while feeding experimental diet and analysed for serum cholesterol[8], triglyceride[9] and high density lipoprotein cholesterol[10]. Body weight was recorded in the beginning and at the end of Ist, 2nd, 4th and 8th weeks.

The data were statistically evaluated by applying students *t* test.

Analysis of blood samples after feeding leaves of *Trigonella foenum gracecum* 10 g daily to the normal albino rabbits at the end of Ist, 2nd, 4th and 8th week revealed that there occurred a continuous lowering in serum cholesterol and triglyceride. On the other hand, HDL-cholesterol showed a continuously rising pattern. Body weight showed an increasing pattern during experiments. Upon statistical evaluation of the data, the observed changes have been found to be highly significant ($P < 00.01$ to < 0.001) (table 1).

Trigonella foenum gracecum leaves form part of spices and are consumed as a vegetable in the Indian dietaries. Though there are reports regarding the hypocholesterolemic effect of its seeds, no studies have yet been made on the biochemical effects caused by administration of the leaves in the body as both seeds and leaves possess almost the same composition[11].

The present study shows that the daily addition of cooked leaves to the diets of normal rabbits within eight weeks can produce significant lowering of cholesterol and triglycerides in circulation. The high density lipoprotein cholesterol is significantly increased. Obviously, the low density lipoprotein cholesterol must decline since total cholesterol shows an overall decrease. Fall in circulatory cholesterol and triglycerides brought about by the administration of fenugreek leaves is probably due to both fibre and saponin[2,5,12,13].

Biochemically, lowering of the total cholesterol, triglycerides, low density lipoprotein cholesterol and rise in high density lipoprotein cholesterol on *Trigonella foenum gracecum* containing diet could provide a valuable tool for maintaining cardiovascular system in healthy conditions[6,7,14].

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LEVELS OF ARGINASE AND TRANSAMINASES IN POLYCHLORINATED DIBENZOFURAN (PCDFs) FED RATS

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THE chlorinated dibenzofurans (PCDFs) have received wide publicity and attention in recent years as potential toxic environmental pollutants. Polychlorinated dibenzofurans are found as contaminants in commercial polychlorinated biphenyls[1], chlorinated phenol mixtures[2] as well as in certain trichlorophenol derived herbicides[3]. PCDF administered rats showed decrease in body weight[4]. The liver was the major depot of PCDFs, where they were metabolized and excreted mainly through the feces[5]. Since, the liver plays an important role in synthesis, degradation and detoxification pathways, special attention was focussed to study the effect of PCDFs on liver metabolism in rats. In the present investigation a few important enzymes of protein catabolism and blood urea have been studied.

PCDF mixture used was a generous gift from Professor Yoshito Masuda, Daiichi College of Pharmaceutical Sciences, Fukuoka, Japan. Its composition is available elsewhere[6]. Weanling albino male rats derived from Wistar strain were obtained from Veterinary College, Madras. The animal were fed with commercial pelleted rat chow with paired feeding and water *ad libitum* along with 100 mcg of PCDF mixture/kg body weight/day orally (sublethal dosage) for 30 days. The dosage was based on the report of Oishi *et al*[4]. The animals were then sacrificed by cervical decapitation and 1 ml of blood was collected with potassium oxalate as anticoagulant, the remaining blood was collected without any anticoagulant and the serum was separated. Blood urea was estimated by the method of Geyer and Dabich[7].

The liver dissected out was immediately washed with ice-cold saline and an appropriate amount of liver was homogenized in 0.1 M tris-HCL buffer pH 7.4. The homogenate was centrifuged at 2,500 rpm for 10 min. The supernatants served as the enzyme source. Aspartate amino tranferase (E. C. 2.6.1.1) and alanine amino transferase (E. C. 2.6.1.2) were assayed by the method of King[8]. Herzfeld and Raper's[9] method was adopted for the assay of arginase (E.C. 3.5.3.1). The protein was estimated by the method of Lowry *et al*[10].

The levels of serum, liver transaminases and arginase of control and experimental rats are given in table 1. Blood urea levels are given in table 2. It is evident that serum transaminases increased significantly whereas the liver transaminases, arginase and blood urea are decreased in PCDF mixture fed rats.

Table 1 *Levels of serum, liver transaminases and arginase in control and PCDF mixture fed rats. Values are expressed as mean ± S.D. from 6 animals in each group*

	Control	Test
SGOT	26.6±3.0	38.2±2.7*
SGPT	10.2±1.6	15.3±1.0*
Liver		
GOT	113±4	83±3*
GPT	219±8	106±5*
Arginase	930±10	435±11*

SGOT, SGPT : Iu/L
GOT, GPT : n mol of pyruvate liberated per min per mg protein.
Arginase : n mol of urea liberated per min per mg protein.

