CHAPTER 2: REVIEW OF LITERATURE

2.1 Introduction

Overweight and obesity is one of the major risk factors for many serious, chronic health conditions, and it is the most important public health issue throughout the world including India (43). World Health Organization (WHO) refers to the conditions of overweight and obesity as a global epidemic. In 2015, it accounted for about 4 million deaths worldwide (44). Overeating food which are energy dense, nutrient poor, accompanied by a sedentary lifestyle leads to this epidemic (45).

2.2 Prevalence of overweight and obesity

Systematic analysis of global data has shown that the prevalence of overweight and obesity is increasing significantly over the past three decades, with marked variations across countries in their levels and trends (46). Between 1980 and 2013, the global increase in obesity and overweight was 27.5% for adults and 47.1% for children, resulting in a total of 2.1 billion overweight and obese individuals (45). According to the recent WHO 2016 data, more than 1.9 billion adults are reported to be overweight. Of these overweight adults, over 650 million are obese (11% of men and 15% of women) which is approximately 13% of the total adult population and 39% of adults (39% of men and 40% of women) are overweight. It is also reported that between 1975 and 2016 prevalence of obesity has nearly tripled worldwide (47).

2.2.1 Obesity in India

In India also the burden of overweight and obesity is significant. In India, obesity is emerging as an important health problem particularly in urban areas, replacing the more traditional public health concern undernutrition (48). According to the 2015-16 Indian National Family Health Survey (NFHS-4), 31.3% of urban women, 15% of rural women and the overall total of 20.7% of women are obese and 26.6% of urban men, 14.3% of rural men and the overall total of 18.9% of men are obese. The prevalence of obesity is high in Kerala (32% in women and 29% in men) and Karnataka (23% women and 22% of men are obese) (49).

2.2.2 Gender differences
After puberty women have more fat than men at all ages and body fat increases with increasing age in both men and women (50). Weight gain is known to associate with health risks even if the body mass index (BMI) is not beyond the limit of 25 kg/m². In women a weight gain >5 kg, makes them susceptible to diabetes and cardiovascular diseases (CVD), while in men weight gain beyond 25 years is associated with increased health risks (51).

### 2.3 Definition and classification of overweight and obesity

Obesity is defined as an abnormal or excessive fat accumulation in the body that may impair health and increase the risk of morbidity due to several pathologies, including hypertension, dyslipidemia, type 2 diabetes mellitus, coronary heart disease, stroke, non-alcoholic fatty liver disease, osteoarthritis, sleep apnea, and cancers of endometrium, breast, prostate, and colon (1). Compared to nonsport persons, sportspersons may be overweight by arbitrary standards, even without having increased adiposity, because of increased muscle mass. So, overweight or obesity should not be defined by body weight alone (52). BMI, waist circumference (WC), waist-hip ratio (WHR), and percentage of fat are the most common parameters to assess overweight or obesity status (52).

**Table 2.1 International classification of adults based on BMI**

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Category</th>
<th>BMI (kg/m²)</th>
<th>Obesity Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Underweight</td>
<td>&lt;18.5</td>
<td>-</td>
</tr>
<tr>
<td>2.</td>
<td>Healthy weight</td>
<td>18.5-24.9</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>Overweight</td>
<td>25.0-29.9</td>
<td>Pre-obese</td>
</tr>
<tr>
<td>4.</td>
<td>Obesity</td>
<td>30.0-34.9</td>
<td>Grade-1 Obesity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>35.0-39.9</td>
<td>Grade-2 Obesity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥40</td>
<td>Grade-3 Obesity</td>
</tr>
</tbody>
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BMI is calculated by dividing a person’s weight in kilograms (kg) by the square of the height in metres (m). According to WHO standards, overweight is defined as BMI ≥25 kg/m² and if, BMI≥30 kg/m² is considered as obese (Table 2.1).

Even though BMI does not measure the body fat percentage directly, research has shown that it is closely correlated with the more direct measures of the body fat obtained from skin fold
thickness measurements, bioelectrical impedance, underwater weighing, and dual energy absorptiometry methods (54). In recent years, there is a growing debate on whether there are possible needs for developing different BMI cut-off points for different ethnic groups. This is because of the increasing evidence for associations between BMI and percentage of body fat. In addition, body fat distribution differs across populations. Therefore, the health risks increase below the cut-off point of 25 kg/m$^2$ that defines overweight in the current WHO classification (54).

