SUMMARY
AND
CONCLUSION
Obesity is one of the greatest health threats of this century. It is associated with such diseases like type 2 diabetes, coronary heart diseases, hypertension and some cancers. Overweight and obesity are the fifth leading causes for global deaths.

Several factors, including high calorie diets, lack of exercise, sedentary lifestyles and the genetic factors contribute to the etiology of obesity. Whatever may be the cause, the basic mechanism involved is imbalance between energy intake and energy expenditure.

In the present study the effect of the cafeteria and high fat diets in causing obesity has been studied. Under the influence of the above said diets obesity occurs in the following four ways.

- As we take food, at some stage we feel palatability and satiation in case of normal food but this satiation does not occur under the influence of high fat and cafeteria diet while taking high caloric food. So, the subject goes on eating which finally leads to obesity.
- Cafeteria and high-fat diets have the tendency to get readily and effectively stored in the body. This storage finally causes obesity.
- These diets accelerate the production of pancreatic lipase which in turn accelerates the digestion, absorption and then accumulation of fat inside the body.
- With the secretion of the hormone Ghrelin, we feel hunger. This feeling subsides and the secretion stops as we consume normal food. But in case of cafeteria and high fat diets this does not occur. So the subject over eats and becomes obese.

Genetics may play a role in the regulation of body weight, body size, body composition and the metabolic response to feeding but it alone cannot explain.

Dietary fat intake often has been claimed as responsible for the rapid increase in adiposity. Human studies have shown that high-fat diets (~30% of energy from fat) can easily induce obesity.
Epidemiological studies conducted in countries such as China, Canada and the United States have shown that, when the average amount of fat in the diet increases, the incidence of obesity also increases. This has led to a worldwide effort to decrease the amount of fat in the human diet.

Diet-induced obesity allows us to generate obese models in a more realistic manner than monogenic or pharmacological manipulations and has therefore been extensively used in the study of obesity-associated disorders. In this respect, high fat diets are regarded as the "gold standard" although they present two major drawbacks: 1) they only poorly mimic Western diets and 2) they are not standardized since, as we have seen in the literature review of this thesis, fat quantity and quality play significant roles in the development of metabolic disorders. Therefore, we decided in this thesis to compare a widely used high fat diet to a cafeteria diet. Cafeteria diets are supposed to elicit an obese phenotype through hyperphagia which is driven by the flavour variety.

We aimed at determining the most appropriate form in which the diets should be offered to the rats. We showed that rats fed powder diets developed the same body weights, regardless of the diet composition, whereas when we fed them the same diets in pellet form, rats on the control diet remained lean and rats on the high fat and cafeteria diet clearly presented an obese phenotype. This surprising finding allowed us to separate pathophysiological changes due to body weight from those due to specific dietary effects. Thus, we could conclude that a cafeteria diet seemed more deleterious for the liver than the commonly used high fat diet and reported some diet-specific changes in the adipose tissue.

Thus, it seems that a cafeteria diet elicits disturbances as or even more severe than a high fat diet, although it is characterised by a lesser energy density and fat content. Hence, cafeteria diet constitutes a good diet to promote diet-induced obesity. To conclude, this work brings new knowledge in the effects of dietary treatment on obesity-associated disturbances, a crucial step in the elaboration of dietary recommendations for obese patients.
A treatment options today for obesity include diet therapy, increased physical activity, behavioral therapy, surgery and pharmacotherapy but, these are not successful. There is a lot of unwillingness to follow diet therapy, do physical activity and listen to the best counseling of a behavioral therapist because the most common tendency today is to eat more and more and do lesser and lesser physical activity. So people prefer pharmacotherapy to other alternatives. Two different types of weight reducing drugs are currently available in the market. One of them is orlistat (Xenical), which reduces intestinal fat absorption through inhibition of pancreatic lipase. The other is sibutramine (Reductil), which is an anorectic, or appetite suppressant. But both drugs have side-effects, including increased blood pressure, dry mouth, constipation, headache, and insomnia. Coming to surgery, bariatric surgery is considered the most successful one in case of highly obese patients only but not suitable to all cases. Further the possibility of several complications that follow after this surgery does not allow its wide range use. Therefore, there is a huge challenge for the scientific community to search for more effective and better tolerable treatments against obesity.

So I strongly recommend herbal therapy as an alternative because it is cost effective and also free from side effects. A large number of herbs have been investigated to test their potential in inducing body weight reduction and preventing diet-induced obesity.

In this search one important herb remains unnoticed. It is *G. sylvester*. There is a claim in Ayurveda (Indian system of medicine) that this plant had been in use for obesity since a long time. It is in this plant I find a solution to those unable to come out of their habit of over eating and remaining forever obese.

Flavonoids, triterpenoids, polyphenols, steroids and saponins are the main active principles identified by modern researchers. It was established that some saponins of particular structure posses anti obese property. So I felt worthwhile to check whether saponins of this herb also posses the same property.
I have tried the saponins from G. sylvestre aqueous leaf extract on cafeteria and high-fat diets fed wistar rats to understand how far this extract can prevent obesity.

It was found that at a dose of 100 mg/kg body weight given for eight weeks along with CA diet and HF diet could substantially prevent obesity. This is evident from the following changes.

**Significant suppression in the increase of**

- Food Consumption
- Body Weight Gain
- Visceral Organs Weight
- Serum Total Cholesterol (TC)
- Triglycerides (TG)
- Very Low Density Lipoproteins (VLDL)
- Low Density Lipoproteins (LDL)
- Atherogenic Index (AI)
- Blood glucose
- Lipid peroxidation

**Significant elevation in the levels of**

- Serum high density lipoproteins (HDL)
- Fecal TC and TG
- SOD, CAT and GPx activities
- Content of Glutathione
Summary and Conclusion

Cafeteria and high-fat diets create an altered metabolic environment in which tendency for obesity, susceptibility to liver and heart diseases via oxidative stress.

SGE might exert its antiobesity action, decreasing intestinal lipid absorption and increasing fecal lipid excretion, through the inhibition of pancreatic lipase activity. Such obesity and its associated problems could be suppressed by the saponins from \textit{G. sylvestre} aqueous leaf extract.

Therefore those Ayurvedic drugs based on this plant can be tried on the generation of the people having the tendency to eat more, do less activity but don’t want to gain weight.

Ayurvedic pharmacopoeia further states that this plant and its derivatives have the potential to reduce accumulated fat even in already obese people. Research in this direction is also worth perusing.