RECENT ADVANCES IN STUDIES ON ACUTE DISEASES OF FISHES

—A REVIEW

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INDIA
Fish disease and its control, pond hygiene and prophylactic measures are some of the aspects of vital importance in fish husbandry. A correct understanding of the problem of fish diseases and their control has become all the more important as we embark upon intensive fish culture in traditional systems as well as in some specialized systems like cage-culture, sewage-fed fish culture, etc. It is well known that fish health is inextricably associated with environment, and environmental stress in many cases has rendered the fish vulnerable to fish diseases. These aspects as well as bacteria, virus and parasites-caused disease of fishes are examined in this review in some detail for the benefit of research workers. It is also hoped that the manual would be of use to fish farm scientists, managers and entrepreneurs interested in culture and maintenance of healthy fish stocks.

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CHAPTER I

Introduction

Captured fishes from natural resources seldom exhibit any disease manifestation, apart in exceptional cases. They may often harbour a few parasites but such an infection less frequently causes much harmful effects on their hosts. As such, captured fishes from natural waters generally look healthy. But when fishes are cultured in smaller bodies of water, they often get diseased for which various reasons can be assigned. It may be mentioned here that terrestrial animals live in a better condition than fresh water fishes, which have to adjust with the constant changes in their environment. Such changes are often caused by temperature, light, dissolved gases, water chemistry, pollution, micro and macro-organisms, etc. In smaller bodies of water, fishes also undergo other changes due to population density, diet composition, excess of nitrogenous and other metabolites, stocking, hauling, handling and disease treatments. As a result, the stocking material suffer from non-maintenance of homeostasis (Wedemeyer, Meyer and Smith, 1976).

Homeostasis

In the last century Claude Bernard first coined this term—“homeostasis”, which meant maintenance of physiological condition within narrow limits of an organism (Nelson, Robinson and Boolootain, 1967).

Fish being a poikilothermal animal has to acclimate much with its environment, which is very much dynamic. As such, the external environment of a fish has a great influence on its internal environment. Thus, there is always a possibility of causing “stress” on the maintenance of homeostasis of a fish by its external environment; such stress if continued for long the fish may pose disease problems.

Stress

Originally “stress” was defined by Seyle (1950) as “The sum of the physiological response by which an animal tries to maintain or reestablish a normal metabolism in the face of a physical or chemical force”. Brett (1958) correlated “stress” with the fish
disease situation, i.e. when the normal functioning is significantly reduced and finally may manifest in death.

Primary effects of stress on fishes

Carps suffering from primary stress exhibit increased level of circulating corticosteroids (Chavin and Singley, 1972; Redgate, 1974; Spieler, 1974; Fryer, 1975 and Singley and Chavin, 1975). Teleosts suffering from depletion of oxygen show increases in their plasma as the primary effects of stress (Nanko and Tomilson, 1967; Mezeaud, 1971).

Secondary effects of a stress on fishes

Secondary effects of stress are manifested in many physiological changes, some of them are mentioned below (Mezeaud, Mozeaud and Donaldson, 1977):
- Increased blood pressure and heart rate;
- Increased level of blood sugar and lactate;
- Increased in the number of thrombocytes;
- Increased level of liver glycogen;
- Decreased in the number of white blood corpuscles;
- Decreased level of serum protein and blood chloride;
- Decreased inflammatory response; and
- Immuno suppression and changes in mucus production.

According to Seyle (1973) a series of morphological, biochemical and physiological changes are manifested in higher vertebrates when they are subjected to stress. Such changes are collectively called as general adaptive syndrome (G.A.S.).

General adaptive syndrome

GAS can be defined as the cumulative effects of environmental stress on higher vertebrates. The changes occurring in a stressed animal have the following three phases:

1. The alarm reaction;
2. The stage of resistance when the animal tries to adapt with its environment, the maintenance of its homeostasis; and

3. The stage of exhaustion when adaptation has failed and the animal has lost homeostasis. In this connexion it can be mentioned that homeostasis of an animal is controlled by its endocrine and nervous systems. Seyle (1950) has differentiated the functions of endocrine and nervous systems of a stressed animal as follows:

- ACTH (Adrenocorticotropic hormone) is released from pituitary after receiving the neurotropic impulse through the hypothalamus. ACTH stimulatory hormones are cortisone, corticosterone, and epinephrine; and
- Sympathetic nervous system is hypertensioned. As a result, (a) spleen contracts and additional erythrocytes enter the circulatory system, and (b) both respiration rate and cardiac output increase, and systolic blood pressure also rises.

