94%, followed by dinner (8.23%) and breakfast (3.58%) in experimental group subjects. (Table 5.36)

Table 5.36: Satiety scores of obese subjects before and after intervention with FOS

Meal time		Placebo group (n=32)	Experiment group (n=51)	Students 't' Test (p-value)
Breakfast	Pre intervention	6.41±0.75	6.41±0.87	0.02^{NS} (0.977)
	Post intervention	6.41±0.87	6.18±0.74	1.28^{NS} (0.203)
	Paired 't' Test	**0.00^{NS} (1.00)**	**1.69^{NS} (0.096)**	
	% decrease	**----**	**3.58%↓**	
Lunch	Pre intervention	6.84±0.76	7.04±0.97	0.95^{NS} (0.340)
	Post intervention	6.78 ±0.75	6.41±0.80	2.08* (0.040)
	Paired 't' Test	**0.49^{NS} (0.625)**	**4.78*** (0.000)**	
	% decrease	**0.87%↓**	**8.94%↓**	
Evening	Pre intervention	6.09±0.64	6.02±0.70	0.48^{NS} (0.631)
	Post intervention	6.09±0.64	6.02±0.88	0.41^{NS} (0.682)
	Paired 't' Test	**0.00^{NS} (1.00)**	**0.00^{NS} (1.00)**	
	% decrease/increase	**----**	**-----**	
Dinner	Pre intervention	7.38±1.00	7.29±1.08	0.34^{NS} (0.735)
	Post intervention	7.31±0.89	6.69±1.01	2.86** (0.005)
	Paired 't' Test	**0.37^{NS} (0.712)**	**3.16** (0.003)**	
	% decrease	**0.94%↓**	**8.23%↓**	
Mean satiety scores	Pre intervention	5.69±0.99	5.53±0.70	0.70^{NS} (0.480)
	Post intervention	6.65 0.47	6.32±0.64	2.46* (0.016)
	Paired 't' Test	**5.13*** (0.000)**	**6.33*** (0.000)**	
	% increase	**16.87%↓**	**14.28%↓**	

Note: NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; Satiety scores 5 –10, where 5= Not hungry, not full, 6 = Feeling satisfied, stomach feels full and comfortable, 7 = Feeling full, definitely don't need more food, 8 = uncomfortably full, 9 = Stuffed, very uncomfortable, 10 = Bursting, painfully full

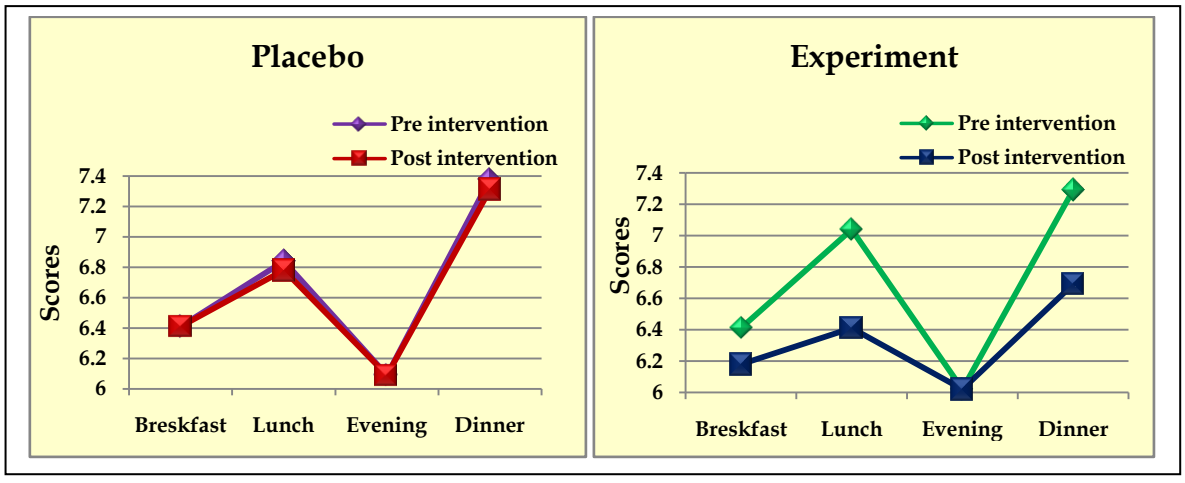


Fig. 5.16: Satiety scores of obese subjects before and after intervention With FOS

Section 5.6.6 Depression status of obese subjects before and after intervention with FOS

A significant improvement was observed in mean depression scores in experimental (29.24%) as compared to placebo (1.36%) group subjects, although scores were in the reference range for both the groups pre and post intervention. (Table 5.37)

Table 5.37: Depression status of obese subjects before and after intervention with FOS

Depression profile	Placebo group (n=32)	Experimental group (n=51)	Students 't' Test (p-value)
Pre intervention	8.81±5.92	9.16±7.13	0.22^{NS} (0.820)
Post intervention	8.69±6.19	6.49±6.30	1.55^{NS} (0.124)
Paired 't' Test	**0.37^{NS} (0.712)**	**3.86*** (0.000)**	
% decrease	**1.36↓**	**29.24%↓**	

Note: NS = non-significant, p < 0.05: *, p < 0.01: **, p < 0.001: ***; 2 tailed

Section 5.6.7 Dietary intakes of obese subjects before and after intervention with FOS

Table 5.38 reveals that consumption of FOS significantly reduced the mean dietary intakes of energy (8.58%), CHO (8.55%), protein (8.39%), and fat (10.42%) in experimental group subjects. Soluble dietary fibre and total dietary fiber intake increased significantly in experimental group. This could be because of supplementation with FOS, considered as hundred percent soluble dietary fibers.