BMI categories for Asian populations have been revised as per the suggestions given by the WHO in their expert consultation which concluded in 2004; since Asians have a higher percentage of body fat than Caucasian people of the same age, sex, and BMI. It is also found that the occurrence of type 2 diabetes mellitus and CVD in Asian populations is more in lower BMI than the WHO cut-off limit of 25 kg/m$^2$ (55). Data on Asian Indians residing in New Delhi showed that 66% of men and 88% women, classified as non-obese based on BMI (WHO cut-offs) had ≥1 cardiovascular risk factors (56). Hence in the present study participants are classified based on the revised BMI classification available for Asian population (Table 2.2) which defines overweight as BMI between 23.0 and 24.9 kg/m$^2$ and obesity as a BMI≥25 kg/m$^2$ (57).

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Category</th>
<th>BMI (kg/m$^2$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Normal</td>
<td>18.0-22.9</td>
</tr>
<tr>
<td>2.</td>
<td>Overweight</td>
<td>23.0-24.9</td>
</tr>
<tr>
<td>3.</td>
<td>Obesity</td>
<td>≥25.0</td>
</tr>
</tbody>
</table>

Table 2.2 Revised classification of adults based on BMI for Asian Indians

Source: Adapted from consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. J Assoc Physicians India, (2009).

One of the drawbacks of BMI is that, it is not able to differentiate fat mass from lean mass (55).

A second anthropometric measurement providing valuable information about overweight and obesity is waist circumference which is measured using a measuring tape midway between the lower rib margin and iliac crest at the end of gentle expiration (7). It is an indicator of abdominal fat, which is considered as a major contributor to health complications associated with overweight and obesity (7). Waist circumference of ≥94 cm and ≥80 cm in men and
women, respectively, are associated with increased risk of metabolic complications, and individuals with waist circumference >102 cm and >88 cm in men and women, respectively, are at higher risk of metabolic complications (7).

Fat mass is a third anthropometric measure used in relation to overweight and obesity. In young adults, body fat >25% in men and >35% in women is defined as obesity (4). Determination of fat mass can be done using techniques such as measurement of total body water, densitometry, or dual-energy X-ray absorptiometry, all of which require sophisticated equipment and are impractical in many clinical and research settings. A more convenient method for estimation of fat is bioelectrical impedance analysis, where the device determines the electrical impedance or opposition to the flow of current through body tissues, thereby providing a measure of total body water, and gives estimates of fat and fat-free mass (59).

2.4 Causes and drivers of overweight and obesity

Obesity or overweight results from increased energy intake, decreased energy expenditure or a combination of the two. Energy intake that exceeds energy expenditure over time is the main physiological cause of obesity and driver of weight gain (60). Underlying mechanisms for overweight and obesity comprise a complex interaction of individual, social, environmental and genetic factors connected by a diversity of pathways (61).

Few obese adults have identifiable specific diseases such as hypothyroidism and Cushing's syndrome. But in most of the obese or overweight, the cause is considered to be an interaction between the environmental and genetic factors (62). At the individual level, predisposing genes contribute significantly to the development of obesity and seem to interact with factors such as physical activity and diet (63). Although there are genetic and hormonal influences on body weight, lifestyle has a major role; that has a combination of many contributing factors such as physical inactivity, unhealthy diet and eating habits, lack of sleep, stress and endocrine factors. Many biological factors like endocrine disrupters and the use of medication are in play and interplay with environmental influences in causing overweight and obesity (64).

Societal changes over time affect individual behaviours and lead to more sedentary living, low levels of physical activity, motorized transport habits, and poor dieting, thus contributing to the rise of obesity (65). Factors in the family, such as unfavourable eating patterns, lack of
healthy food, lack of equipment and family support for physical activity, impact negatively on health-related behaviours and body weight of individuals (66).

