CHAPTER-IV

DISCUSSION
During the last two decades a great deal of attention has been paid to hyperlipidemia and hypercholesterolemia because of the steadily mounting evidence of their association with atherosclerotic disease and its complications. It is well documented that elevations of either low density lipoproteins (LDL) or very low density lipoproteins (VLDL) of serum are indeed associated with a high incidence of atherosclerotic disease; particularly coronary disease (Ewer and Oliver, 1971; Illefon et al., 1971; Fallon and Ross, 1968). The compounds capable of correction of hyperlipidemia and hypercholesterolemia are considered to be of a paramount importance and help in the chemotherapy of atherosclerosis. It has become standard practice to employ dietary modifications, hypolipidemic drug therapy or partial ileal by-pass surgery to achieve plasma lipid reduction in hyperlipidemic patients. In this connection most current hypocholesterolemic drugs have been tested, but the side effects restrict their use. Obviously the search for an ideal hypolipidemic agent with minimum side effects is still underway.

Bengal gram (Cicer arietinum) also known a chickpea, Garbanzo (Spanish) or chana (Hindi) is one of the important pulse crops of Indian subcontinent and accounts for a turnover of nearly Rs. 50 crores annually in the gross national product (Times of India, 1973). It is extensively cultivated in northern India and
is a relatively inexpensive cereal. It belongs to family
leguminosae. It has been observed that people of low socio-
economic group consuming large amount of Bengal gram in diet
show a lesser incidence of coronary heart disease (Arishma
Murti, 1974). The effect of this legume in lowering blood
cholesterol in experimentally induced hypercholesterolemia
has also been established in rats, rabbits and human beings
(Mathur et al., 1963; 1964a; 1964b; 1965; 1966; 1972; Kadhavan
et al., 1973), indicating the lipid lowering action of Bengal
gram is not species-specific. Bengal gram affects appreciably
the serum cholesterol levels and the extent of fecal excretion of
bile salt and co, Ca and cholesterol contents of liver, suggesting
thereby that the synthesis of cholesterol is somehow depressed
in Bengal gram fed diet. Mathur et al. have also shown that the
cholesterol lowering effect is located in the whole defatted
flour (1964a) or the lipids extracted from whole flour (1964b).
A long term study lasting 67 weeks has been carried out by the
same investigators in 30 human male volunteers of 15-50 years
of age (Mathur et al., 1964a). In this study, Bengal gram did
indeed lower the induced hypercholesterolemia in 16 out of 20
subjects along with a statistical significant increase in the 24
hours fecal excretion of bile salts.
The beneficiary effect of bengal gram has not, however, been confirmed by Mitta Pand and Kapoor (1973) in experimental hypercholesterolemia of rabbits. These workers used a diet made up of wheat flour, 13; sucrose, 60; casein, 15; vitamins and mineral mixture, 5; and hydrogenated vegetable oil, 7%. When bengal gram was added, it replaced an equivalent amount of sucrose. To another group of animals, they gave a stock diet, which did not contain any sucrose at all but contained cholesterol or cholesterol + bengal gram. The results of this study lasting 120 days indicate that hypercholesterolemia and atherosoma formation, judged by histopathological scoring, were due to sucrose in the diet, the extent of hypercholesterolemia produced by cholesterol feeding alone was less pronounced and bengal gram did not influence the course of this effect.

Scrutiny of the data obtained by Kather et al. (1963) in rats receiving control diet (sucrose + cholesterol + hydrogenated oil) and in those receiving bengal gram at 40% level at the expense of sucrose has indicated the possibility of sucrose being the atherosclerosis inducing agent. However, when the control group (hypercholesterolemic) and the bengal gram + lipid fed group received the same amount of sucrose in the diet, a significant lowering of serum cholesterol as well as increased output of fecal bile salts were noticed in the latter group.
In an independent study, Devi and Kuup (1970) have shown that in rats fed for 3 months a pellet diet (24% protein and 4% ether extractives—sucrose not mentioned) supplemented with 15% hydrogenated vegetable oil and 25% cholesterol, powdered germinated bengal gram does not lower either blood, liver or aortic cholesterol or blood glucose. In view of the extensive data adduced by Mathur et al. notwithstanding the above two contradictory reports, the hypercholesterolemic effect claimed for bengal gram can not be discarded without more vigorously conducted experiments. It would be necessary to explore the nature of the substance (?) that can bring about the lowering of cholesterol or blood glucose.