*$P<0.001$.

Table 2 *Levels of blood urea in control and PCDF mixture fed rats. Values are expressed as mean ± S.D. from 6 animals in each group*

	Control	Test
Blood urea mg/dl	26.4±2.2	22.0±1.7*

*$P<0.01$

Adolph and Lorenz[11] have pointed out that severe hepatocellular lesions with hepatic parenchymal cell necrosis are usually accompained by a marked elevation of serum transaminases. We have also observed hepatic parenchymal cell necrosis in PCDF mixture fed rats (unpublished data) and this may be the principal cause for the increased level of transaminases in serum. It has been reported earlier that blood urea was decreased in PCDF mixture fed rats[4]. The decreased blood urea level is accompanied by a concomitant decrease in the activity of liver arginase indicating decreased protein catabolism in PCDF mixture fed rats. This is also supported by the observed decrease in hepatic aspartate amino transferase and alanine amino transferase activities in the experimental animals.

It is therefore suggested that the decrease observed in hapatic arginase and transaminases may lead to decreased protein catabolism in PCDF mixture fed rats.

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SALIVARY IMMUNOGLOBULIN 'A' IN PEPTIC ULCER PATIENTS

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IMMUNOGLOBULIN A (IgA), which represents a relatively small fraction of the serum immunoglobulins, is the predominant species in most external secretions; i.e, secretions that bathe mucous membranes having continuity with the external environment. In man, secretory IgA and polymeric IgA come from the mucosal sites, especially the gut. In healthy adults, 80–90% of intestinal immunocytes produce IgA, and their levels tend to rise in patients with a variety of mucosal inflammation diseases[1]. We do not know much about their levels in peptic ulcer patients. In this study, the levels of IgA in saliva of peptic ulcer patients have been measured to find if they differ from those in normal persons.

Saliva was collected from patients and control subjects and centrifuged to remove particulate matter. The supernatant was used for IgA determination by the single radial diffusion technique[2]. The required monospecific antiserum was mixed with agar before overlaying it on the slides and the test solutions (10μl) are put in wells cut in the gel. Standard samples of varying concentrations are also put in wells on each test plate and the plates are incubated for 18 hr at room temperature. Depending on the concentration, immunoglobulins in the test diffuse out to form precipitation rings around the wells. The diameter of the rings is proportional to the immunoglobulin concentration in the saliva.

Table 1. *Salivary IgA levels in peptic ulcer patients and controls*

Sample	No.	IgA (mg/dl) $X \pm$ SD		Interpretation
Controls	24	6.93 ± 1.93		
Duodenal ulcer	40	6.95 ± 2·33	0.029	$P < 0.05$
Gastric ulcer	19	11.55 ± 3.95	5.030	$P < 0.01$

Levels of IgA in the saliva of 24 controls, 40 duodenal ulcer and 19 gastric ulcer patients have been determined. Results are shown in table 1.

Duodenal ulcer patients have levels similar to those of controls, while gastric ulcer patients have levels nearly 1½ times greater than that in controls. It was also found that all gastric ulcer patients with levels above the normal range ($\bar{X} \pm$ 2SD of controls) had associated gastritis, whereas gastritis was seen in only one patient with IgA level in the normal range.

In the external secretions, IgA serves to protect against infection and limits access of antigens to the general circulation[3]. The gut is the major source of IgA in the body and immunoglobulin-deficient patients have been found to have an increased prevalence of gastric disorders[4].

One of the theories put forward for the decreased resistance of the gastrointestinal mucosa to acid-pepsin action is that hematogenous infection with organisms of low grade virulence may cause inflammatory foci in the stomach wall leading to necrosis with subsequent digestion and ulcer formation[5]. Many workers are of the view that chronic ulcer does not develop in a healthy mucosa and that gastritis precedes development of an ulcer. There is general agreement that local immunoglobulin formation is important for the maintenance of a normal intestinal mucosa.

Results have shown a significant increase in the IgA levels in saliva of patients with gastric ulcer compared to those in controls and duodenal ulcer patients. Since it is known that gastric ulcer is often associated with gastritis and an inflammatory change, we could suppose that under such conditions, the gut is stimulated to produce more IgA. It is not yet clear whether this causes other mucosal membranes also to secrete an excess of IgA. But such a significant difference as has been found clearly indicates that measurement of salivary IgA helps in the differential diagnosis of gastric ulcer associated with gastritis.