2.5 Stress and obesity

Stress is one of the environmental factor that may contribute to the development of obesity. Stress is a challenge to the natural homeostasis of an organism which can be caused by external stressors such as employment or social strains or by intrinsic stressors such as sleep deprivation (67). Stress response is characterized by acute behavioural and physical adaptations, including increased cognition, analgesia, gluconeogenesis, lipolysis, and inhibition of reproduction (68). Stress is thought to influence human eating behaviour and it appears to alter overall food intake, which may be influenced by stressor severity. Chronic life stress seems to be associated with more preference for energy and nutrient-dense foods and thereby contributing to the development of overweight and obesity (69). Chronic stress, often arising from poor interpersonal relationships, job or unemployment stress, poor self-esteem, and low socioeconomic status leads to the activation of sympathetic nervous system and hypothalamic-pituitary adrenal axis (HPA axis) contributing to an anabolic state that promotes fat storage within visceral depots causing obesity (70).

Studies show that stress can enhance weight gain and deposition of fat by bringing changes in feeding behaviour (70). Obese subjects with uncontrolled stress frequently have increased cortisol level and this, in turn, causes the release of glucose and fat into blood, which triggers the release of insulin. This promotes the conversion of excess sugars into fat leading to its accumulation around the visceral organs and thereby enhances the risk of non-communicable diseases. The mortality risk of affected subjects increases 2-3 folds and shortens their life expectancy by several years (71).

2.6 Consequences of overweight and obesity

Compared to those with normal weight, obese adults are linked with multiple negative health consequences with an increased risk of development of type 2 diabetes mellitus, CVD, hypertension, obstructive sleep apnoea, gall bladder disease, certain forms of cancer and it also affects psychological wellbeing of the individual. Severe obesity reduces the life expectancy of obese individuals by seven to fourteen years (72). There is a substantial economic burden of obesity in terms of health service delivery and loss of productivity due to
absenteeism and reduced ability to perform the tasks. Prevention of overweight and obesity is more cost-effective than treatment, in terms of health care and personal costs (72).

2.6.1 Dyslipidaemia in overweight and obesity

Dyslipidaemia or altered lipid profile, associated with overweight and obesity is a major risk factor for the development of CHD and CVD (73). Typical dyslipidaemia of obesity consists of increased triglycerides (TAG) and free fatty acids (FFA), decreased HDL cholesterol (HDL-C) with HDL dysfunction and normal or slightly increased LDL cholesterol (LDL-C) with increased small dense LDL. An increase in the level of plasma apolipoprotein (apo) B is attributed to overproduction of liver of apo-B containing lipoproteins (74). Central adiposity is associated with increased risk for abnormal serum lipids. It is noted in several studies that overweight, obesity especially abdominal obesity is associated with hyperlipidaemia, reduced HDL, raised LDL and increased TAG (75, 9). High levels of LDL-C lead to atherosclerosis increasing the risk of heart attack and ischemic stroke (76).

The important finding of dyslipidaemia in obesity is elevated fasting and postprandial TAG in combination with the increase of small dense LDL and low HDL-C (77). The major cause for lipid abnormalities and formation of small dense LDL found in obesity is hypertriglyceridemia (Fig. 2.1), since it leads to delayed clearance of the TAG-rich lipoproteins (78, 79). Lipolysis of TAG-rich lipoproteins is impaired in obesity by reduced mRNA expression levels of lipoprotein lipase (LPL) in adipose tissue, reductions in LPL activity in skeletal muscle and competition for lipolysis between VLDL and chylomicrons (81). Increased postprandial lipemia leads to elevated levels of FFA, resulting in detachment of LPL from its endothelial surface (82, 83). LPL may remain attached to VLDL and IDL contributing to further TAG depletion. The exchange of TAG from these remnants for cholesterol-esters from HDL by cholesterol ester transfer protein (CETP) with the concerted action of hepatic lipase, ultimately leads to the formation of small dense LDL (78). In the presence of hyper-triglyceridemia, the cholesterol-ester content of LDL decreases, whereas the TAG content of LDL increases by the activity of CETP. However, the increased TAG content within the LDL is hydrolyzed by hepatic lipase, which leads to the formation of small, dense LDL particles. The development of small dense LDL in obesity is mainly due to increased TAG concentrations and does not depend on total body fat mass (84). Small dense LDL is relatively slowly metabolized with a five day residence time, which enhances its atherogenicity (85).
Chylomicron remnants and LDL may migrate into the sub-endothelium and become trapped in the sub-endothelial space where they can be taken up by monocytes/macrophages (86-88). Small dense LDL has a higher affinity for arterial proteoglycans resulting in enhanced sub-endothelial lipoprotein retention (89). However, sub-endothelial remnants of chylomicrons and VLDL do not need to be modified for uptake by scavenger receptors of macrophages in contrast to the native LDL (77). It is described that small dense LDL is more susceptible for oxidation, in part due to less free cholesterol and anti-oxidative content (90). It should be noted that the lipoprotein size is a limiting factor for migration through the endothelium and that LDL particles migrate more easily than chylomicron remnants, but the number of migrated particles do not necessarily translate into more cholesterol deposition, since chylomicron remnants contain approximately 40 times more cholesterol per particle than LDL (88). Alternatively, LPL-enriched remnants of chylomicrons and VLDL may be transported to the tissues where interaction with proteoglycans and lipoprotein receptors lead to particle removal. This process takes place in the liver and acts as an anti-atherogenic mechanism. But it may also happen in other tissues where cholesterol cannot be removed efficiently, leading to cholesterol accumulation and the initiation of an atherosclerotic plaque (91, 92)
Studies show that plasma FFA are elevated in obese people as a consequence of an increased fatty acid release from adipose tissue and a decrease in plasma FFA clearance (93). The increase in FFA and obesity-induced inflammation, play a crucial role in the development of insulin resistance. Increased level of long chain saturated fatty acids, palmitic acid, as well as lauric acid and stearic acid can stimulate the synthesis of pro-inflammatory cytokines, and thereby inducing inflammation in obesity (93).