ACTH release, controlled by hypothalamus in teleost, has also been recognised by Hill and Henderson (1968). Many workers have also demonstrated the secretion of stress hormones in salmonids (Black et al., 1961; Hans et al., 1966; Donaldson and Mc Bride, 1967; Fagerlund, 1967; Nakano and Tomilson, 1967; Hill and Fromm, 1968; Wedemeyer, 1969).

Clinical methods for the assessment of the effects of environmental stress on fish health have been documented by Wedemeyer and Yasutake (1977).

From the above statement it is evident that for disease manifestation of cultured fishes, an interrelation among host, pathogen and their environment is always in existence.

**Relationship among host, pathogen and their environment**

In a body of water fishes and their pathogens may be encountered without any disease manifestation of the former in general. This triad relationship was first considered by Meyer (1970) and Wedemeyer (1970), who postulated that stress is a very important factor in the outbreaks of infectious disease of fishes. Or, in other words, actually disease manifestation in fishes is a complex process where so many factors viz., environmental conditions, host susceptibility, virulence of pathogen, are involved. As such, it
can never be considered as a simple interaction between the host and the parasite. Sometimes a fourth factory may also take part to cause the disease of a fish; carp hemorrhagic septicemia may be taken for such an example; poor sanitary condition of water tells upon fish health and causes host’s susceptibility for infection vis-a-vis helps in rapid multiplication of the pathogen *Aeromonas liquefaciens*. Infection of *A. liquefaciens* is supposed to be a pre-disposing factor for the chronic viral infection (VHS) of the carp (Wedemeyer and Wood, 1974). For disease manifestation in fishes another aspect is also to be considered and that is Pathogenesis.

**Pathogenesis**

The genesis involved in “Pathos” (disease manifestation) of an organism can be termed as pathogenesis. A fish may suffer from disease due to either change in its environmental condition or infection of a pathogen. A generalised information has already been given as to how environmental stress acts upon disease manifestation. Pathogenesis due to viruses, bacteria, fungus, and invertebrates infections needs further clarification.

A successful pathogen, may potentially be infectious, must first find a susceptible host for its lodging and multiplication. This is not easy because the body of fish is covered with scales and there are indefinite number of epidermal cells which secrete mucus to get rid of a pathogen trying to get lodged on a susceptible fish. Water taken in through mouth is also thrown out by means of gills and opercular space. It is, thus, difficult for a fish pathogen to find its entry into its host. If it finds a way through the mouth, the pathogen is to survive in the acid and alkaline media of the alimentary tract. To tolerate the same is not easy for every pathogen. An easier way by which a pathogen gets entry in its host is a lesion on the skin or through other openings like mouth, eye, nostril, etc. As soon as the pathogen enters the circulatory system of its host, the former meets with various defence mechanism of the latter. Inflammation, Immune responses, etc., are such mechanisms (Anderson, 1974; Ritzkers 1980). However, getting an entry the pathogen must establish itself on the host so that it can multiply. A successful pathogen keeps its host alive for a longer period and thus causes a choronic disease of its host. But virulent pathogens cause acute disease to their hosts when the latter die within a short period.

