presented in Table II. It is discernible that the incidence of *V. parahaemolyticus* was significantly higher ($P = 0.05$) in detritus feeders than in other feeding groups.

In all four feeding groups, *V. parahaemolyticus* could be isolated more frequently from faecal samples of fishes than from the external surface or gills (Table I), suggesting that the gastro-intestinal tract of fishes may provide a suitable niche for their proliferation. The rich nutrient store available in the gut of fishes[16] may also aid the colonization of this pathogen especially during periods of unfavourable environmental conditions. Kaneko and Colwell[4] have postulated that *V. parahaemolyticus* can survive low temperatures of winter in sediments and in association with, or in, shell-fish and scavenger fish such as gobies. The results of the present study aver with the above observations, since it appears that the gut of fishes might serve as a 'provisional' environment to tide over unfavourable conditions like hypersalinity (tropical systems) and low temperatures (temperate systems). In planktivores, isolation of *V. parahaemolyticus* from the gills was quite high (Table I). The association of *V. parahaemolyticus* with zooplankton has been well documented[17]. The gills of planktivores are suitably developed to sieve plankton from water and such contact may result in *V. parahaemolyticus* adhering to the mucus of gill surface, resulting in the greater incidence of this pathogen in this region.

V. parahaemolyticus caused gastroenteritis, is less frequent in India compared to Japan since food items are well cooked and spiced here. However, chances of cross contamination *via* kitchen utensils or by handling may result in infection especially while handling mullets, the potential reservoirs of this pathogen. Degutting fishes immediately after the catch and washing them well would be advisable precaution to reduce the load of *V. parahaemolyticus* in such contaminated fishes.

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1. Miwatani, T. and Takeda, Y., *Vibrio parahaemolyticus, A Causative Bacterium of Food Poisoning*, Saikon Publishing Co., Ltd., Tokyo, 1976.
2. De, S. P., Banerjee, M., Deb, B. C., Sengupta, P. G., Sil, J., Sircar, B. K., Sen, D., Ghosh, A. and Pal, S. C., *Indian J. Med. Res.*, 1977, **65**, 21.
3. Natarajan, R., Martin Abraham, Ravi Gautam and Balakrish Nair, G., *Proceedings of the International Sym. on Environmental Agents and Their Biological Effects*, Int. S-2-59, 1978.
4. Kaneko, T. and Colwell, R. R., *J. Bacteriol.*, 1973, **113**, 24.
5. Hugh, R. and Sakazaki, R., *J. Conf. Public Health Lab. Directors*, 1972, **30**, 133.
6. Bockemuhl, J. and Triemer, A., *Bull. World Health Organ.*, 1974, **51**, 353.
7. Varga, S. and Hirtle, A., *J. Fish. Res. Board Can.*, 1975, **32**, 541.
8. Kampelmacher, E. H., Mossel, D. A. A., Van Noorle Jansen, L. M. and Vincentie, H., *J. Hyg.*, 1972, **68**, 189.
9. Chun, D., Chung, J., Seol. S. Y. and Tak, R., *Am. J. Trop. Med. Hyg.*, 1974, **23**, 1125.
10. Thompson, C. A., Vanderzant, C. and Ray, S. M., *J. Food Science*, 1976, **41**, 117.
11. Manavalan, S., Dhevendaran, K., Vanajakumar and Selvakumar, N., *Curr. Sci.*, 1977, **46**, 821.
12. Thomson, J. M., *Aust. J. mar. Freshwat. Res.*, 1954, **5**, 469.
13. Mary, P. P., *Ph.D. Thesis*, Annamalai University, India, 1977.
14. Moriarty, D. J. W., *J. exp. mar. Biol. Ecol.*, 1976, **22**, 131.
15. Bailey, N. T. J., *Statistical Methods in Biology*, The English Language Book Society, London, 1959.
16. Doudoroff, M., *J. Bacteriol.*, 1942, **44**, 461.
17. Kaneko, T. and Colwell, R. R., *Appl. Microbiol.*, 1975, **29**, 269.

HAEMATOLOGICAL STUDIES IN *SAROTHERODON* (TILAPIA) *MOSSAMBICA* (PETERS) EXPOSED TO LETHAL (Lc_{50}/48 HRS) CONCENTRATION OF SUMITHION AND SEVIN

ORGANOPHOSPHATE and carbamate insecticides are now extensively used in plant protection operations on account of their less persistence in the environment. But their excessive and indiscriminate use produce more hazards to non-target and useful fauna of freshwater environment. It was found that exposure to Sumithion and Sevin inhibited enzymes like AchE, SDH but increased Ach content, LDH activities[1] and inhibited tissue respiration in the fish *T. mossambica*[2]. Many vital physiological processes are altered in *T. mossambica* after pesticide treatment[3]. The present communication deals with the effect of Sumithion an organophosphate compound and Sevin, a carbamate insecticide on certain haematological parameters.