It is likely that the flour from whole seeds or the flour from seed-coat removed pulse can give different results although the indigestible residue is not very high in gram. Subba Rao and Desai (1964) have shown that in rats receiving different types of diet, the maximum fecal bulk was noticed generally in pulse diets, particularly the bengal gram diet. Besides, Subrahmanyan et al. (1958) have called attention to the fact that diets containing large amount of bengal gram may favour the growth and sustenance of coliform bacteria in the rat gut. The relevance of these reports in assessing the
therapeutic value of bengal gram in hypercholesterolemia remains to be elucidated.

Ever since Siddiqui and his colleagues demonstrated the presence of isoflavones in the bengal gram (Siddiqui, 1945; Krishna Murti et al., 1948; Shandari et al., 1950; Bose, 1956; Bose, 1958), this legume has been extensively used for the study of biosynthesis of flavonoids. A variety of flavonoids have been isolated from bengal gram (Krishna Murti, 1974). In view of the beneficiary effect of bengal gram in lowering blood lipids, as discussed above, Siddiqui and Siddiqui (1976) focussed their attention on biochanin A and formononetin constituents of bengal gram. They have succeeded in demonstrating hypolipidemic activity of these isoflavones in Triton X-1399 induced hyperlipidemia in male albino rats. Further exploration revealed that these isoflavones may prevent the hyperlipidemia induced by massive doses of vitamin E2 and cholesterol in olive oil, cholesterolemic diet, hyperlipidemia inducing diet. Sharma (1979) has also observed the hypolipidemic activity of these isoflavones isolated from bengal gram in hypercholesterolemia-inducing diet fed rats.

It may be noted that biochanin A and formononetin are not apparently present in dormant seeds and all these isoflavones detected in seedlings of gram are presumably synthesised de novo during germination. It is quite likely that Nitya Nand and
Kapoor (1973) failed to observe the beneficial effect of bengal gram because they fed ungerminated bengal gram. Although Devi and Surup (1970) used powdered germinated bengal gram yet they could not observe the desired hypolipidemic effect, presumably the amount of the germinated bengal gram powder used was not sufficient to have the effective isoflavone (S) levels. In view of the contradictory reports it was decided to reinvestigate the hypolipidemic property of bengal gram. The lermen was germinated for 72 hr, dried at 60°C and powdered.

When two groups of hyperlipidemic rats received 40 and 70% germinated bengal gram powder at the expense of sucrose in hypercholesterolemic diet, it was observed that supplementation of 40% germinated bengal gram powder tends to bring the serum and liver lipid levels significantly towards normalcy. The insignificant reduction was observed in phospholipid levels of liver. Aorta triglycerides were decreased significantly while decrease in aorta cholesterol and phospholipid levels was suggestive. However, 70% germinated bengal gram powder failed to influence tissue lipid levels except serum triglycerides. Tables 2-4 confirm the earlier findings of Hathur et al. that bengal gram does exert hypolipidemic activity. It appears that under the experimental dietary conditions bengal gram is only capable of combating sucrose induced atherogenecity as
is evident from the failure of 70% bengal gram powder to
decrease the lipid parameters of serum. Supplementation of
70% bengal gram powder in the diet completely replaces sucrose
from the experimental diet although comparable control had 68%
sucrose. These findings also explain the failure of Aitya Sand
and Kapoor (1973) in observing the beneficiaries effect of bengal
gram.

The powdered germinated bengal gram was fractionated
according to the method used by Sevi and Kurup (1972) for black
gram. The scheme of fractionation is shown in Fig. 1. Among
the isolated fractions of bengal gram, the yield of insoluble
carbohydrates was maximum while that of protein was minimum.
The hypolipidemic activity of each fraction was tested in various
systems.

