

<http://www.pjbs.org>

PJBS

ISSN 1028-8880

**Pakistan
Journal of Biological Sciences**

ANSI*net*

Asian Network for Scientific Information
308 Lasani Town, Sargodha Road, Faisalabad - Pakistan

Proliferative Kidney Disease at Romsey Fish Farm on the River Test, Hampshire, U.K

Momin Naich, ¹Fatima Mujib Bilqees and ²Nasira Khatoon

Department of Zoology, Government Degree Science and Commerce College Lyari, Karachi-75600

¹Department of Parasitology, Baqai Medical University Karachi-75600

²Department of Zoology, University of Karachi, Karachi-75270, Pakistan

Abstract: Proliferative kidney disease was investigated at a Romsey fish farm on the River Test, Hampshire, U.K. A total of 200 fish of age 0+ were collected from one selected tank between August and September 1987. The degree of PKD infection was classified low, medium and high. Regression analysis showed significant difference between PKD infected and uninfected fish. Logistic regression indicated that PKD had significant relationship with both (-ve) time and (+ve) temperature. Chloramine-T and Oxolinic acid were used as a chemical management.

Key words: Fish, proliferative kidney disease, romsey fish farm, river test, U.K

INTRODUCTION

Proliferative kidney disease (PKD) has become one of the most important disease of cultured rainbow trout *Salmo gairdneri* (Richardson) in European countries (Roberts and Shepherd, 1974). The disease was also noted in rainbow trout in North America and West coast of Canada (Smith *et al.*, 1984; Arther and Lom, 1985; Lester, 1974). This disease is associated with water quality and temperature (Clifton-Hadley *et al.*, 1984).

This investigation examines the outbreaks of proliferative kidney disease at Romsey fish farm on the River Test, Hampshire, U.K.

MATERIALS AND METHODS

The Romsey fish farm on River Test was selected for the investigation. The detail information about the fish farm is provided elsewhere (Naich and Bilqees, 1991). Twenty fish per sample were collected during August and September 1987, on ten occasion (i.e 200 fish of age 0+). They were randomly collected from one selected tank by dip net. Fish were transported to the laboratory for postmortem. Fish were killed by the approved method of anaesthesia benzocaine followed by pithing. (Under the Animals Scientific Procedure Act, 1986) within two hours of sampling. Clinical signs of PKD were noted and classified as low, medium or high according to kidney infection levels (Clifton-Hadley *et al.*, 1987). Zero PKD infection was also noted. Statistical analysis of data was computed with the help of IBM computer.

RESULTS

Fish were scored as uninfected or infected by looking at the kidney. The degree of infection was then classified as low, medium or high depending upon the kidney condition. The prevalence of PKD infected fish was high at higher temperature (Table 1). Infection prevalence declined with the decrease of temperature and increase of fish age. Consequently this may be the reason of PKD significant -ve relationship with time (age) and also +ve relationship with temperature (Fig. 3 and 4). Fish mortality was also high at high PKD prevalence. The detail of infection level (i.e low, medium and high) are also tabulated in Table 1.

At high PKD prevalence both Malachite green and Oxolinic acid were used to control the PKD. The decline PKD infection prevalence may have been influenced by growth of fish (= age), temperature and chemical treatment. Fish with heavy infection (mostly small fish) had abdominal distension and lateral swim bladder displacement. Liver and spleen were slightly enlarged and the gill colour was significantly paler only in small fish with infection. Heavy infected fish (compared with uninfected fish) had less fat deposited in the caecal region. Small fish with heavy infection had watery intestinal contents.

Proliferative kidney disease (PKD) significantly affected fish weight ($F=54.56$; $DF=1$ and 197 ; $P<0.001$), the decrease amongst fish with infection was -37.4 gm (at $-47.40-17.42$; confidence limits) (Fig. 1) and for fish length ($F=46.12$; $DF= 1$ and 197 , $P<0.001$), the decrease

Table 1: The prevalence of PKD infection together with the degree of gross pathology of infected fish and mortality per day with both time and temperature

Sample	Date	Temp.	PKD%	L%	M%	H%	Mort/ pay	C.M.T
1	24.8.87	16	95	47	21	32	60	-----
2	27.8.87	16	75	27	40	33	13	ML+OX
3	03.9.87	17	80	31	19	50	71	ML+OX
4	07.9.87	15	70	50	29	21	25	ML+OX
5	10.9.87	16	70	36	29	35	48	OX
6	14.9.87	14	65	46	31	23	40	OX
7	17.9.87	15	85	18	35	47	65	OX
8	21.9.87	16	50	20	50	30	62	-----
9	24.9.87	14	45	45	22	33	25	-----
10	28.9.87	12	30	50	33	17	25	-----