Table 5.38: Dietary intakes of obese subjects before and after intervention

Parameters		Placebo group (n=31)	Experiment group (n=50)	Student 't' Test (p-value)
Energy (Kcals)	**Pre Intervention**	2710.56±568.52	2776.18±548.77	0.52^{NS} (0.602)
	Post intervention	2712.89±561.40	2567.91±508.15	0.87^{NS} (0.478)
	Paired 't' Test	**1.10^{NS} (0.198)**	**4.97*** (0.000)**	
	% increase/decrease	**0.08%↑**	**7.50%↓**	
CHO (g)	**Pre Intervention**	359.96±81.47	364.05±71.45	0.24^{NS} (0.811)
	Post intervention	370.58±79.07	352.91±77.47	1.99^{*} (0.048)
	Paired 't' Test	**2.10* (0.042)**	**4.56*** (0.000)**	
	% increase/decrease	**2.95%↑**	**3.06%↓**	
Protein (g)	**Pre Intervention**	77.90±16.14	82.21±23.83	0.36^{NS} (0.716)
	Post intervention	75.29±15.98	75.31±20.52	0.00^{NS} (0.996)
	Paired 't' Test	**6.39*** (0.000)**	**5.52*** (0.000)**	
	% decrease	**3.35%↓**	**8.39%↓**	
Fat (g)	**Pre Intervention**	101.76±37.32	104.95±39.47	0.89^{NS} (0.371)
	Post intervention	99.23±36.85	94.01±34.98	0.64^{NS} (0.519)
	Paired 't' Test	**2.83** (0.008)**	**5.36*** (0.000)**	
	% decrease	**2.48%↓**	**10.42%↓**	
Soluble Dietary Fibre (g)	**Pre Intervention**	5.29±1.68	5.37±1.85	0.21^{NS} (0.816)
	Post intervention	5.20±1.62	22.98±2.80	13.65^{***} (0.000)
	Paired 't' Test	**0.38^{NS} (0.708)**	**12.89*** (0.000)**	
	% decrease/increase	**1.7%↓**	**327.93%↑**	
Insoluble Dietary Fibre (g)	**Pre Intervention**	15.18±5.78	15.30±6.45	0.08^{NS} (0.991)
	Post intervention	14.50±5.77	15.24±6.00	0.55^{NS} (0.597)
	Paired 't' Test	**1.23^{NS} (0.208)**	**0.18^{NS} (0.846)**	
	% decrease	**4.48%↓**	**0.39%↓**	
Crude Fibre (g)	**Pre Intervention**	9.49±3.50	9.22±2.25	0.42^{NS} (0.564)
	Post intervention	9.76±3.53	9.35±2.30	0.65^{NS} (0.508)
	Paired 't' Test	**0.84^{NS} (0.385)**	**0.73^{NS} (0.445)**	
	% increase	**3.16%↑**	**1.4%↑**	
Total Dietary Fibre (g)	**Pre Intervention**	20.92±7.59	21.31±8.56	0.21^{NS} (0.816)
	Post intervention	20.11 ± 7.58	40.98±7.03	10.98^{***} (0.000)
	Paired 't' Test	**1.05^{NS} (0.257)**	**23.47*** (0.000)**	
	% decrease/increase	**3.87%↓**	**92.30%↑**	

Note: NS = non-significant, $p < 0.05$: *, $p < 0.01$: **, $p < 0.001$: ***; 2 tailed

Section 5.6.8 Atherogenic indices of obese subjects before and after intervention with FOS

Both the groups were statistically similar before intervention. A reduction was seen in overall atherogenic profile where total cholesterol and serum triglycerides decreased significantly by 15.23% and 22% respectively in experimental group after supplementation. Low density lipoproteins and very low density lipoproteins (VLDL) were also reduced significantly by 16.02% and 21.99% in experimental group post supplementation. However, a significant reduction in TC, STG, and VLDL were also observed in placebo group post intervention but to a minor extent. HDL remained unaffected in both the groups. (Table 5.39)

Table 5.39: Atherogenic profile of obese young adults before and after intervention

Parameters (mg/dl)		Placebo Group (n=32)	Experiment Group (n=39)	Student 't' Test (p-value)
Total Cholesterol Mean ±SD	Pre Intervention	184.47±27.42	182.40±32.30	**0.46^{NS} (0.641)**
	Post intervention	161.67±41.55	154.62±44.36	**0.68^{NS} (0.495)**
	Paired 't' Test	**2.33* (0.026)**	**3.15** (0.003)**	
	% decrease	**12.35%↓**	**15.23%↓**	
Serum TG Mean ±SD	Pre Intervention	189.26±60.94	198.92±73.80	**0.59^{NS} (0.551)**
	Post intervention	158.78±40.08	155.14±43.98	**0.36^{NS} (0.719)**
	Paired 't' Test	**2.52* (0.017)**	**3.82*** (0.000)**	
	% decrease	**16.10%↓**	**22.00%↓**	
HDL Mean ±SD	Pre Intervention	36.60±7.75	36.62±7.65	**0.01^{NS} (0.990)**
	Post intervention	36.44±7.82	34.55±6.36	**1.12^{NS} (0.265)**
	Paired 't' Test	**0.07^{NS} (0.940)**	**1.30^{NS} (0.200)**	
	% decrease	**0.43%↓**	**5.65%↓**	
LDL Mean ±SD	Pre Intervention	110.21±30.41	106.01±32.82	**0.72^{NS} (0.473)**
	Post intervention	92.92±38.15	89.02±40.74	**0.41^{NS} (0.681)**
	Paired 't' Test	**1.85^{NS} (0.074)**	**2.03* (0.049)**	
	% decrease	**15.68%↓**	**16.02%↓**	
VLDL Mean ±SD	Pre Intervention	37.66±12.30	39.78±14.76	**0.65^{NS} (0.516)**
	Post intervention	31.76±8.01	31.03±8.79	**0.36^{NS} (0.719)**
	Paired 't' Test	**2.42* (0.021)**	**3.81*** (0.000)**	
	% decrease	**15.66%↓**	**21.99%↓**	
TC/HDL Ratio Mean ±SD	Pre Intervention	5.24±1.32	5.22±1.51	**0.15^{NS} (0.881)**
	Post intervention	4.58±1.47	4.61±1.53	**0.08^{NS} (0.934)**
	Paired 't' Test	**1.70^{NS} (0.098)**	**1.73^{NS} (0.091)**	
	% decrease	**12.59%↓**	**11.68%↓**	
LDL/HDL Ratio Mean ±SD	Pre Intervention	3.18±1.27	3.10±1.38	**0.35^{NS} (0.725)**
	Post intervention	2.68±1.30	2.69±1.38	**0.02^{NS} (0.977)**
	Paired 't' Test	**1.39^{NS} (0.173)**	**1.34^{NS} (0.188)**	
	% decrease	**15.72%↓**	**13.22%↓**	

Note: Level of significance:* p- value <0.05;**p-value<0.01 ***p-value<0.001; NS = Not Significant;