Changes in the lipid profile, especially hypercholesterolemia, may contribute to changes in vascular stiffness, which is an important determinant of CVD risk (94). Recent studies have investigated the association between arterial stiffness and non-HDL-C, as it measures the cholesterol content of all atherogenic lipoproteins including LDL-C (95).

2.6.2 Oxidative stress in overweight and obesity

Oxidative stress represents an imbalance between the production and removal of reactive oxygen species (ROS) and the ability of a biological system to readily detoxify the reactive intermediates or to repair the resulting damage. Oxidative stress is a relatively recent concept that is widely implicated by medical fraternity in the last two decades. It is involved in the pathophysiology of many highly prevalent diseases such as diabetes mellitus, hypertension, and CVD. Disturbances in the normal redox state of tissues can cause toxic effects through the production of peroxides and ROS, that damage all components of the cell. Some ROS can even act as messengers in redox signalling (96, 97).

Even though the cause-effect relationship of oxidative stress in obesity is not known, various epidemiological studies have illustrated that obesity is coupled with altered redox state and increased metabolic risk (98, 99). Complications of overweight and obesity associated conditions are attributed to oxidative stress which may by itself be one of the causes for the development of obesity. Studies show that one of the reasons for oxidative stress associated with obesity is accumulated fat, which in turn increases the activity of NADPH oxidases and stress in the endoplasmic reticulum of the adipocyte which further enhances more ROS production (100, 101). Prolonged over nutrition with carbohydrate and fat rich diet, persistent inflammation, tissue dysfunction, and low antioxidants, contribute to ROS production by nitric oxide, oxidative phosphorylation and other mechanisms (102-104). Oxidants produced in obesity activate specific redox-sensitive transcription factors, including
nuclear factor-kB (NF-kB) and activator protein-1 (AP-1), which drive the expression of pro-inflammatory cytokines. These cytokines, in turn, enhance production of ROS, thus contributing to the onset and maintenance of oxidative stress (Fig. 2.2) (105). In obese subjects, excess accumulation of fat in adipose tissue leads to dysfunction of adipose tissue which contributes to the onset of oxidative stress by increasing the expression of adipokines, viz., monocyte chemoattractant protein (MCP)-1 and macrophage inflammatory protein (MIP) -1α, -1β, -2α that trigger macrophage infiltration and subsequent overproduction of ROS, and inflammatory cytokines (106, 107).