Carattini et al. (1961) and Anoletti (1962) suggested the
use of Triton-induced hyperlipidemia as an approach to screen
or to differentiate the mechanism of action of hypolipidemic
drugs. It is evident from Table 5 that three fractions of bengal
gram viz., fatty acids, globulin and insoluble carbohydrates
effectively counteracted the hyperlipidemic response of Triton in
rats. The cholesterol and triglyceride levels were decreased in
animals treated with all these fractions. Significant decrease in
phospholipid level was observed in animals treated with fatty acids.
and insoluble carbohydrates. Prolamine fraction caused a
significant lowering of triglyceride level. There was no
significant difference in any of the lipid levels in animals
treated with unsaponifiable portion and hot water extractable
portion. Cholesterol to phospholipid ratio (C/P), believed to
be an index of atherogenicity was markedly decreased in all
active fractions. Since Triton is known to physically alter
VLDL and bengal gram fractions are capable of counteracting
Triton-induced hyperlipidemia, the possibility of these
fractions exerting their hypolipidemic effect through inhibition
of increased lipoprotein synthesis appears likely. However, it is
also possible that bengal gram fractions accelerate the removal
of VLDL from blood possibly by release of Triton inhibition of
lipoprotein lipase. The latter mechanism gets support by the
ability of these fractions to counteract alcohol induced
lipidemia.

Therefore, the beneficiary effect of four active fractions
of bengal gram - fatty acids, globulin, prolamine and insoluble
carbohydrates, in Triton-1339 system was further investigated
under various dietary stresses.

Besides contributing to additional calories, alcohol
is known to accentuate hypertriglyceridemia by stimulating
fatty acid release from the adipose tissue and increasing VLDL
and chylomicrons from the circulation (Kuo, 1974). The increased \( \text{CHO:CHO} \) ratio generated by oxidation of ethanol could possibly result in increased hepatic triglyceride synthesis and decreased fatty acid oxidation.

Sabbiah and Kavi (1977) gave 35% alcohol in water and a cholesterol-free diet to pigeons and noted 40% higher plasma triglycerides than controls. They also concluded that alcohol feeding induces cholesteryl ester accumulation in the arterial walls, which may eventually lead to atherosclerosis. Genesis of alcohol-induced hypertriglyceridemia may be explained on the basis of observation of Kalformik et al. (1975). They have indicated the predominant role of peripheral lipolysis instead of the possibility of enhanced synthesis of palmitic acid in the liver. It is evident from tables (7-9) that bengal gram fractions viz., total lipids, fatty acids, gloelulin and insoluble carbohydrates fractions at the dose of 50 mg/kg brought a significant reduction of almost same magnitude as 200 mg nicotinic acid/kg body weight and 50 mg NaCl/Kg body weight (Yousufzai et al., 1976) and offered almost total protection against the lipemic effect of alcohol. Although it is premature to suggest the possible mechanism by which bengal gram fractions interfere with the observed modification of alcohol-induced enhancement of alimentary lipemia, it is tempting to suggest that interference of
bengal grass fractions in any of the metabolic processes could lead to the observed effects. As the decrease in serum lipids was not accompanied by a rise in liver lipids, the possibility of active bengal grass fractions inhibiting the release of lipoproteins could be excluded. The decrease in serum triglycerides caused by administration of bengal grass fractions suggests that like nicotinic acid (Barboriak and Leade, 1971) and NAC (Yousufzai et al., 1976) bengal grass fractions may inhibit the mobilization of free fatty acids from endogenous lipid stores. Recently Barona and Lieber (1975) have reviewed the effect of alcohol on lipid metabolism. They have discussed the role of alcohol on the development of atherosclerosis and coronary heart disease. Experiments in rats indicate that alcohol increases cholesterol synthesis in liver (Lefèvre et al., 1972) and the small intestine (Middleton et al., 1971). The increase in HDL has been emphasized in alcoholics by Johansson and Laurell (1969) and Johansson and Redhus (1974).

The data given in table 9-10 show decrease in serum and liver lipids in normocholesterolemic rats. There was no significant decrease in serum cholesterol levels. Serum triglyceride levels were decreased significantly in insoluble carbohydrates treated rats. Serum and liver phospholipid levels were decreased in rats treated with total lipids, fatty
acids, globulin and insoluble carbohydrates. There was no significant decrease in liver cholesterol. Liver triglycerides were decreased in total lipids, fatty acids and insoluble carbohydrates treated rats.

Bengal gram fractions were simultaneously administered for two weeks along with 0.5% cholesterol in basal diet. Total lipids and insoluble carbohydrates significantly checked the rise of serum lipids. Liver triglycerides and phospholipids levels were also decreased except phospholipid level in globulin treated animals (Tables 11-12). In contrast to the effect in normocholesterolemic rats, the percent decrease was low varying from 3.4% to 22.7%. The effect shown by prolamine and globulin was very low and insignificant while that of unsaponifiable portion was negligible. It is quite likely that two week duration of treatment with bengal gram fractions was not enough to overcome the hypercholesterolemic effect of cholesterol supplementation in diet. Nevertheless these fractions appear effective in combatting cholesterol-induced hyperlipidemia.