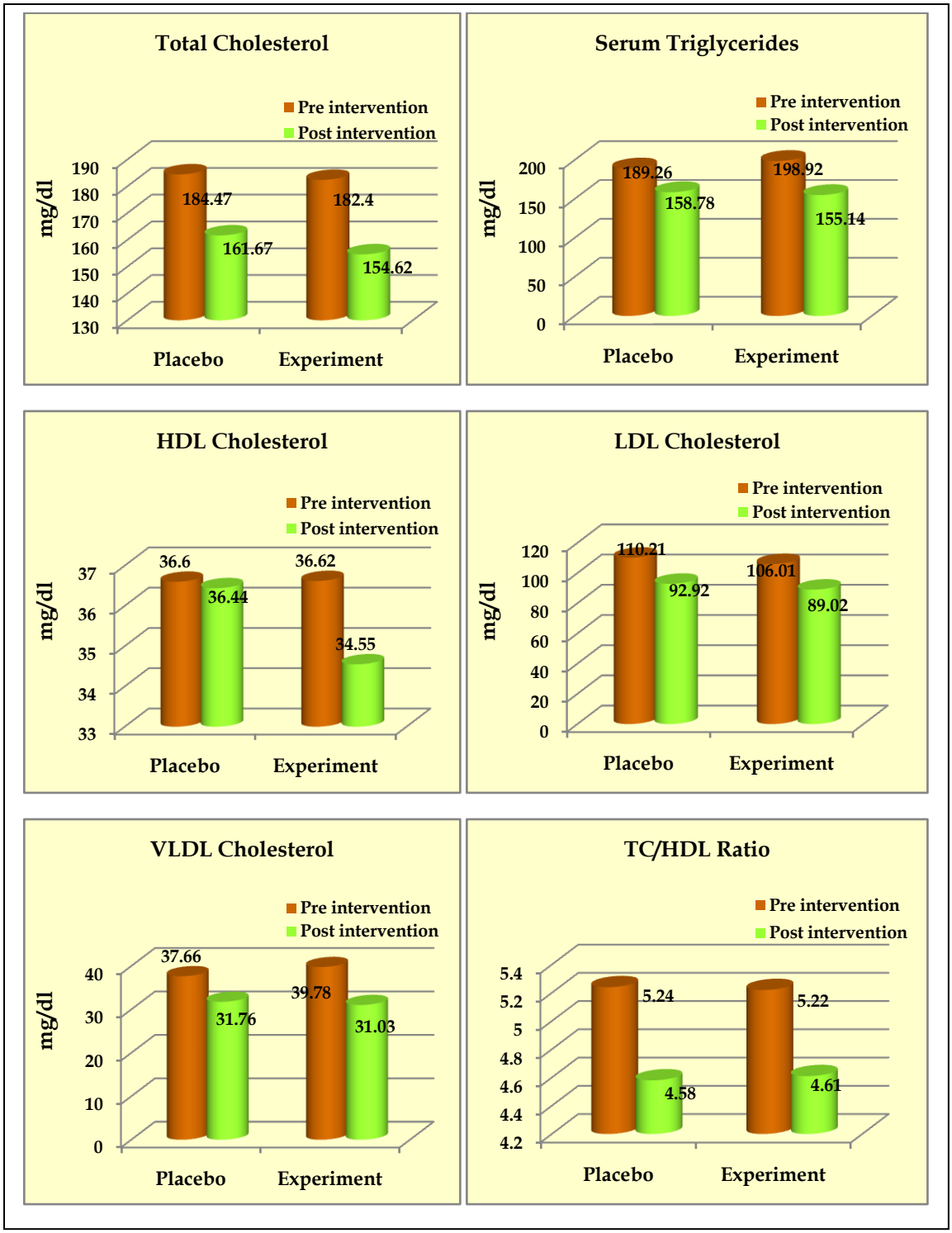


Fig. 5.17: Atherogenic profile of obese subjects before and after intervention with FOS

Section 5.6.9 Endotoxemia in obese subjects before and after intervention with FOS

Table 5.40 depicts that LPS levels reduced non-significantly by 14.5% in placebo group and 4.03% in experimental group post intervention. There was no significant difference was found in the LPS levels of both the group before and after intervention.

Table 5.40: Mean LPS levels (pg/ml) of obese bank employees before and after intervention

	Placebo group (n=25)	Experiment group (n=30)	Student 't' Test (p-value)
Pre Intervention	22.59±15.41	25.03±20.64	**0.48^{NS} (0.835)**
Post intervention	19.31±12.69	24.02±11.59	**1.43^{NS} (0.101)**
Paired 't' Test	**1.04^{NS} (0.321)**	**0.27^{NS} (0.912)**	
% decrease	**14.5%↓**	**4.03%↓**	

NOTE: NS = non-significant.

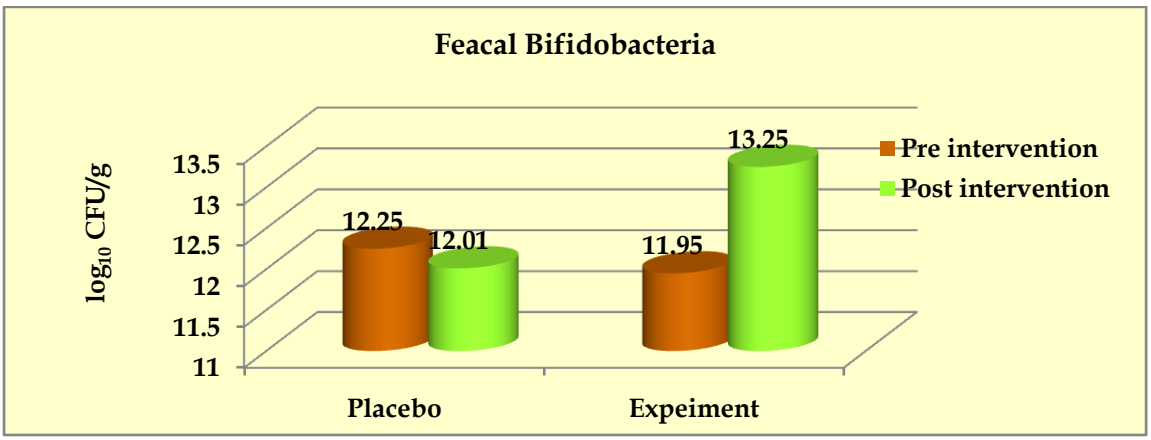
Section 5.6.10 Gut microflora counts in terms of *Bifidobacteria, LAB, Bacteroides and Clostridium* of obese subjects before and after intervention with FOS