Obesity-associated excessive increase in intracellular TAG triggers lipotoxicity by inhibiting the adenine nucleotide translocator, which leads to ATP accumulation in the mitochondria. Accumulation of ATP within the mitochondria decreases the availability of ADP and thereby reduces the speed of oxidative phosphorylation and mitochondrial uncoupling promotes electron leakage and free radical generation (108). Persistent inflammation and dysregulation of adipocyte-derived factors associated with obesity causes endothelial dysfunction which activates endothelial NADPH oxidase and increases the levels of free radical levels (109). Hyperleptinemia found in obesity is associated with increased mitochondrial and peroxisomal fatty acid oxidation, with subsequent stimulation of ROS synthesis via the electron transport chain of mitochondria (110,111). Increased leptin level also stimulates the activation of monocytes/macrophages, with the production of proinflammatory cytokines (IL-6 and TNF-α) that intensify oxidative stress (112).
Oxidative stress could play a causative role in the development of obesity by stimulating white adipose tissue deposition altering food intake (113). Systemic oxidative stress and inflammation are also key factors in the pathogenesis of obesity-related diseases, such as, type 2 diabetes mellitus, atherosclerosis, and cancer (114, 115). It is recently proposed that altered oxidative status may be a primary factor in the etiology of obesity-induced insulin resistance and type 2 diabetes mellitus (116). Chronic oxidative stress and adipokine secretion lead to the death of β-cells, as they have low scavenging ability. Adipocyte oxidative stress leads to the production of glutathionylated products of lipid peroxidation that results in insulin resistance, inflammation, protein oxidation and misfolding, thereby resulting in proteasomal dysfunction, which contributes to the onset of insulin-resistant and obese phenotype (117-119).

Circulating FFA, insulin resistance, oxidative stress, mitochondrial and endothelial dysfunction are also key pathogenic factors of obesity-associated cardiovascular pathologies (120). Studies have shown that ROS overproduction triggers DNA damage, thus leading to genomic instability associated with the activation of oncogenes and/or inactivation of tumor suppressor genes (121-123). A recent systematic meta-analysis documented that oxidative stress, together with visceral adipose tissue, is one of the pathogenic mechanisms accounting
for polycystic ovary syndrome. Likewise, controlling oxidative stress in obesity is critical for the reduction of future health problems and morbidity associated with obesity.

2.6.3 Impact of overweight and obesity on the quality of life

Obesity increases the rate of morbidity as well as mortality. It is one of the most prominent preventable causes of death worldwide (124). Increased mortality and complications arising in obesity may be the direct result of obesity or can also be an indirect consequence. The consequences of obesity on health may be attributed to increased fat mass, as seen in osteoarthritis, obstructive sleep apnoea or due to an increased number of fat cells as seen in diabetes, CVD, cancer and non-alcoholic fatty liver disease (125). Studies which evaluated the relationship between obesity and quality of life, concluded that they are inversely related (126). Quality of life is found to be very poor in obesity surgery seekers (127). The comorbidities associated with overweight and obesity lead to productivity losses and enhance health care expenditures (128).

2.7 Management of overweight and obesity

Overweight and obesity if not treated leads to health problems like type 2 diabetes mellitus, atherosclerosis, sleep apnea, CVD, osteoarthritis and certain types of cancer. To prevent these health risks, it requires ongoing treatment and lifestyle interventions. The requirement to improve the ailing health, functioning, and quality of life of treatment-seeking obese individuals has led to the development of three treatment modalities, viz., lifestyle interventions, pharmacotherapy and bariatric surgery (129,130). If overweight and obese adults are not able to maintain or lose excess of body weight with lifestyle intervention, pharmacotherapy may be needed to prevent health risks. The urgency and selection of treatment modalities in overweight and obesity should be based on BMI and a risk assessment (9). Overweight being a mild condition, compared to obesity, can be easily monitored through lifestyle interventions.

2.7.1 Pharmacotherapy in the management of obesity
The main objective of obesity treatment is to improve or prevent the complication of metabolic diseases, not just weight loss. So, it is not enviable to prescribe medicine to an overweight or obese individual who wants to lose only body weight. The first step of obesity treatment should be using non-pharmacologic lifestyle interventions. If the patient does not achieve adequate weight loss by lifestyle intervention for 3-6 months, pharmacotherapy can be considered (131). Orlistat, lorcaserin, and the combination of phentermine and topiramate are approved drugs for long-term use and benzphetamine, diethylpropion, phendimetrazine, and phentermine are approved drugs for short-term use in the treatment of obesity. Lorcaserin and combination of phentermine and topiramate have been recently approved by US Drug and food administration (131).

Orlistat is an inhibitor of intestinal lipase. It promotes weight loss by fat malabsorption but it causes a significant decrease in fat-soluble vitamins. Noradrenergic agents reduce obesity by decreasing hunger but cause side effects like insomnia, increases heart rate and blood pressure (132). Lorcaserin, an inhibitor of serotonin, decreases food intake and thereby reduces body weight, but causes side effects like headache, nausea, dizziness, fatigue, dry mouth, and constipation (131).