**Chronic and acute diseases**

Chronic diseases of fishes are caused generally by less virulent bacteria, fungus,
protozoans, trematodes, cestodes, nematodes, annelids and crustaceans. Due to chronic disease mortalities within a fish population occur for a longer period when hardly 50% of the stocking material is lost altogether. But due to an acute disease the entire population is lost within a limited period. Due to oxygen depletion or pollution of lethal toxicants the entire fish stock may succumb within few hours. Virulent viruses or bacteria may cause acute disease of fishes too when the entire fish stock is lost within a period of 5-6 days. A lot of literature is already available on the chronic diseases of fishes and their remedial measures. As such, the present communique is restricted to the diseases of fishes due to environmental stress, viral, and bacterial infections. Attempts have also been made to supply information on preventive and remedial measures taken against such infectious diseases which generally break out in epidemic proportions. It will not be out of place to mention that purpose of the present communique is just to make the reader conscious about the comment of Dr. Sniessko (1972) — "... ... the most voluminous is the literature on fish parasites ... ... altogether 71% dealt with descriptions of parasites. Of the remaining 29%, 16% had the word ‘disease’ in the title, 7% were on ecology etc., 6% on control methods and 1.5% on Immunity". This is in sharp contrast to reports on diseases of fish caused by bacteria and virus where the study of the actiological agents received much less attention”.

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CHAPTER II

Environmental stress and diseases of fishes

In the first chapter homeostasis and stress have been defined. The relationship among host (fish) pathogen and environmental condition has also been identified. However, the present chapter is to elaborate this relationship for a better understanding, because this is an important aspect of fish culture and the basis for making the enterprise a profitable one. Before the same is done, the physiology of freshwater fishes needs to be discussed along with their environmental condition. It is well known that every fish prefers an optimal condition for its growth and reproduction. Any alteration in the environmental condition causes 'stress' on the fish. If such an alteration increases arithmetically, the stress caused by the same on a community of fish will manifest in geometrical proportion which may reflect in the mortality of the entire community. As such, certain aspects of fish physiology in relation to environmental condition will be highlighted.

Fishes are poikilothermal animals and most of the freshwater fishes are amnolytic in habit. Accordingly, Na\(^+\) and Cl\(^-\) ions are taken in by a freshwater fish and NH\(_4\)\(^+\) and HCO\(_3\)\(^-\) are expelled out to maintain the ionic balance. Two enzymes viz., deamination and carbonic anhydrase present in the gill epithelia take active part in maintenance of the ionic balance. Blood flow in gills is also regulated by hormones viz., adrenaline and acetylcholine (Steen and Kruysse, 1964). Acetylcholine regulates blood flow when oxygen demand for metabolic purposes is low. When fishes are stressed and demand for oxygen in more adrenaline (epinephrine) helps in increased blood flow through the thin gill lamellae for facilitating maximum gas exchange rather oxygen uptake. For freshwater fishes the environment is hypo-osmotic; as such, the kidneys of freshwater teleosts play a vital role in osmoregulation. In fact, the large volume of water that enters the fish body through diffusion (gill and permeable surface of pharynx) is excreted out through the kidneys. As a result the kidney faces the problem of maintaining electrolyte balance, because blood plasma has the same concentration of electrolytes as the blood; as such urine formation begins with ultrafiltration of blood plasma. Had the filtration not been done, a lot of salts would have been lost and the fish would have toiled hard to replace those electrolytes. In fact, salts are mostly reabsorbed in kidney tubules and the urine of a freshwater fish contains much less amount of salt than its external environment or ambient water (Wedemeyer, Meyer, and Smith, 1976). Hickman and Trump (1969) have mentioned that certain freshwater fishes have no glomerulue.
The complex and specific mechanisms of ionic exchange and osmoregulation of fishes have been well dealt by Maetz (1974). However, the significant amount of ions that are lost through the huge volume of urine of freshwater fishes are replaced by diffusion of Na\(^+\) and Cl\(^-\) through the gills as well as by absorption of food through the gut to maintain the equilibrium.