Rats maintained on fat rich cholesterol diet supplemented with bengal gram fractions for four weeks showed marked reduction in serum, liver and aorta lipids except aorta triglycerides (Tables 13-15). Total lipids and insoluble carbohydrates showed pronounced activity. Fatty acids and globulin caused good
effect while activity of unsaponifiable portion and prolamine was negligible.

Bengal gram fractions were administered along with atherogenic diet for four weeks in rats. It significantly lowered almost all serum and liver lipids. Aorta cholesterol were decreased by insoluble carbohydrates only. Total lipids and insoluble carbohydrates caused a significant reduction in aorta triglycerides and phospholipids. Fatty acids caused significant reduction only in aorta triglyceride level. Total lipids and insoluble carbohydrates showed marked activity (Tables 16-19).

The diet consumption of respective control and rats treated with fractions of bengal gram on cholesterolemic, fat-rich cholesterol diet and atherogenic diet was same. The average weight gain in treated groups was less than their respective control groups. From failure of treated animals to gain weight normally, one would like to conclude that lipid lowering effect of bengal gram fractions could be non-specific and due to reduced food intake or absorption. However, it may be noted that bengal gram fractions were administered simultaneously along with diets. In agreement to earlier findings in rabbits (Yusufi and Siddiqi, 1974), the increase in body weight of control groups may be due to fat deposition in other
tissues except liver where insignificant gain was observed. Therefore, it is quite likely that obesity induced by these fatty diets is counteracted by bengal gram fractions resulting in decreased weight gain in treated animals.

On the basis of above findings only four fractions of bengal gram viz., total lipids, fatty acids, globulin and insoluble carbohydrates appear to have hypolipidemic activity. Therefore these fractions shall henceforth be referred as "Active bengal gram fractions".

In accord with Niska and Ojala (1965) and Mill (1970), 10% fructose (v/v) in drinking water increases serum and liver lipids specially triglycerides. The decrease in cholesterol, triglyceride and phospholipid levels in serum, liver and aorta suggests that "Active bengal gram fractions" interfere at the site of cholesterologenesis and also inhibit fructose induced increase in fatty acid synthesis (Borts and Lynen, 1963) which ultimately results in less triglyceride formation.

Atherosclerosis has been produced in rats supposed to be highly resistant to atherosclerosis by intubating a mixture of Olive Oil, Vitamin D2 and cholesterol (1973). As shown in tables 22-25, lipid fractions of bengal gram at 50 mg/kg level effectively counteracted the hyperlipidemic and atherosclerotic
response of massive doses of vitamin \( \text{B}_2 \) and cholesterol in olive oil. The present study supports the findings of Altman (1973) and Yousufzai and Siddiqi (1976b) that a mixture of vitamin \( \text{B}_2 \) and cholesterol dissolved in olive oil results in hyperlipidemic conditions in rats. On oral administration of total lipids and fatty acids, all serum, liver and aorta lipids were significantly decreased except aortic cholesterol. There was little or insignificant lipid lowering effect of unsaponifiable portion observed in serum, liver and aorta. Our present study suggests that total lipid and fatty acid fractions of bengal gram may prevent hyperlipidemia and were also effective in regressing the atherosclerotic response of massive doses of vitamin \( \text{B}_2 \) and cholesterol. Other "active bengal gram fractions" viz., globulin and insoluble carbohydrates could not be treated due to their insolubility in olive oil. Kastogi (1977) has indicated the polyunsaturated vegetable oils as "serum cholesterol reducing agent". Mathur et al. (1964b) have reported that lipid portion of bengal gram possesses marked hypocholesterolemic activity in rats. In subsequent study they also compared the hypocholesterolemic activity of lipid extract of bengal gram with safflower oil, mustard oil and ground nut oil in rats (Mathur et al., 1968). They concluded that the lipid extract of bengal gram is the most potent
hypcholesterolemic fraction compared to other vegetable fats. Lipid extract of black gram has also been reported to possess hypolipidemic activity in rats (Devi and Kurup, 1972). Tables 22-25 indicate that almost entire activity of total lipids resides in its saponifiable portion i.e. fatty acids. The unsaponifiable portion as in the case of blackgram (Devi and Kurup, 1972) is ineffective.