Temp Temperature PKD proliferative kidney disease % L Low PKD infection level %
 M Medium PKD infection level % H High PKD infection level % Mort/day Fish mortality per day
 C.M.T Chemical treatment ML Malachite green OX Oxolinic acid

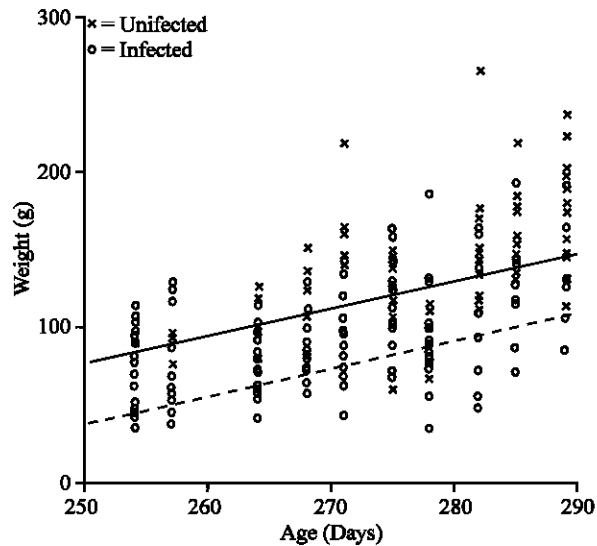


Fig. 1: Regression with age of the weight of rainbow trout *Salmo gairdneri* (Richardson) for fish infected and uninfected with PKD

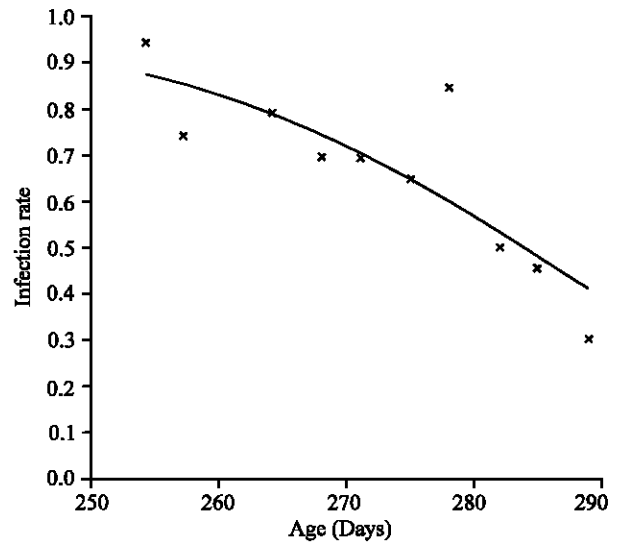


Fig. 3: Relationship between age and PKD infection rate in rainbow trout *Salmo gairdneri* (Richardson)

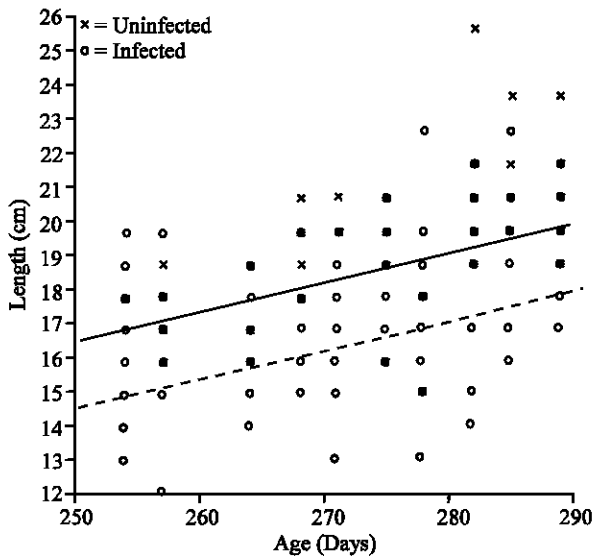


Fig. 2: Regression with age of the length of rainbow trout *Salmo gairdneri* (Richardson) for fish infected and uninfected with PKD

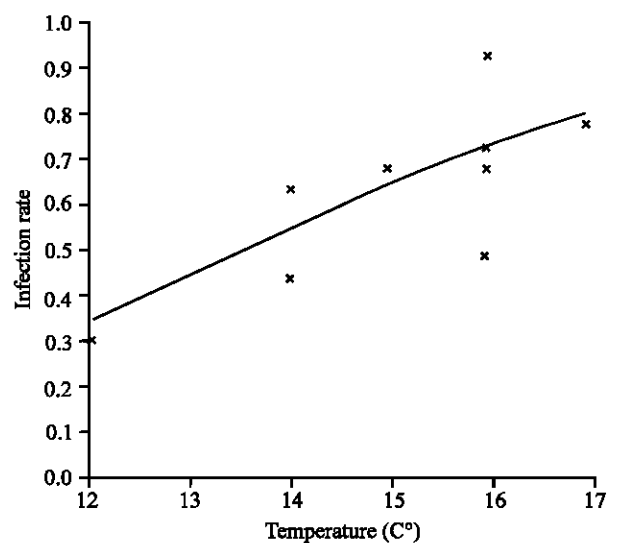


Fig. 4: Relationship between temperature and PKD infection rate in rainbow trout *Salmo gairdneri* (Richardson)

in fish length with infection was -2.02 cm (at -2.61, -1.43 confidence limits) (Fig. 2).