Maintenance, size and weight range of fish used have been described earlier[1]. Lc_{50}/48 hrs value as calculated by probit method[4] was 6 mg/l for Sumithion

and 10 mg l for Sevin. Commercial grade Sumithion (Fenitrothion, dimethyl 4-methyl-4-nitrophenyl phosphorothionate) and Sevin (carbaryl; 1-naphthyl N-methyl carbamate) were used in the experiment. Blood for red-blood cell count, packed cell volume, and haemoglobin content was obtained by direct heart puncture. Red cell count was made with Neubauer crystalline counting chamber; haemoglobin content was estimated by acid haematin method. Packed cell volume was obtained after centrifuging the blood at 3,000 rpm for 5 minutes in a haematocrit tube. The erythrocyte column height in millimeters gives the percentage packed cell volume. Mean corpuscular volume (MCV) representing the average volume of the red cells was obtained from the packed cell volume (PCV) and red cell count (RBC). PCV is divided by red cell count and the result is multiplied by 10. MCV is expressed in cubic micra ($c\mu$). Mean corpuscular haemoglobin (MCH) represents the average weight of haemoglobin contained in each cell. For getting the MCH the haemoglobin is usually divided by red cell count and the result is multiplied by 10. MCH is expressed as micromicrograms (μ/g). MCHC represents the average concentration of haemoglobin in the red cells. To obtain MCHC, haemoglobin is divided by packed cell volume and the result is multiplied by 100. MCHC is expressed in terms of percentage.

Blood values, namely, red blood corpuscles (RBC), Mean corpuscular volume (MCV), packed cell volume (PCV), Haemoglobin concentration (Hb), Mean corpuscular haemoglobin (MCH) and Mean corpuscular haemoglobin concentration (MCHC) of *T. mossambica* exposed to lethal (Lc_{50}/48 hrs.) concentration of Sumithion and Sevin are presented in Table I.

TABLE I

Haemogram of T. mossambica *exposed to lethal* (Lc_{50}/48 *hrs.*) *concentration of Sumithion and Sevin*

Blood parameter	Control	Sumithion	% change	Sevin	% change
(A) Red blood corpuscle (RBC) ($\times 10^6$ m'c.m.m.)	2·56 ±0·454	2·12[a] ±0·394	−17·18	2·15[a] ±0·261	−12·10
(B) Mean corpuscular volume (MCV) ($c\mu$)	111·32 ± 21·64	119* ± 18·12	+7	122·88* ± 16·45	+10·38
(C) Packed cell volume (PCV) (%)	28·5 ±1·20	25·23 ±0·698	−11·47	26·42 ±1·22	−7·29
(D) Haemoglobin concentration (Hb) (gms/100 ml.)	8·2 ±0·322	7·55[b] ±0·575	−8	7·45 ±0·677	−9·14
(E) Mean corpuscular haemoglobin (MCH) ($\mu\mu$ gms)	32·03 ± 6·89	35·61* ± 7·06	+11·17	34·65* ± 5·19	+8·17
(F) Mean corpusuclar haemoglobin concentration (MCHC) (%)	28·77 ±1·86	29·92* ± 2·71	+4	28·19* ± 2·60	+2·01

Values expressed are mean ± S.D. for 6 individual observations.
Changes after pesticide treatment are statistically significant $P < 0·001$.
a, $P < 0·05$, *b*, $P < 0·02$; * Not significant.

Exposure to Sumithion and Sevin decreased blood values like RBC, PCV and Hb concentration. Increase observed in other parameters namely, MCV, MCH and MCHC, however was not statistically significant. With Sumithion, the decrease of RBC (− 17·18%; $P < 0·05$), PCV (− 11·47%; $P < 0·001$) and Hb (− 8%; $P < 0·002$) was statistically significant. Similarly with Sevin, the per cent decrement of RBC (− 12·10%; $P < 0·05$) PCV (− 7·29; $P < 0·01$) and Hb (− 9·14%; $P < 0·01$) was significant.

