studied by following the method of Bidari and Reddy[3]. The aphids for the study included were *Myzus persicae* Sulz, *Aphis gossypii* Glov, *Aphis craccivora* Koch, *Rhopalosiphum maidis* Fitch and *Hysteroneura setariae* Thomas.

This virus was successfully sap-inoculated and transmitted by aphids, *M. persicae*, *A. gossypii* and *A. craccivora* to *C. annuum* cvs California Wonder and Byadgi Kaddi. It was not seed borne.

The characteristic symptoms of this virus on *Capsicum* of both cultivars are prominent vein clearing on young leaves followed by mild chlorosis and cupping of leaves (figure 1, 2). After a month, young leaves showed green vein banding along the veins at the base of the lamina and flagging of older leaves (figures 3, 4). The main branches became much elongated. In hot days plants showed stuntedness and wilting. Fruits of infected plants were small, showed line patterns. Ripened fruits showed brownish coloured areas. *Capsicum frutescence* Tobacco showed top necrosis. These symptoms agree with that strain of TEV reported earlier[4-6]. Anderson and Corbett called this disease caused by TEV as "vein banding crinkle" in Central Florida. It also produced mild necrotic etching on *Nicotiana tabacum* cv 'Xanthi' and White Burley and later mosaic motlling.

The present virus resembled TEV in insect transmission as reported by other workers[6-9]. Physical properties of the virus were studied by following the method of Noordam[10]. The virus had dilution end point between 1:1000 and 1:5000, and thermal inactivation point between 60 and 70°C and longevity *in vitro* of 48 hr at 21–26°C. It differred slightly in its physical properties with that of strains of TEV as reported earlier[9,11,12].

In Ouchterlony test, the present virus did not show any reaction with antisera of 11 viruses reported on chilli indicating lack of serological relationship with these viruses. Electron microscopic studies of this virus showed the presence of flexuous rods measuring 623 × 12.0 nm (figure 5). The virus in its morphology and size resembled typical TEV as reported by the above workers. Based on these characters it was considered as TEV on chilli. Natural occurrence of TEV on *Capsicum* spp has not been reported earlier in India.

VBB is thankful to Dr D. Z. Maat, Wageningen, the Netherlands and Dr D. N. Raichoudhury, Calcutta for sending the antisera and identification of aphids respectively.

8 December 1984, Revised 7 October 1985

1. Kulkarni, G. S., *Poona Agric. Coll. Mag.*, 1924, **16,** 6.
2. Mc Rae, W., *Annu. Rept. Board Sci., Advice India*, 1924, **23,** 31.
3. Bidari, V. B. and Reddy, H. R., *Curr. Sci.*, 1984, **53,** 1045.
4. Greenleaf, W. H., *Phytopathology*, 1953, **43,** 564.
5. Greenleaf, W. H., *Phytopathology*, 1956, **46,** 371.
6. McKeen, C. D., *Can. J. Bot.*, 1954, **32,** 96.
7. Laird, E. F. and Dickson, R. C., *Phytopathology*, 1963, **53,** 48.
8. Kassanis, B. K., *Nature* (*London*), 1944, **3896,** 16.
9. Herold, F., *Plant Dis. Reptr.*, 1970, **54,** 344.
10. Noordam, D., *Identification of plant viruses.* Oxford and IBH Publishing Company, New Delhi, 1973.
11. Kassanis, B. K., *Ann. Appl. Biol.*, 1941, **28,** 238.
12. Verma, C. S. and Lal, R., *Proc. Indian Sci. Congr.*, 1964, **51** and **52,** III, 328.

OBSERVATIONS ON FUNGAL INFECTION OF *CARASSIUS CARASSIUS* L.

G. C. SRIVASTAVA, S. K. SINHA and S. K. PRABHUJI*

Department of Botany, St. Andrew's College, Gorakhpur 273001, India.

**Department of Botany, U. N. Postgraduate College, Padrauna 274304, India.*

THE parasitism of teleosts in freshwater by saprolegniaceous fungi is a worldwide phenomenon. These fungi are found to infect the eggs, fries, fingerlings and even adult fish. Generally, healthy fishes do not get infected by fungi, but once they get injured either mechanically or by the attack of aquatic predators, the infection sets in. Such infections usually result in epidemics causing even 100% mortality of the fish population[1].