2.7.2 Bariatric surgery in the management of obesity

Weight-loss surgeries done for severely obese patients are either restrictive (limiting the amount of food the stomach can hold and slowing gastric emptying), such as laparoscopic adjustable silicone gastric banding, or restrictive-malabsorptive, such as Roux-en-Y gastric bypass (9). Bariatric surgery should be considered for individuals with severe obesity (BMI≥40 kg/m²) or moderate obesity (BMI≥35 kg/m²) associated with a severe medical condition, with repeated failures of other therapeutic approaches (9).

2.7.3 Lifestyle interventions in the management of overweight and obesity

The lack of physical activity, insufficient physical activity and or the lack of physical activity or low levels of physical activity with overeating or unhealthy eating are the most common causes of overweight and obesity in adults. In order to overcome this, the most important strategy is lifestyle modification. The major lifestyle interventions for overweight and obesity
include behavioural therapy, dietary modification, and an increase in the intensity of physical exercises.

2.7.3.1 Behavioural therapy (BT)

Optimal treatment of an overweight or obese patient is initiated with a combination of diet and exercise. Adherence to both these treatment plans requires changes in behaviour which can only be brought about by BT. BT can help individuals predisposed to obesity to develop a set of skills that can help them to achieve a healthier weight. The key components in the BT are self-monitoring of the intake of food and calories by maintaining food diaries and activity logs (132). It also requires altering the environment that activates overeating, modifying it so as to avoid overeating and slowing the speed of consumption of food (133,134). Moreover, it can be achieved by setting realistic goals for overweight or obese subjects intending to lose body weight in terms of weight loss per week/month (135). Similarly, reinforcement of successful outcomes or rewarding good behaviours in the form of small gifts or even financial benefits also helps in this direction (136). In the form of nutritional education, a structured meal plan devised for an individual patient in consultation with a dietician results in a greater weight loss when compared to the absence of a structured meal plan (137,138). Increasing the intensity of physical exercises is another important component of a successful BT package. Research findings also show that self-monitoring and increasing physical activity are consistently associated with better outcomes in the achievement of ideal weight (139). Behavioural modification is effective in the long term where the overweight or obese patient receives support from spouses and other members of the family. A meta-analysis has concluded that including family members led to an additional 3 kg weight loss compared to programs that did not include family members (140).

2.7.3.2 Dietary intervention in the management of overweight and obesity

Dietary intervention is the cornerstone of weight loss and weight maintenance therapy in overweight and obesity (141). A negative caloric balance is essential to promote weight loss. Weight loss is generally achieved by a low-calorie diet (LCD) that provides approximately 800–1500 kcal/day. A caloric deficit of 500–1000 kcal/day will promote the desired weight loss of 0.5-1 kg/wk (142). Very-low-calorie diets (VLCDs) generally provide 800 kcal/day or even less. They promote a higher short term weight loss of 1.4-2.5 kg/wk, but at 1 year of treatment with a VLCD, weight loss is not significantly lower than that of an LCD (143,144).
VLCDs necessitate intensive medical supervision, monitoring of electrolytes, and supplementation of minerals and vitamins. Therefore, their role in overweight or obesity management is only limited to cases, in whom there is a need for rapid weight loss, and they should not be widely implemented (145).

2.7.3.3 Physical activity in the management of overweight and obesity

Physical activity in the form of structured exercise contributes to the creation of a deficit of energy by increasing total energy utilization, and this can promote loss of body weight. Studies have evaluated the effect of short term physical exercise intervention alone and also in combination with reduced intake of energy on body weight. Comparison of such studies have demonstrated that reduction in the intake of energy in the form of LCD have a greater impact on weight loss than changes in physical activity, with the combination of LCD and exercise having the greatest impact on loss of body weight (146). A study by Ross et al., (2000), shows that exercise can be an effective intervention as a LCD for precipitating the initial loss of body weight in overweight and obese adults (147). But to achieve significant changes in body weight physical exercises should be continued not less than twelve months (148) even though the weight loss is comparatively more in the first 6 months (149).