Sub-lethal parathion treatment reduced haematocrits, number of erythrocytes and leucocytes in the blood of golden shiners, *Notomigorus crysoleucars*[6]. Similarly, treatment of organophosphate pesticide DDVP to chicken decreased leucocyte count, but increased peripheral blood leucocyte and erythrocyte count[6]. It has been reported that repeated administration of Carbyne, Maneb, Eptam, Yelan and TMTD (Thiram) (all carbamates) orally to rats decreased the protective properties of leucocytes especially the ability

of neutrophils to digest phagocytosed microbes and increased the coagubility of blood indicating thrombiotic and haemorrhagic complications in the poisoned animals[8]. It is apparent from Table I that the per cent reduction of RBC, PCV and Hb values in Sumithion and Sevin treated fish did not exhibit any appreciable difference between them. The decrease of RBC, PCV and Hb values, results in 'hypochromic microcytic anemia' which was attributed to deficiency of iron and its decreased utilisation for 'Hb' synthesis[9]. It is well known that glycolysis is concerned with the reduction of methemoglobin as soon as it is formed, thus maintaining the iron of the 'Hb' in the ferrous form in which state only, it acts as an efficient oxygen carrier. The increased activity of LDH, decreased cellular oxidations[1] and cellular respiration[2] in Sumithion and Sevin exposed fish indicate the prevailing of anaerobic segment of glycolysis. The disruption of iron synthesising machinery due to inhibition of aerobic glycolysis could be the reason for the decrease of blood values in the stressed fish.

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1. Ranganatha Koundinya, P. and Rama Murthi, R., *Irdian J. Exp. Biol.*, 1978, **16**, 809.
2. — and —, *Indian J. Environ. Health*, 1978, **20**, 426.
3. —, *Ph.D. Dissertation* submitted to Sri Venkateswara University, Tirupati, 1978.
4. Finney, D. J., *Probit Analysis*, Cambridge University Press, London, 1964.
5. Butler, J. R., *J. Miss. Acad. Sci.*, 1970, **15**, 33.
6. Dimitriev, A. I. and Leningr, *Vet. Inst.*, 1975, 40, 223.
7. Olefir, A. I. and Karpenko, V. N., *Gig. Tr.*, 1971, 7, 89.
8. Karpenko, V. N., *Vrach Delo*, 1975, **1**, 122.
9. Bhai, Idrees and Nath, N., *Proc. Soc. Exp. Biol. Med.*, 1971, **138**, 597.

EFFECT OF BRAIN AND CORPORA ALLATA EXTRACTS ON THE LIPID PROFILE OF THE HAEMOLYMPH OF *ACHAEA JANATA* L.

The lipid profile of insect haemolymph varies with the physiological state of the animal and reflects the role of this tissue as a medium for the transportation of materials from sites of absorption or synthesis to sites of utilization or storage. That corpora allata, control certain aspects of lipid metabolism in the various tissues of insects is now very well understood. Allatectomy increases the total lipid content and also stimulates turn-over of phospholipids and triglyceride fractions[1]. In the allatectomised *Schistocerca gregaria* Walker and Bailey[2] have demonstrated considerable increase in triglyceride content of the fat body but no appreciable effect on the haemolymph lipid level. The role of cerebral neurosecretory material on carbohydrate as well as on the lipid metabolism in the fat body of some insects has also been suggested[3,4]. While working with *Cecropia* moth Gilbert and his associates[5] have shown that corpora allata stimulate incorporation of (1-C^{14}) palmitate into ovarian glycerides. It is well understood now that glycerides provide some of the energy for embryogenesis. *In vitro* studies on the female *Lucophaea maderae*[6] have suggested that the corpora allata act on both the fat body and ovary by making more lipid available for storage in the maturing oocytes. It is apparent from the aforementioned observations that the corpora allata appear to control lipid metabolism.

Adult female moth *Achaea janata* L., the larvae of which are serious pest on castor plant, has a very well developed corpora allata. It is believed that the glycerides from the fat body are being released into the haemolymph, which are then transported to the developing oocytes. The experiments outlined below are meant to ascertain the influence of the extracts of corpora allata and brain, on the lipid release from the fat body of the moth *Achaea janata* L.

The adult female moths used in the present experiments were collected from the laboratory culture. The procedure for the collection of haemolymph and the fat body was essentially similar to that described elsewhere[6]. The corpora allata and brain were removed carefully from several individuals, pooled and homogenized at 3–4° C in a known volume of distilled water. The homogenates were used as such to test their influence on the lipid release. The fat body as well as the haemolymph pooled from various individuals were used for each set of experiment. The incubation mixture in the Erlenmayer flask (10 ml capacity) consisted of the fat body (100 mg), 0·5 ml freshly collected haemolymph and 0·2 ml corpora allata extract (CAE) or brain extract (BE). The incubation was carried at 26° C for 90 min with constant shaking. The incubation mixture without CAE or BE in the incubation mixture served as control. Extraction of lipids from the incubation medium and its separation into monoglyceride (MGL), 1,2-diglyceride (1,2-DGL), 1,3-diglyceride (1,3-DGL), triglyceride (TGL) and free fatty acids (FFA) as well as their estimation was essentially similar to that described elsewhere[7].

The results obtained on the various glycerides and FFA levels of the haemolymph are summarised in Table I.

The medial neurosecretory cells of the brain in three species of mosquitoes is implicated in the regulation