During the course of investigations on fungi associated with fish diseases, 28 heavily infected specimens. of *Carassius carassius* L. (11 males and 17 females), ranging between 140 and 200 mm in length, were collected from the Garden water-tank of the office of N. E. Railway, Gorakhpur, during January–February, 1983. The infected fish when placed in clean water, showed white cottony patches and black and yellow galls scattered on their body (figure 1), but no infection was observed in the gill region. The infection

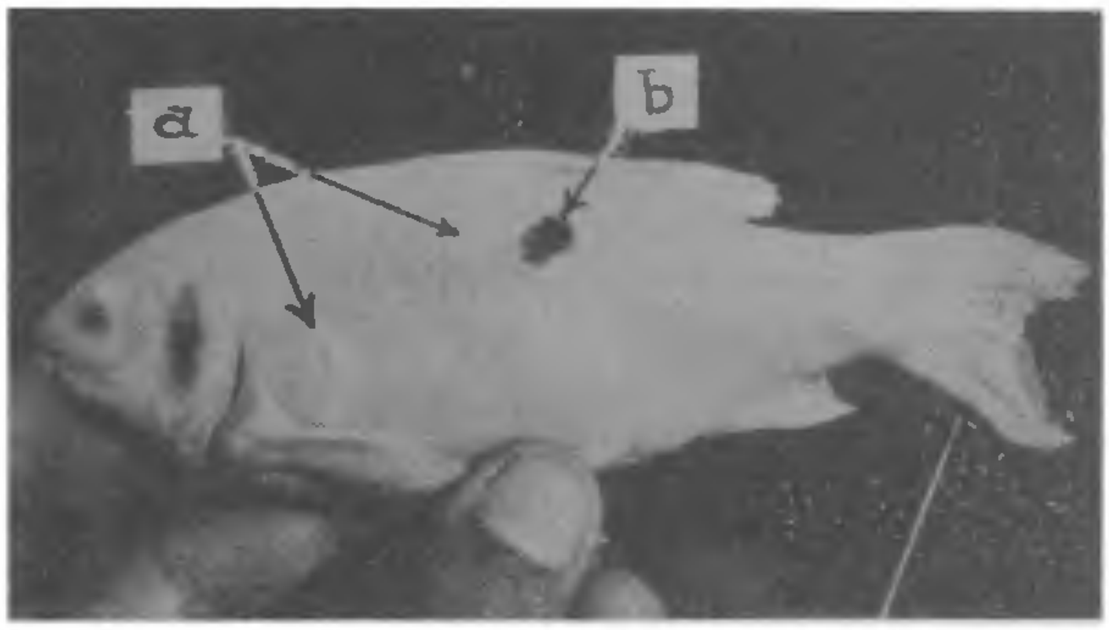

Figure 1. *Carassius carassius* L. bearing a white cottony patch (a) and a black gall (b) caused by *Saprolegnia diclina* Humphrey.

which was localized in nature resulted in mass mortality of the infected hosts.

Infected living and dead fish specimens were collected using hand-nets and brought to the laboratory in large-sized polythene bags half filled with fresh water. Small bits of mycelium were taken out from white cottony patches and rinsed thoroughly in distilled water and were then placed in petri-dishes containing 10 ml of sterile distilled water on boiled hempseed halves. Unifungal, bacteria-free cultures of the fungus were propagated on the lines described earlier[2–4]. The fungus was identified as *Saprolegnia diclina* Humphrey using the monograph of Seymour[5] and the fish specimen was identified as *Carassius carassius* L. with the key provided by Jhingran[6].

To ascertain the parasitic ability of the fungus, controlled infection test was conducted using *Cirrhinus reba* Ham, *Channa marulius* Ham, *Nandus nandus* Ham and *Notopterus notopterus* Ham as test fish on the lines of Scott and O'Warren[7] at room temperature ranging between 20 and 25°C. Hyphae of the fungus were observed protruding from the experimentally injured areas of the test fish within 9–15 hr of placing the fish in the infection troughs. These individuals died of infection resulting in dermal ulceration within 22–48 hr of exposure to the fungal inoculum (table 1). The fungus growing on these artificially-infected fish was isolated and compared with the culture of the original inoculum. It was found identical to the original fungus. For maintaining a control for the experiment, three test fish of each species were kept under identical conditions, but were not exposed to the fungal inoculum.

As the infection was on the body surface and no part of the body was found specially susceptible to the fungus, it could not be said definitely which part of the body of the fish succumbs first to the infection. Furthermore, no relation between the severity of infection and the sexuality of the naturally infected host fish could be established, as the symptoms of infection were similar in males and females.

Table 1 *Controlled infection studies demonstrating the parasitic ability of Saprolegnia diclina Humphrey*

Name of fish	Mycosis evident within hr	Death occurred within hr
Cirrhinus reba Ham	9–15	42–48
Channa marulius Ham	11–14	32–36
Nandus nandus Ham	10–12	22–24
Notopterus notopterus Ham	11–15	25–30
Control	–	–

Number of fish studied 3; Mycosis evident and number of fish dead 3.

In a host range study Srivastava and Srivastava[8] found that *Isoachlya anisospora* var *indica* (= *Saprolegnia diclina*)[5] was a virulent parasite of fish and their eggs. Later, Srivastava and Srivastava[9] reported this fungus as a parasite of *Cirrhinus mrigala* Ham, *Puntius sophore* Ham, *Labeo rohita* Ham, *Catla catla* Ham and *Labeo calbasu* Ham. Recently, Srivastava *et al*[10] reported the natural infection of *S. diclina* on *Cyprinus carpio* var *communis* L. In the present study *Saprolegnia diclina* has been found to be a naturally occurring parasite of *Carassius carassius* L. and its host range has been found extending to *Cirrhinus reba* Ham, *Channa marulius* Ham, *Nandus nandus* Ham and *Notopterus notopterus* Ham.