Adequate levels of physical activity are required to realize the benefits of weight control. An equivalent of at least 150 min/wk of moderate-intensity physical activity is necessary to realize improvements in health-related outcomes (150,151). For long-term weight loss outcomes, higher levels of physical activity are required. A study by Jakicic et al., (2003), has reported that long-term weight loss improved in overweight and obese women with the addition of 200-300 min/wk of physical activity (149).

2.7.4 Yoga in the management of overweight and obesity

2.7.4.1 Introduction to yoga

Yoga is an ancient Indian way of life, which includes changes in mental attitude, diet, and the practice of specific techniques such as yogaasanas (postures), breathing practices (pranayamas), and meditation to attain the highest level of consciousness (152). Yoga is the oldest known ancient Indian system of science, philosophy, culture, tradition and holistic means of self-development and maintaining better through physical, mental and spiritual control (152). The Sanskrit word yoga means the union of body and mind through breath
control methods, asanas, and meditation. Regular yogic practices endow perfect physical and mental health to its practitioner. According to Patanjali’s Yoga Sutra, to lead a meaningful and purposeful life each individual should follow eight limbs of yoga, which include yama, niyama, asana, pranayama, prathyahara, dharana, dhyana, and samadhi. One who does the practice of these eight limbs systematically achieves self-realization, which is the supreme goal of performing yoga (153).

In Indian philosophy, there are two types of physical illness, and each requires a different approach. The first is the illness with a strong physical element, such as contagious diseases and accidental injuries. These are most effectively dealt with by conventional medicine, though yoga can play a substantial supporting role by preventing the occurrence of such ailments by improving general health (154). A second type of illness arises through disturbances in the mind and includes all psychosomatic and degenerative ailments where psychological factors play a much greater role, and conventional treatment alone is not usually an effective cure. Since these ailments are caused by strong likes and dislikes which are amplified, established, and distort personality, and they obstruct the flow of positive energy. In such conditions, yoga plays an important role by correcting the mental imbalances that underlie them (155). In the last few decades, studies are conducted to elucidate the therapeutic effect of yoga on psychosomatic diseases and non-communicable diseases like obesity, diabetes mellitus, and CVD. These studies reported the beneficial effect of yoga in these and other such conditions, signifying that yoga can be used as an alternate form of lifestyle modification, i.e., a complement to conventional medicine for treating conditions like overweight and obesity (153).

2.7.4.2 Effect of yoga on anthropometric measurements, blood pressure and lipid profile in overweight and obesity

An observational study involving long-term yoga practitioners showed that, a consistent, long-term Hatha yoga practice in women over 45 years was linearly associated with decrease in BMI even after correcting for non-yogic exercise hours and processed food consumption (156). In a study involving 16 postmenopausal women with more than 36% body fat, the practice of yoga improved adiponectin level, serum lipids, and metabolic syndrome risk factors (157). A study by Kristal, (2005), shows that regular practice of yoga for four or more years is highly effective in reducing weight gain, particularly among subjects who were overweight (158). Study by Telles et al., (2014), where obese participants have undergone a
yoga program, which included 5 hours of yoga/day and had a low fat, high fibre, vegetarian diet for six days, has shown significant decrease in: anthropometric variables, viz., BMI, waist and hip circumference, lipid profile parameters such as total cholesterol and HDL-C, fasting serum leptin level and an increase in postural stability and hand grip strength (159).

In a randomised control trial (RCT) involving 23 adults, the yoga group were subjected to a 3-month yoga intervention of twice-weekly yoga sessions and the education group received health information every 2 weeks. The study showed significant improvements in body weight, blood pressure and insulin levels in the yoga group than the education group (160). A study by Yang et al., (2011), which compared the effect of transcendental meditation (TM) and health education in individuals with coronary artery disease and the metabolic syndrome. The study demonstrated, significant difference in blood pressure and positive beneficial changes in plasma glucose and insulin levels in the interventional group as compared to the education group (161).

Another randomized, placebo-controlled clinical trial conducted by recruiting subjects (n=103), with stable coronary artery disease and metabolic syndrome showed significant improvement in blood pressure and insulin resistance in subjects who have undergone 16 weeks of TM compared to the health education group (162). Practice of 30 min of suryanamaskar has resulted in a loss of 230 kcal of energy in subjects with obesity. The study concluded that regular practice of suryanamaskar can maintain or improve cardiorespiratory fitness, and promote weight management (163).