It has already been made clear that every fish prefers an optional environmental condition for its maintenance of homeostasis, growth and reproduction. Combination of several parameters viz., temperature, light, dissolved gases, physico-chemical properties, are reflected in the environmental condition and an alteration beyond acceptable limit of any of these parameters will cause stress to the fish. Productivity of the ecosystem, which supply food to the fish, is also to be considered for the growth and maturation of the fish. In an extensive culture system productivity is of great importance (Macan, 1969; Odum, 1971). However, the present chapter will be restricted to other parameters of fish environment. They are discussed below:

1. **Temperature**: Fish prefers an optimal temperature for its growth and maturation. Immune response of a fish is also dependent on temperature. Warm water mirror carps do not produce antibodies when ambient temperature is less than 12\(^\circ\) C but cold water trouts produce antibodies when water temperature is even lower than 5\(^\circ\) C. Roberts (1975) and Anderson and Roberts (1975) have shown that both defence mechanism and susceptibility to disease of a fish are dependent on temperature. With slightly higher than optimal temperature the wound healing of a fish is quicker. But higher temperature beyond limits would cause exhaustion for the fish. Solubility of oxygen in water is also dependent on temperature. Higher the temperature lesser is the solubility of dissolved gases. But reverse is true for the pollutants (heavy metal, pesticides and crude oils).

2. **Light**: The growth and maturation rates of fishes are also controlled by light or photoperiod. In other words, light has a primary role in food production in extensive fish culture. Excess of light stops photosynthetic action and may cause sun-burns of the fishes (Roberts, 1978).

3. **Dissolved gases**: Two dissolved gases viz., oxygen and nitrogen play vital role in fish life. Though concentration of oxygen in air is 260 ppm the same is quite scarce in water (0-14 ppm). Further solubility of gases in water is inversely proportional with both temperature and salinity. Though oxygen is very much needed for respiration but nitrogen is biologically inert. However, supersaturation of both these gases in water may cause
gas embolism (Rucker, 1972) for the fishes. On the contrary depletion of oxygen in water will result in asphyxia of the fishes. As oxygen is less soluble in blood plasma hemoglobin binds the same for active transport. Unloading of oxygen is accelerated by CO₂ released from tissue respiration. However, this loading and unloading of oxygen and the equilibrium maintained between O₂ and CO₂ are largely dependent on the pH of ambient water. In acid water carrying capacity of hemoglobin is reduced. Oxygen is more needed for fish during its specific dynamic action i.e., food intake and digestion. As such, the minimum amount of dissolved oxygen required for the good growth of fish is 5 mg/l.

Though carbon dioxide is most soluble in water yet its minute presence in water is due to less availability in air (0.04%). For healthy growth of fish 3 mg/l or less of free CO₂ is permissible in pond or hatchery waters. This high amount of free CO₂ present in water is due to chemical or biological activity. The effects of dissolved and free carbon dioxide have been reviewed by Doudoroff and Katz (1950). However, the only record of gas embolism due to CO₂ has been made by Mrsic (1933).

4. pH, ammonia, and bicarbonates: Best growth of fish is expected in water having a pH range between 6.7 and 8.6. But the trace elements mostly remain in available form in neutral water. It is known that NH₄⁺ is harmless to fish but NH₃ is toxic. In water NH₃ combines with H₂O to form NH₄OH which readily dissociates to NH₄⁺ and OH⁻. But these combination and dissociation are dependent on temperature and pH. In high pH toxic NH₃ is formed. According to Trussell (1972) the total ammonia (free NH₃) in water should not exceed to 0.02 ppm for healthy growth of fish.

Similarly bicarbonate content of water is also dependent upon pH and temperature. In high pH HCO₃⁻ quickly dissociates to H⁺ + CO₃⁻. In low pH H⁺ combines with CO₃⁻ to from H₂CO₃. In medium to low pH H₂CO₃ can dissociate to form CO₂ and H₂O. However, according to Hart et al. (1945) for good growth of fish the ambient water should not have more than 180 mg/l of bicarbonates. The permissible limit of pH is 6.0—9.0, but in acid waters CO₂ attains toxic limit to cause fish mortality.