Day (1962) suggested the role of phospholipids in the development of aortic lesion because its distribution was similar to those of cholesterol and neutral lipids. Peeters et al. (1970) believed that the structure of phospholipids may play a role in the stability of lipoproteins and their interaction with the arterial wall. Howard et al. (1972) and Schettler (1973) have also discussed the role of phospholipids in the development of atherosclerosis. Since cholesterol to phospholipid ratio is generally considered to be an index of atherogenecity, cholesterol appears to be incorporated into the cells of lesion, esterified within the cell and transformed into amorphous droplets of cholesteryl esters (Beller et al., 1968). Furthermore the presence of lecithin cholesterol transacylase in the arterial wall provides evidence for the esterification of cholesterol in aorta, since only free cholesterol rather than esterified cholesterol is exchanged with the blood during atherosclerosis.
Cholesterol is a well known major constituent of atherosclerotic lesions (Frantz and Moore, 1969). The accumulation of cholesterol in aorta parallels remarkably well with the serum cholesterol (Nichols et al., 1971). Velican (1972), Hess (1973), Govern and Robert (1974) have also studied the accumulation of cholesterol during the genesis of atherosclerosis. Stammann and Ahoads (1977) have discussed the role of serum triglyceride as the main risk factor of atherosclerosis. Nabega et al. (1977) have indicated the importance of triglyceride in the biochemical stage of atherosclerosis. Thus, the triglycerides play a critical role in the etiology of atherosclerosis.

Results described herein show that irrespective of dietary manipulations 50 mg of "active bengal gram fractions" per kg body weight exerts a significant hypolipidemic effect in normal and experimental rats. Among nine fractions of bengal gram, four fractions viz., total lipids, fatty acids, globulin and insoluble carbohydrates exhibited marked hypolipidemic activity. Table 26 summarizes the range of per cent reduction in lipid levels in various dietary conditions. Prolamine has also shown hypolipidemic activity although of smaller magnitude.
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<td>TRIGLYCERIDES</td>
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Mode of inducing hyperlipidemia for evaluation of lipid lowering activity of bengal gram fractions is indicated in parentheses:

A - Normcholesterolemic diet, B - Cholesterolemic diet, C - Fat-rich Cholesterol diet, D - Atherogenic diet,
E - Triton treatment, F - Alcohol intubation, G - Epinephrine feeding, H - Massive doses of vitamin D2 and cholesterol in olive oil.
Bombay et al. (1975) have shown that essential oils of onion and garlic are more effective than clofibrate. Kastogi (1972) has indicated the role of vegetable oil as a potent cholesterol reducing agent. Mathur et al. (1964b, 1968) have also discussed the role of lipid extract of bengal gram in counteracting hyperlipidemia. The probable mechanism of action of lipid extract of bengal gram can be explained in terms of increased excretion of cholesterol and their end products and differ from other unsaturated fats which cause a redistribution of cholesterol between serum and tissues. Oral administration of lipid fractions (total lipids, fatty acids and unsaponifiable portion) to hyperlipidemia induced by Vitamin D2 and cholesterol in olive oil has proved that the hypolipidemic activity of the total lipids resides in its fatty acid portion.

The nature of dietary proteins has been shown to have a definite effect on lipid level both in man and experimental animals. Casein has been found to be atherogenic in both (Albanese et al., 1959; Magee and Fragola, 1959; Meeker and Kesten, 1941; N. Math et al., 1959), while a diet rich in leguminous seeds has been shown to lower serum cholesterol levels in comparison with a diet containing sucrose, milk and vegetables. A significant decrease in serum cholesterol in man
was observed when a diet high in animal protein was changed to diet containing 25gm legume or cereal protein per day.

Cybulskaj $et$ al. (1979) have indicated the role of vegetable proteins in prevention of development of atherosclerosis.

Devi and Kurup (1972) have also observed the protective role of blackgram globulin in hyperlipidemic rats. In case of bengal gram, Mathur $et$ al. (1964b) have shown the hypolipidemic activity of its protein fraction. The results now obtained indicate that globulin fraction has considerable hypolipidemic effect.

Prolamine fraction is also hypolipidemic although to a little extent. As evident from Fig. 1, the prolamine fraction is obtained from alcohol extract of defatted powder of germinated gram. Biochanin $A$ and formononetin have also been extracted from germs of germinated bengal gram using 90-95% alcohol. Alcohol used for prolamine extraction was 70%. Little but significant activity of prolamine fraction may be due to the presence of biochanin $A$ and formononetin which are not totally extracted because of using dilute alcohol or using whole germinated gram instead of germ only.