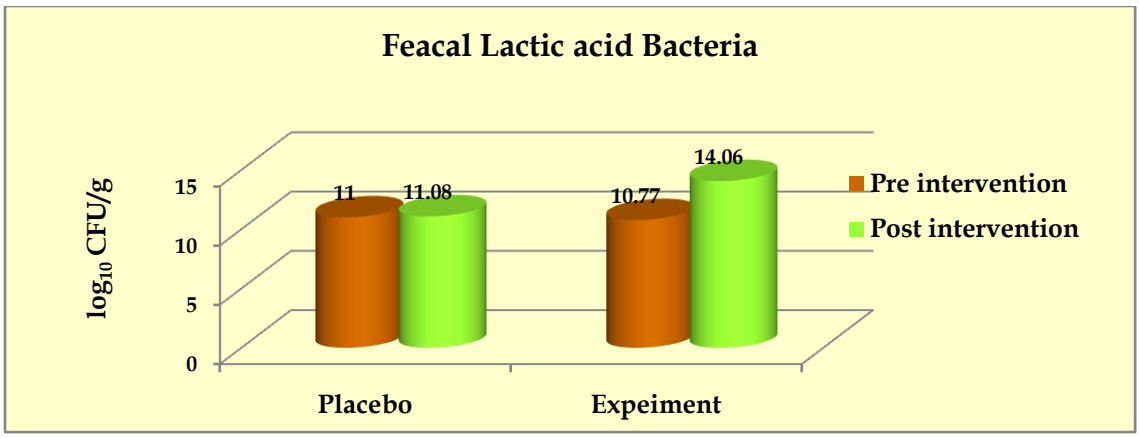
Table 5.41 depicts that both the group were statistically similar with respect to their gut profile before intervention. This 90 days trial place FOS as a successful prebiotic in significantly increased the colonization of the gut with the friendly bacteria like *Bifidobacterium* and *Lactobacillus* by 10.87% and 30.54% respectively. Also, significant reduction was observed in the counts of *Bacteroides* by 11.40 % ($p<0.001$) and non significant reduction in *Clostridium* counts by 1.95%. However, the gut of placebo group also showed a significant reduction in *Bacteroides* counts by 2.76% post intervention.

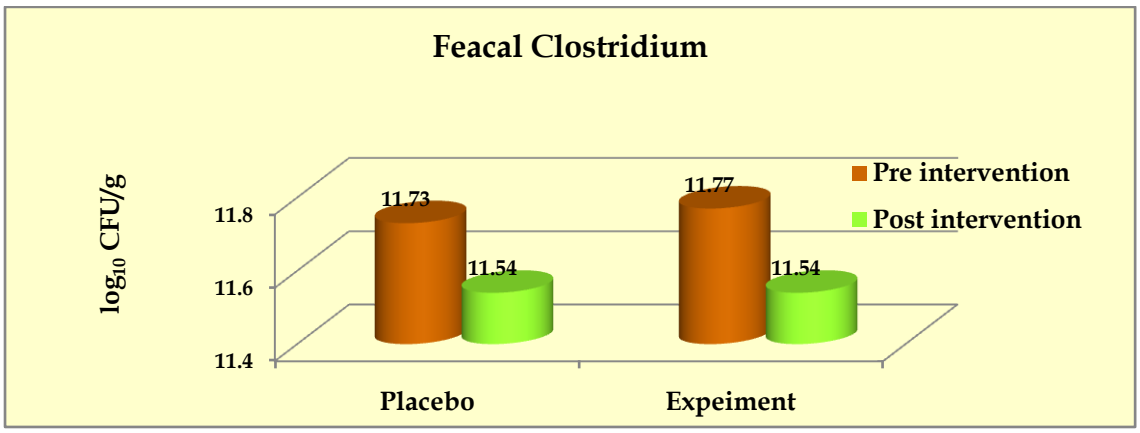
Table 5.41: Gut profile of obese young adults before and after intervention

Parameters log $_{10}$ values (CFU /g)		Placebo group (n=31)	Experiment group (n=50)	Student 't' Test (p-value)
Faecal	**Pre Intervention**	12.25±1.06	11.95±1.11	**1.18^{NS} (0.238)**
Bifidobacteria	**Post intervention**	12.01±1.03	13.25±1.14	**4.91*** (0.000)**
	Paired 't' Test	**0.81^{NS} (0.423)**	**5.06*** (0.000)**	
	% decrease/increase	**1.95%↓**	**10.87%↑**	
Faecal	**Pre Intervention**	11.00±1.07	10.77±1.17	**0.86^{NS} (0.389)**
Lactobacillus	**Post intervention**	11.08±1.36	14.06±1.08	**10.86*** (0.000)**
	Paired 't' Test	**0.25^{NS} (0.799)**	**14.97*** (0.000)**	
	% increase	**0.72%↑**	**30.54%↑**	
Faecal	**Pre Intervention**	11.73±0.47	11.77±0.31	**0.39^{NS} (0.694)**
Clostridium	**Post intervention**	11.54±0.48	11.54±0.31	**0.00^{NS} (0.999)**
	Paired 't' Test	**1.60^{NS} (0.118)**	**3.49** (0.001)**	
	% decrease	**1.61%↓**	**1.95%↓**	
Faecal	**Pre Intervention**	13.75±0.77	13.77±0.96	**0.08^{NS} (0.935)**
Bacteroides	**Post intervention**	13.37±0.36	12.20±0.21	**18.28*** (0.000)**
	Paired 't' Test	**2.29* (0.029)**	**10.98*** (0.000)**	
	% decrease	**2.76%↓**	**11.40%↓**	

NOTE: $p < 0.001$: ***; $p<0.01$:**; NS=Non-significant







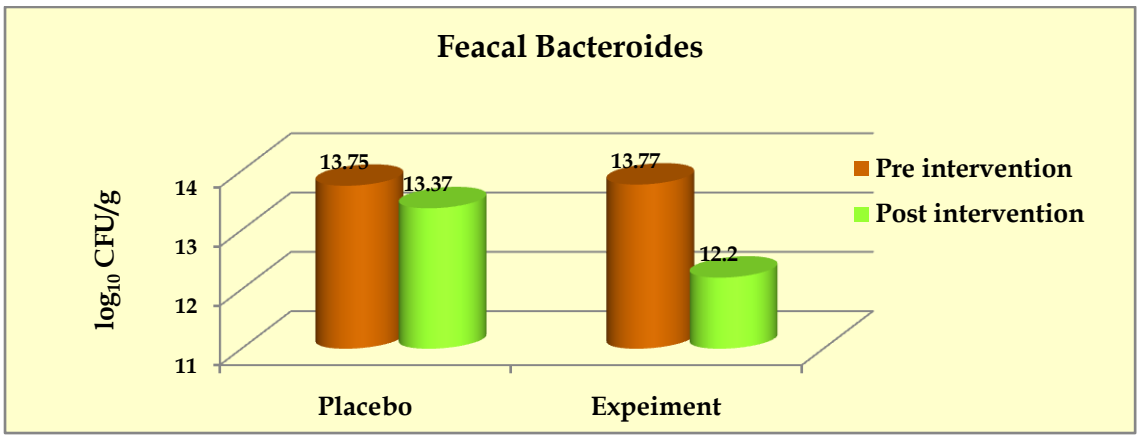


Fig. 5.18: **Gut profile of obese subjects before and after intervention with FOS**