Logistic regression analysis of the data showed that PKD infection had significant relationship with both time and temperature. Fish age was calculated in days, therefore with increase of time (= days) PKD infection rate decreased by an approximate -6.8% (i.e. relative risk) of infection per day (at -9.6, -3.9% confidence limits) (Fig. 3), where PKD infection risk rate increased by an approximate 54.8% with the increase of temperature per degree (at 23.8, 93.6% confidence limits (Fig. 4).

DISCUSSION

Literature survey indicates that PKD is a seasonal problem; which is associated with water quality, temperature and time of the year. Affected fish by PKD may show (symptoms) dark colour, abdominal swelling, abnormal behavior anaemia and gill pale in colour reviewed by Clifton-Hadley *et al.* (1984). During the investigation above symptoms were also observed in number of PKD infected fish. Particularly small fish having heavy infection showed swelling of the abdomen and were also in dark colour, effectively starving with low body fat (deposited in the caecal region), watery fluid in the intestinal contents, gills in pale colour and there was significant difference between the mean weight and length of PKD infected and uninfected fish. However fish were also heavily infected with *Trichodina* and *Gyrodactylus*. Therefore fish morbidity, mortality, symptoms and difference between weight and length may be due to combined factors i.e PKD and stressing factors (i.e parasitic infection low water quality and high temperature) as suggested by Seagrave *et al.* (1981).

Roberts (1978) noted the outbreaks of PKD at low pH values, whereas Scott (1979) described PKD outbreaks where the water supply was from a chalk stream, being alkaline (pH 7.9 to 8.3) CaCO₃ about 230 ppm and high eutrophic. Whereas at Romsey fish farm PKD outbreak occurred where the river water supply was from a mixture of source with a pH ranging from 6. to 7.6. On three different occasion water supply remain poor (i.e low oxygen, suspended particles) all at high temperatures (Pers. Com. Manager). At Romsey fish farm PKD showed

significant relationship with both time and temperature. In this study Malachite green and Oxolinic acid were used three and six times respectively. Therefore, decline in PKD prevalence and fish mortality may have been due to decrease in temperature, treatment, time or development of an immune response, needed more investigation.

REFERENCES

- Arther, J.R. and J. Lom, 1985. *Spphaerospora araii* n.sp. (Myxosporidia; Sphaerosporidae) from the kidney of a longnose skate *Raja rhina* (Jordan and Gilbert) from the pacific ocean off Canada. Can. J. Zool., 63: 2902-2906.
- Clifton-Hadley, R.S., D. Bucke and R.H. Richards, 1984. Proliferative kidney disease of salmonid fish; a review. J. Fish Dis., 7: 363-377.
- Clifton-Hadley, R.S.D. Bucke and R.H. Richards, 1987. A study of the sequential clinical and pathological changes during proliferative kidney disease in rainbow trout, *Salmo gairdneri* (Richardson). J. Fish Dis., 10: 335-352.
- Lester, R.J.G., 1974. Parasites of *Gasterosteus aculeatus* near Vancouver, British Columbia. Syesis., 7: 195-200.
- Naich, M. and F.M. Bilqees, 1991. Outbreaks of the Gyrodactyliasis at a commercial fish farm on the river Test, Hampshire, U.K. Proce. Parasit., 12: 46-59.
- Roberts, R.J. and C.J. Shepherd, 1974. Hand book of trout and salmon disease. Fishing News (Book) Ltd. Surrey.
- Roberts, R.J., 1978. The Pathophysiology and systemic pathology of teleosts. In "Fish pathology" Ed. Roberts. R.J. Balliere. Tindall, London, pp: 55-91.
- Seagrave, C.P., D. Bucke, E.B. Hudson and McGregor, 1981. A survey of the prevalence and distribution of proliferative kidney disease (PKD) in England and Wales. J. Fish Dis., 4: 437-439.
- Scott, P.W., 1979. The occurrence of proliferative kidney disease on a chalk stream. Veterinary Record, 105: 330-331.
- Smith, C.E., J.K. Morrison, H.W. Ramsey and H.W. Ferguson, 1984. Proliferative kidney disease: First reported outbreak North America. J. Fish Dis., 7: 207-216.