Insoluble carbohydrates fraction of bengal gram has shown maximum hypolipidemic activity in almost all systems investigated in the present study (Table 26). Several other reports also indicate that dietary carbohydrates have influence serum
cholesterol both in man and rats. Polysaccharide fractions associated with the seed coat of some legumes have been shown to exert a cholesterol lowering effect by facilitating the fecal excretion of bile salts (Devi and Kurup, 1970, 1973; Vijayagopal and Kurup, 1975; Prem and Kurup, 1975). Tapioca starch exhibits significant hypolipidemic action in rats (Nishida et al., 1958). Another polysaccharide fraction containing 2.53% N has been isolated from bran and husk of paddy which has a marked hypolipidemic effect in rats fed a high fat high cholesterol diet (Vijayagopal and Kurup, 1972). This polysaccharide fraction keeps the total cholesterol and phospholipid of the serum, liver and aorta at a similar low level to that in animals fed glucose.

By preliminary characterisation of "Active bengal gram fractions", we may have an idea of chemical nature of the same. Total lipids seem to contain high molecular weight fatty acids due to its low saponification value. IR spectra clarifies that it is nothing but a complex mixture of fatty acids, unsaponifiable matters, sterols etc. We have proved that whole activity of total lipids resides in its fatty acids portion. IR spectra of fatty acids shows that no unusual group like OH, Keto, epoxy, cyclopropane, acetylenic, conjugation and trans unsaturated allene are present in fatty acids. GLC of the same
indicates that fatty acids are mixture of saturated (Palmitic and Stearic acid) and unsaturated fatty acids (Oleic, Linoleic and Linolenic acid). The proteins of gram are generally deficient in tryptophan, cystine and methionine and show an essential amino acid score at only 33.5 as compared to 100 given by a standard reference protein (Nao and Subramanian, 1970). The most active fraction of bengal gram is insoluble carbohydrates. It is a mixture of starch, polyfructosans, soluble sugars and some hexoses. The starch constitutes about 50% of the dry weight of dormant bengal gram. Krishna Murli (1974) has studied the per cent change in content of starch polyfructosans, hexoses and soluble sugars during germination. The degradation of starch and polyfructosans in bengal gram during germination gives rise to freely metabolizable sugars. The activation of α-glucosidase and α-amylase suggests that degradation of starch is by the hydrolytic pathway. The insoluble carbohydrate fraction of black gram is probably an amino sugar containing 3.36% N. Same fraction of bengal gram may be considered as amino sugar because it also contains 3.4% nitrogen content.

A preliminary effort revealed that total lipid fraction has both biochanin A and formononetin. The insoluble carbohydrates of bengal gram contain a compound similar to biochanin A
but also has two more UV fluorescent spots. It is, therefore, concluded that hypolipidemic activity of various bengal gram fractions may not be exclusively due to biochanin A and formononetin. This gets support by the insignificant effect of biochanin A and formononetin (Siddiqui and Siddiqi, 1978) and significant effect of "active bengal gram fractions" in normocholesterolemic rats. Krishna Murti (1974) has already suggested that bengal gram has a large variety of hypolipidemic compounds.

The results so far obtained suggest that various fractions of bengal gram are well tolerated and non-toxic in rats. This is not surprising in view of the chemical composition of the bengal gram, a legume commonly consumed by a large section of population throughout the world.

The work reported in this thesis definitely shows that bengal gram has potent hypolipidemic action and supports the earlier studies of Mathur et al. (1963, 1964a, 1964b, 1965, 1968, 1972). It further shows that bengal gram has at least four hypolipidemic fractions (total lipids, fatty acids, globulin and insoluble carbohydrate). This study resolves the controversy posed by the work of Devi and Kurup (1970) and Mitra and Nanavati (1973) and establishes beyond doubt the therapeutic value of bengal gram in hyperlipidemia. Being a major legume in our dietaries, the potential
of development of new hypolipidemic drugs from bengal gram with minimum side effects in treatment of coronary heart disease is eminent. It may be interesting to point out that in Indian customs bread made out of bengal gram flour is prescribed to heart patients. The studies so far completed suggest that eating of 1.5" germinated bengal gram would be useful to patients suffering from heart ailments provided it does not cause diarrhoea and constipation.