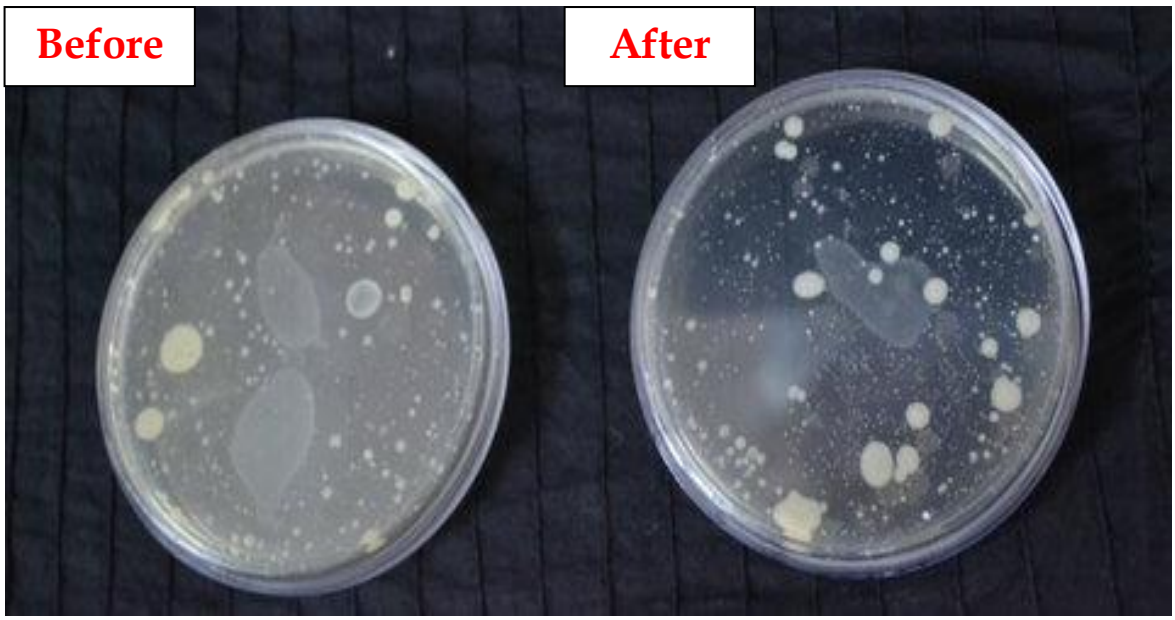


Plate 5.1:Feacal *bifidobacteria* counts before and after FOS supplementation in experiment group

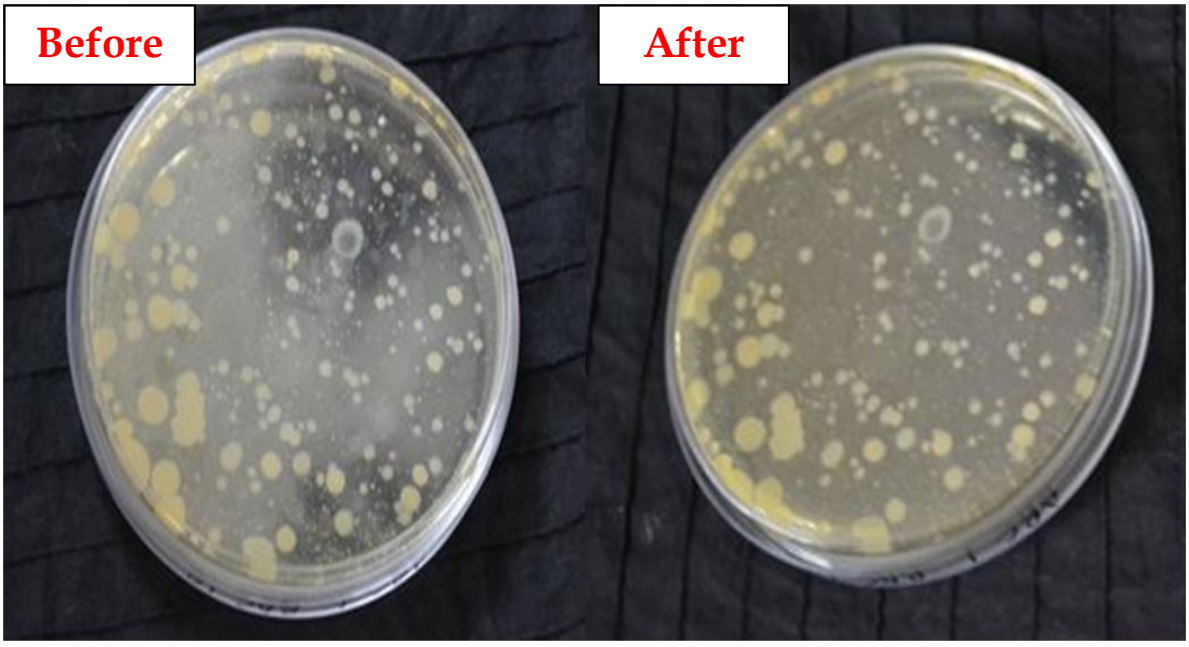


Plate 5.2: Feacal *lactic acid bacteria* counts before and after FOS supplementation in experiment group