The authors are grateful to UGC, New Delhi, for financial assistance and to Dr J. Patterson. Thanks are also due to Prof. T. J. Varghese, Fisheries College, Mangalore for his kind help, and to authorities of N. E. Railway, Gorakhpur for assistance.

18 October 1984; Revised 18 April 1985

1. Scott, W. W., *Dev. in Ind. Microbiol.*, 1964, **5**, 109.
2. Raper, J. R., *Science*, 1937, **85**, 342.
3. Tiffney, W. N., *J. Elisha Mitchell Sci. Soc.*, 1939, **55**, 134.
4. Johnson, T. W. Jr., *The genus Achlya: Morphology and taxonomy*, University of Michighan Press, Ann. Arbor., Michighan, 1956.
5. Seymour, R. L., The Genus *Saprolegnia*, *Nova Hedwigia*, 1970, **19**, 1.
6. Jhingran, V. G., *Fish and fisheries of India*, Hindustan Publ. Corporation, India, 1983.

7. Scott, W. W. and O'Warren, C., *Tech. Bull. Vir. Polytech. Inst. Virginia*, 1964, **171**, 1.
8. Srivastava, G. C. and Srivastava, R. C., *Fert. Tech.*, 1977, **14**, 140.
9. Srivastava, R. C. and Srivastava, G. C., *Mycopathologia*, 1978, **63**, 121.
10. Srivastava, G. C., Sinha, S. K. and Srivastava, S. K., *Curr. Sci.*, 1983, **52**, 927.

SERUM IMMUNOGLOBULIN STUDIES IN VDRL POSITIVE PATIENT

SHRAWAN KUMAR
Human Biology Unit, Anthropological Survey of India, Calcutta 700016, India.

IMMUNOGLOBULIN disorders are associated with a variety of symptoms and diseases. The immunoglobulin possesses many antibody activity and functions as a defence against foreign particles. Immunoglobulin G contains the majority of antibacterial, antiviral and antitoxic antibodies. Additional antibodies have been demonstrated including anti-insulin[1], anti-nuclear factors[2] and incomplete R_h antibodies[3]. Immunoglobulin A has been shown to possess a variety of functions such as antitoxins[4], anti-bacterial agglutinins[4,5], isoagglutinins[6], anti-insulin[2], skin-sensitizing antibodies or allergic reagins[7] and others. Immunoglobulin M possess most 'natural' antibodies, the ABO blood group isoantibodies, cold agglutinins, rheumatoid arthritis factors, antinuclear factors[2], heterophile agglutinating antibodies of infectious mononucleosis and antibacterial antibodies[4], especially those directed against gram-negative microorganisms[8].

Recent advancements in immunological studies have provided greater understanding about the functions of extremely complicated immune systems and also polyclonal and monoclonal gammopathies. The defective function of immune system results in disease. The immunoglobulin level plays an important role in maintaining homeostasis and health. Immunoglobulin disorders, if not treated or diagnosed correctly, can result in death. Thus, it becomes increasingly important to know the clinical symptoms associated with immunoglobulin disorders which will help in taking measures for prevention and proper treatment of the disease. In the present study, an attempt has been made to examine the immunoglobulin levels (G, A and M) in VDRL-positive patient and to find out any association between them.

In total 52 serum samples which showed strong VDRL-positive reaction, tested by VDRL slide flocculation test[9,10], were included in the present study. The control sera were collected from 44 normal adult individuals which showed no abnormality after haematological and clinical pathological investigation. Their health status was also studied to see that none of the individuals suffered with any current infections or parasitic diseases. The serum immunoglobulin level was determined by single radial immunodiffusion technique[11]. The details are reported elsewhere[12].

Table 1 shows the mean, standard deviation and standard error values of G, A and M immunoglobulin concentration in VDRL positive samples as well as in normal controls. The distribution of serum immunoglobulin concentration differed significantly from normality. Therefore, the skewed data on IgG, IgA and IgM are normalized by applying logarithmic transformation and the data are given in table 1. The IgG level is a little higher in VDRL positive patient as compared to normal controls, whereas, IgA level is

Table 1 *Serum immunoglobulin levels in VDRL-positive patient and normal individuals*

	VDRL positive patient (n = 52)			Normal individuals (control) (n = 44)		
Immunoglobulins	Mean	S.D.	S.E.	Mean	S.D.	S.E.
Raw data						
IgG (mg/100 ml)	1771.09	638.46	88.54	1539.77	567.99	85.63
IgA(mg/100 ml)	230.15	131.25	18.20	216.37	114.85	17.31
IgM(mg/100 ml)	205.82*	67.61	9.37	153.68	79.30	11.95
Log transformation						
log IgG	3.217	0.168	0.023	3.150	0.192	0.029
log IgA	2.286	0.265	0.036	2.226	0.389	0.058
log IgM	2.284*	0.168	0.023	2.128	0.229	0.034

*$P < 0.001$ as compared to normal individuals.