Yogic practice by middle-aged obese adults for the period of 12 weeks, six days per week of one hour in the evening has shown significant (p<0.01) reduction in plasma TAG. The yogic practices given to the subjects include loosening exercises, suryanamaskar, padahastasana, parvaatasana, pachimuttasana, ardhamatsyendrasana, bhujanagasana, shalabasana, sarvangasana, halasana, dhanurasana, nadishodana, shavasana and meditation (164). A study on body composition, cardiovascular complication and aerobic power of yoga practitioner revealed that there was a significant reduction in waist-hip ratio compared to the non-yoga practitioners (165). A RCT by Tundawala et al., (2012), conducted with obese, hypertensive and dyslipidaemia subjects (n=150), where yoga group practiced 3 months of yoga, and for the subjects of the control group instructions were given about dietary and lifestyle modification without yoga. The study showed a significant reduction in (p<0.05)
BMI and WHR, blood pressure—both systolic and diastolic, and a significant improvement in lipid parameters in the *yoga* group (166).

Behaviour modification including *yoga* in obese subjects with type 2 diabetes mellitus observed a significant reduction in BMI from 34.2 ± 0.8 kg/m² to 30.6 ± 1.1 kg/m² (167). A study by Schmidt *et al.*, (1997), with 3 months kriya yoga training shows significant reduction in BMI of obese adults (168). Manchnada *et al.*, (2000), showed a weight reduction of 6.8 ± 8.2% (p=0.0019) after yogic lifestyle intervention in obese coronary atherosclerotic patients (169). Six weeks of *yoga* asana training has significantly changed health-related physical fitness such as, cardio-respiratory endurance, abdominal strength, and endurance. In addition, there was an increase in the flexibility and performance of the obese subjects (170). A study which compared the effect of *yoga* based lifestyle program and physical exercise in patients with chronic low back pain has found a significant reduction in pain-related disability and improvement in spinal flexibility of the *yoga* group with a reduction in pain, depression, and anxiety as well (171). Hatha *yoga* practice for 8 weeks which consisted of 10 min of pranayamas (breath-control exercises), 15 min of dynamic warm-up exercises, 50 min of asanas (*yoga* postures), and 10 min of supine relaxation in shavasana (corpse pose) has shown significant changes in body composition in healthy subjects (172).

Kapalbhati pranayama practice in male students (n=30) has resulted in significant changes in body fat (%), lean body mass (kg), body water content (%) and basal metabolic rate (173). Another study conducted at Haridwar by recruiting male junior footballers (n=20) to find the effect of *pranayama* (voluntary regulated breathing) and *yogasana* (*yoga* postures) on lipid profile has shown significant changes (p<0.001) in serum triglycerides, serum cholesterol, LDL-C, and VLDL-C. This demonstrated that pranayama and *yogasana* can be used as supportive therapy in patients with lipid disorders (174).

### 2.7.4.3 Effect of *yoga* on stress in overweight and obesity

In subjects with abdominal obesity due to chronic stress, overstimulation of HPA axis alters the diurnal secretion of cortisol. Abnormal regulation of the HPA axis and perceived stress—dependent cortisol levels are strongly related to abdominal obesity with metabolic abnormalities (175,176). Eight weeks *yoga*-based stress reduction and relaxation program has shown a significant decrease in medical and psychological symptoms, and improvement in
self esteem in overweight and obese individuals (177). Previous studies have reported that, after practicing yoga, stress reduction was seen in individuals with mental stress (178). Yoga-based lifestyle intervention has shown a decline in free radical levels in blood and lower seminal oxidative stress (179). A review article by Subramanya et al., (2009), found that practice of cyclic meditation twice a day reduces occupational stress levels and baseline autonomic arousal by bringing the shift in the sympathovagal balance towards vagal dominance. It is also suggested that the practice of cyclic meditation during day time improves the quality of sleep by increasing the percentage of slow wave sleep in the subsequent night (180).

2.8 Lacunae in the review of literature

In conclusion, it is evident from the review of literature that yoga, a non-conventional method of lifestyle intervention, has shown its efficacy in reducing body weight, BMI, lipid levels and oxidative stress in healthy subjects and subjects with various pathologies. But its efficacy in the management and prevention of overweight and obesity has not been studied extensively. Therefore, the present study will be an attempt to fill that gap in the literature.