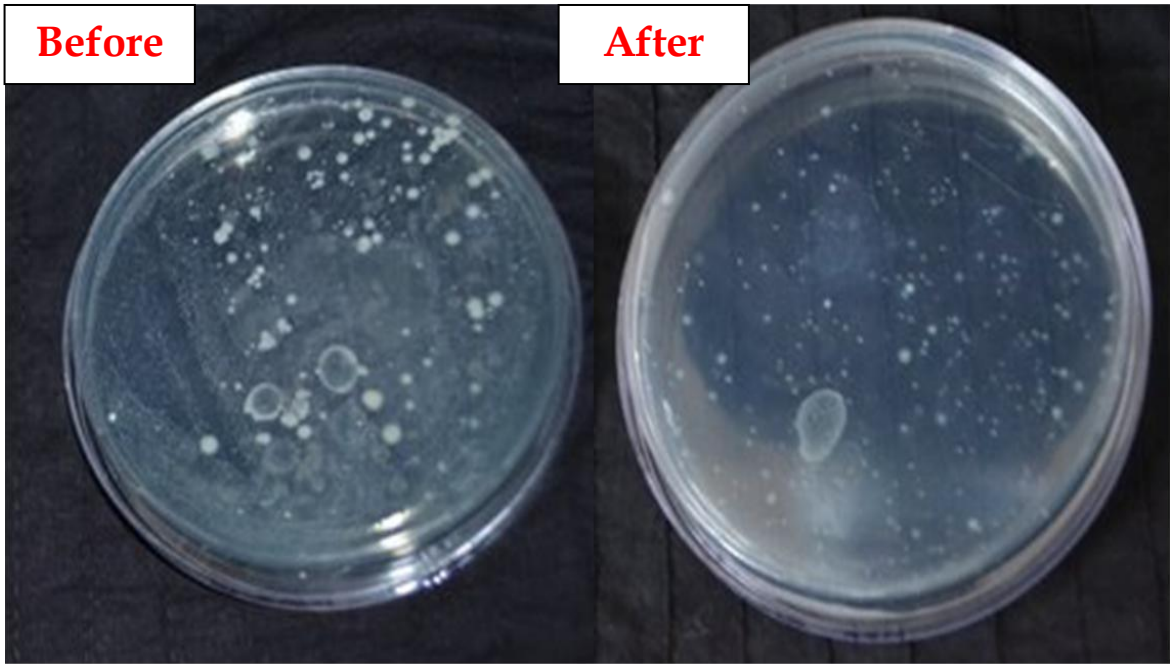


Plate 5.3: Feacal *clostridium* counts before and after FOS supplementation in experiment group

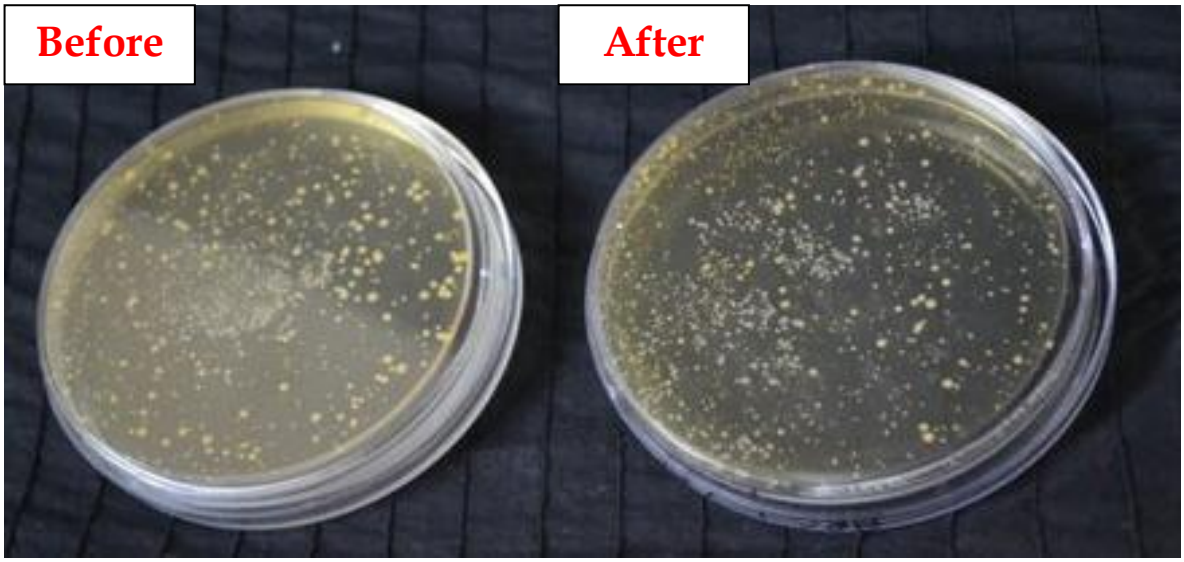


Plate 5.4: Feacal *bacteriodes* counts before and after FOS supplementation in experiment group

Results Highlights of Phase IV

- FOS supplementation showed significant reduction in weight (1.44%) and BMI (1.32%). A significant reduction was also seen in waist circumference by 2.18%, WHR by 2.15% and percent body fat by 2.92% after intervention.
- Systolic blood pressure values significantly ($p<0.001$) reduced by 1.79%, where diastolic blood pressure values also reduced non-significantly in experimental group subjects after intervention.
- Defecation profile significantly improved after intervention in experimental group subjects. Degree of constipation reduced significantly ($p<0.001$) from moderate to absence of constipation after intervention in experimental group subjects.
- A significant reduction was seen in the appetite by 12.83% and 15.12% during lunch and dinner time respectively in experimental group subjects after intervention.
- A significant improvement was seen in achieving early satiety during most of the meal times.
- Significant reduction was seen in mean dietary intakes of energy (7.50%), CHO (3.06%), protein (8.39%), and fat (10.42%) in experimental group subjects with increase in soluble fiber (327.93%) and total dietary fiber (92.30%) intake.
- Atherogenic profile significantly improved in experimental group after intervention.
- LPS levels reduced non-significantly by 4.03% in experimental group post intervention.
- FOS as a successful prebiotic in significantly colonizing the gut with the friendly bacteria like *Bifidobacterium* and *Lactobacillus* and a significant reduction was observed in the counts of *Bacteroides* and non significant reduction in *Clostridium* counts.

DISCUSSION

The present phase of the study established that intake of FOS for 90 days as a successful prebiotic supplement in managing obesity seeing that it reduced the weight and BMI of the subjects and also improved the lipemic profile and gut profile of the subjects.

It was well established that obesity is itself a problem or a precursor of many other co-morbidities. It needs to be addressed timely. The management of obesity has become progressively more imperative to public health practitioners or a dietician. Recent researches are evidence for the key role of gut microbiota; plays in the onset of obesity. They identified the dissimilarity between obese people gut and lean people gut (Zhang H et.al., 2009), also offer a insight of the role of gut microbiota in energy homeostatic, glucose metabolism and lipid storage mechanism (Cani PD and Amar J et.al., 2007).

A bibliographic survey on 61 original articles from PubMed, ScienceDirect, Lilacs and SciElo databases carried out to understand the relationship between gut microbiota, obesity and possible impact of prebiotic and prebiotic concluded that after dietary manipulation with prebiotics and probiotics, the growth of *bifidobacteria* was obtained in 10 studies, involvement with weight reduction, adipogenic effects of diet, intestinal permeability and inflammatory markers (Da Silva, dos Santos, & Bressan, 2013).

Gibson and Roberfroid brought out the concept of prebiotic (Gibson GR and Roberfroid MB, 1995) years back recently improved as "*A non digestible compound that, through its metabolization by microorganisms in the gut, modulates composition and/or activity of the gut microbiota, thus conferring a beneficial physiological effect on the host"* (Bindels LB et. al., 2015).

In view of the fact that gut microbes are accountable for the causation of obesity to some extent, modulation of microbiota can be a potential tool in the prevention and treatment of disease. It was evident that growth of beneficial microbiota and closing the intestinal barrier thus modifies the metabolism of

endotoxin in the blood can be achieved by adding prebiotics in the diet (Everard et al., 2013).

Very few data are available on effect of FOS on weight reduction and other anthropometric indices till date. Findings of a study conducted by Sheth and Gupta (2014) observed significant weight loss and BMI reduction (1.06%) in Sixty five obese subjects working in an industrial setting (BMI 25-31kg/m2, aged 25-55 yrs) supplemented with 12 g FOS for 12 weeks were similar to the findings of present study where a significant ($p<0.001$) reduction in weight (1.44%) and BMI (1.32%) was observed. in addition to this WC, WHR and percent body fat also reduced significantly.

Similar results were obtained by Parnell JA et al where in, oligofructose supplementation, independently from any lifestyle changes, were able to decrease body weight, primarily by losing fat mass, and could help manage caloric intake in overweight and obese adults (Parnell JA, et.a., 2009l). A study conducted by Nakamura Y and team in 2011 assessed efficacy of FOS supplementation on suppression of high fat induced body fat accumulation revealed that body weight and percent body fat were lower in mice fed FOS than in controls. Furthermore, the weight of the visceral adipose tissue, and the weight and triglyceride content of the liver were significantly lower in the high-fat plus FOS group. These results indicate that dietary FOS suppresses high-fat diet-induced body fat accumulation, and inhibit intestinal absorption of dietary fat (Nakamura Y eta l in 2011).

In the present study supplementation of FOS for 90 days also reduced the systolic blood pressure significantly. It may be because weight and BMI of the subjects reduced, as it is a well established fact that obesity and hypertension anti-reciprocally associated with each other. Various mechanisms have been hypothesized to explain the ability of prebiotics to reduce the risk of hypertension. One of the possible mechanisms is via lowering of blood lipid and cholesterol. The cholesterol lowering effect could reduce the stiffness of large arteries and thus could potentially reduced blood pressure (Kathryn E et.al., 2002). In another study, Lairon et al (2005) suggested that the reduction

of obesity upon consumption of prebiotics such as soluble fiber could prevent the elevation of blood pressure. Similar results was found in present study where total cholesterol and serum triglycerides decreased significantly by 15.23% and 22% respectively and reduction in body weight (1.44%) and BMI (1.32%) was also observed in experimental group after supplementation.

Since present study revealed FOS had positive effect on defecation profile. It reduced the hardness of stool and also improved the odor of stool. FOS intervention increased frequency of passing stool and improved feacal output. Similarly a review done on potential health benefits of FOS discussed that intake of FOS between 4-15 g/day will reduce constipation to healthy subjects (Sabater MM and Larque E, et.al., 2009). A randomized, double-blind, placebo-controlled trial for 4 weeks in 120 patients with Parkinson disease (PD) and constipation conducted to check the efficacy of probiotics and prebiotics observed that fermented milk containing probiotics and prebiotics resulted in a higher increase in the number of complete bowel movements (Barichella M and Pacchetti C et.al., 2016). Similar results were seen in a 3-week trial on a population of hemodialysis patients. Patients who had fructooligosaccharides added to their basic dietary protocol had less constipation compared to patients receiving the same dietary protocol without FOS (Cockram DB et.al., 1998). In a 2001 study of seven elderly men, taking 10 grams of FOS for 30 days (after a 30-day control, low-fiber diet period) improved bowel movements, stool output and stool bulk in the colon without any adverse effects (Chen HL and Lu YH et.al., 2000). Subjects having constipation pre-intervention reported normal defecation after consuming FOS and got relieved from constipation post intervention. In a 2011 randomized, double-blind, placebo-controlled study of 20 women and 16 men, significant improvement in overall self-reported gastrointestinal symptoms and bowel habits was observed in the group taking a synbiotic combination of FOS with a combination of probiotics (Nova E et.al., 2011). According to the proposed mechanism by Cummings prebiotics firstly stimulates the microbial growth followed by increase in

bacterial cell mass, and consequently, stimulation of peristalsis through increased bowel content (Cummings JH, 1994).

The present study also reported that after intake of FOS for 90 days appetite of the subjects significantly reduced and early satiety attained at specific meal timings i.e. lunch and dinner. Overall 14.28% increment in satiety scores was seen indicating early satiety in experimental group. Overall dietary intake of all macronutrients and total dietary fibre also reduced. Similar results was reported in a study where intake of 8 g oligofructose twice a day for periods of 2 weeks in 10 healthy subjects (five men and five women) aged 21-39 years, where increase in satiety following breakfast and dinner, reduces hunger and prospective food consumption following dinner, Breakfast, lunch and total energy intake that were significantly reduced as compared to maltodextrin treatment (PD Cani, E Joly, Y Horsmans and NM Delzenne, 2006). Another randomized cross over study (1 week seperation) reported that intake of boiled barley kernels (BK meal) on 19 healthy volunteers aged 20-35 years significantly reduced their energy intake by at lunch by 12%. There was a lower feeling of hunger at breakfast and lunch after BK meal (Elin V Johansson et.al., 2013). The possible mechanism suggested by Everard A and Cani PD in 2007 and 2011, in rodents model stated that oligofructose feeding may promote epithelial L-cell differentiation in the gut, resulting in higher GLP-1 production (Cani PD el.al., 2007 and Everard A et.al., 2011) which in turn reduce food intake and increase satiety in both obese and lean humans (Verdich C et.al., 2001). Parallel to these results several studies have shown that supplementation of FOS lead to increase in SCFA formation in the gut and related beneficial effects on the host metabolism like increased GLP-1 incretin and resultant improved satiety and reduced hunger (Cani PD, Joly E et.al., 2006 and Cani PD, Knauf C et.al., 2006).

The atherogenic profile of the subjects revealed that total cholesterol and serum triglycerides decreased significantly in experimental group after supplementation by 15.23% and 22% respectively. Low density lipoproteins and very low density lipoproteins were also reduced significantly by 16.02%

and 21.99% after intervention in experimental group. Similar results were found in a study conducted by causey et al (Jennifer LC et.al., 2000), who observed a significant reduction in serum TG in subjects with moderate hyperlipidemia given 18 g/d inulin for 3 weeks. In a study conducted on fifty-eight middle aged subjects with moderately raised blood lipid concentrations, subjects consumed 10 g/d of inulin in a powdered form found no significant changes in total LDL or HDL cholesterol either of the groups over the 8 weeks intervention with reduced serum TG levels by 19% after intervention in the inulin treated group (Kim G et.al., 1999), indicating that a higher dose of FOS supplementation for at least 3 months period is required to bring about desirable changes in the LDL cholesterol whereas, lower levels of supplementation for a shorter duration may bring about improvements in serum TG levels which is consistent with the findings of the present study. Although evidence suggests that TG lowering effect of prebiotic occurs via a reduction in VLDL and TG secretion from the liver due to reduction in the activity of all lipogenic enzymes and in the fatty acid synthase, via modification of lipogenic gene expression (Delzenne NM and Kok N, 1998 and Rebecca Wall et.al., 2012), one of the proposed mechanisms is also through the type of beneficial gut microbiota which gets colonized in the gut.

In 2007 Cani PD and team suggested that specific modulation of gut microbiota with prebiotics influences fat mass development and lipid metabolic disorders associated with obesity (Cani et.al., 2007). A study conducted on 40 institutionalized elderly subjects (>60 years) revealed significant 7.4% reduction in mean total cholesterol values and increased counts of Bifidobacteria and lactobacillus after supplementing probiotic curd for 6 weeks (Parnami S and Sheth M, 2011) was again analogous to present study in which bifidobacteria and lactobacillus increased significantly ($p<0.001$) increased by 10.87% and 30.54% respectively. Although, reduction in total cholesterol and serum TG was also observed in placebo group subjects.

The impact of gut microbiota on the progression or slowing down of obesity is not yet fully known. It is believed that obesity is associated with elevated

serum levels of lipopolysaccharide (LPS), which is a component of the cell wall of Gram-negative bacteria (Amar *et al.*, 2011a; Amar *et al.*, 2011b). It was shown that the growth of beneficial microbiota, consequently closing the intestinal barrier and changes in the metabolism of endotoxin in the blood can be modulated by the addition of prebiotics to the diet (Everard *et al.*, 2013). Prebiotics unchanged reached to the large intestine where they are food for the bacteria (Kowalska-Duplaga, 2003). Resistant dextrins derived from potatos had a bifidogenic effect and stimulate the growth of gut microbiota, thus limiting the growth of *Clostridium* strains (Barczynska *et al.*, 2010; Barczynska *et al.*, 2012). Lecerf et.al., in 2012 conducted a study where a mixture of inulin and xylooligosaccharides added to the diet effectively lowered the blood plasma LPS level (Lecerf *et al.*, 2012). A similar result was found where FOS and inulin (10g/d) added to the diet encouraged the growth of bifidobacteria, in particular *Bifidobacterium adolescentis* (Ramirez-Farias *et al.*, 2009).

Several studies have shown that *Bifidobacterium* spp. may be involved in the regulation of gut barrier function and in the diminution of gut lumen endotoxin levels in addition to improvement of mucosal barrier function (Griffiths EA et.al., 2004; Wang Z et.al., 2004 and 2006). Among the probable mechanisms clearing up the development of metabolic endotoxemia, obese and diabetic mice display enhanced intestinal permeability, that participate to the occurrence of LPS-induced inflammation and metabolic disorders (Brun P et.al., 2007; Cani PD et.al., 2008-2009). In accordance with this hypothesis, selective modulation of the gut microbiota by using prebiotics improves gut barrier, reduces metabolic endotoxemia, lowers inflammatory and glucose intolerance (Cani PD et.al., 2007 and 2009). These were parallel to present study findings where LPS levels reduced by 4.03% after supplementation with FOS (20g/d).

In addition, the present study also revealed positive shift in the colonization pattern of the gut, where the counts of *Bifidobacteria* and *Lactobacillus* improved drastically along with reduction in the counts of *Clostridium* and *Bacteroides*. FOS has been extensively studied as a prebiotic and there is ample evidence in

human subjects, including infants, as well as in animal and in vitro studies that, prebiotics significantly increase the proportion of fecal *Bifidobacteria* and sometimes *Lactobacillus* even at fairly low levels of consumption (5–8 g per day) (ILSI Europe, 2011).

These findings are supported by a study (Cani PD and Delzenne NM, 2010), analyzing the effects of fermentable (oligofructose) and non-fermentable (cellulose) fibers on the intestinal microbiota of obese mice, revealing significant increased total content of, *Bifidobacteria* and *Lactobacillus* in the groups that received oligofructose, compared to control. Another study (Shinohara K et.al., 2010) also supports our findings that assessed the effects of oligofructose consumption in healthy volunteers and found increased content of *Bifidobacteria* and *Lactobacillus* in faeces while *Bacteroides* presented reduction. The authors contend that the increased amount of SCFA, resulting from the increase of certain groups of bacteria by prebiotic fermentation, inhibited the growth of *Bacteroides*. Several animal studies that analyzed microbiota modulation reported increased amount of *Bifidobacteria*, which was followed by reduced weight gain. These findings suggest that the reduced counts of *Bifidobacteria* and *Lactobacillus* play a significant role in the development of obesity and its related comorbidities. Also, a 12-week FOS supplementation to obese humans resulted in weight loss and meal related suppression of the orexigenic hormone ghrelin (Parnell JA and Reimer RA 2009).

Concluding Remarks

From the above discussion it can be concluded that FOS is able to counteract several metabolic alterations linked to obesity including blood pressure, defecation profile, hunger and satiety, psychological depression, dietary intakes, lipemic parameters, and gut microbiota (LAB, bifidobacteria, bacteroides and clostridium) in obese grade-I adults.