5. Acidity: Neess (1949) encountered hypersensitivity to bacteria of carps in acid waters (pH below 5.5). But, according to EIFAC (1968) only few experiments have been conducted to prove that fishes cultured in low pH of ambient waters are more susceptible to diseases. However, other effects of acidic waters have already been discussed.
The effects on environmental stress on the outbreaks of infectious diseases of fishes have already been discussed by Snieszko (1974) and Stankiewiez (1979). Nutritional diseases of fishes are generally encountered in intensive fish culture where wrong formulations or deficiency of vitamins are the other factors for disease manifestations (Snieszko, 1972; Roberts, 1978). As such the following table indicate the stress mediated disease of fishes encountered in hatcheries, raceways, and impoundments:

<table>
<thead>
<tr>
<th>Sl. No.</th>
<th>Diseases</th>
<th>Factors associated with the diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Furunculosis</td>
<td>Crowding, handling and low level of D. O. in water</td>
</tr>
<tr>
<td>2</td>
<td>Bacterial gill disease</td>
<td>Crowding, low level of D. O. and higher concentration of ammonia in water</td>
</tr>
<tr>
<td>3</td>
<td>Corynbacterial kidney disease</td>
<td>Diets containing corn-gluten and low total hardness (less than 100 ppm as CaCO₃) of water</td>
</tr>
<tr>
<td>4</td>
<td>Columnaris</td>
<td>Crowding, handling and higher water temperature</td>
</tr>
<tr>
<td>5</td>
<td>Aromonas &amp; Pseudomonas hemorrhagic septicemia</td>
<td>Low D. O. in water, handling, crowding, prior sufferings from infection of Costia or Trichodina; non-sanitary condition of ambient water together with more bacterial load; Chronic exposure to pesticides</td>
</tr>
<tr>
<td>6</td>
<td>Coldwater</td>
<td>Low temperature of ambient water</td>
</tr>
<tr>
<td>7</td>
<td>Spring viremia of carps</td>
<td>Bad handling associated with low temperature</td>
</tr>
<tr>
<td>8</td>
<td>Nephrocalcinosia</td>
<td>Overstocking</td>
</tr>
<tr>
<td>9</td>
<td>Gill necrosis</td>
<td>Over drugging with formalin</td>
</tr>
<tr>
<td>10</td>
<td>Cataract of Catla catla</td>
<td>Poor sanitary condition of ambient water</td>
</tr>
<tr>
<td>11</td>
<td>Dropsy of carps and catfishes</td>
<td>Poor sanitary condition of ambient water, over stockocking and chronic exposure to low level of D. O,</td>
</tr>
<tr>
<td>12</td>
<td>Tumours of Anabas testudineus</td>
<td>Same as above</td>
</tr>
<tr>
<td>13</td>
<td>Reddish blotches of Hypophthalmichthys molitrix</td>
<td>Same as above</td>
</tr>
<tr>
<td>14</td>
<td>Infectious hemato poe tic necrosis</td>
<td>Low temperature of ambient water</td>
</tr>
<tr>
<td>15</td>
<td>Vibriosis</td>
<td>Bad handling, low level of D. O. in ambient water</td>
</tr>
<tr>
<td>Sl. No.</td>
<td>Diseases</td>
<td>Factors associated with the diseases</td>
</tr>
<tr>
<td>--------</td>
<td>-----------------------------------</td>
<td>------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>16</td>
<td>Tail and fin rot</td>
<td>Crowding, improper diet, temperature and chronic exposure to PCBS</td>
</tr>
<tr>
<td>17</td>
<td>Blue-sac</td>
<td>Crowding; accumulation of nitrogenous metabolites in ambient water</td>
</tr>
<tr>
<td>18</td>
<td>Gas-embolism</td>
<td>Supersaturation of O₂, N₂ and CO₂ in water</td>
</tr>
<tr>
<td>19</td>
<td>Trichodinosis</td>
<td>Crowding, low level of D. O. in water</td>
</tr>
<tr>
<td>20</td>
<td>Coagulated yolk</td>
<td>Rough handling, mineral loss in ambient water</td>
</tr>
<tr>
<td>21</td>
<td>Handling loss</td>
<td>Repeated netting-cum-rough handling</td>
</tr>
<tr>
<td>22</td>
<td>Fatty infiltration of liver</td>
<td>Deficiency of essential fatty acids in diet</td>
</tr>
<tr>
<td>23</td>
<td>Improper growth</td>
<td>Deficiency in essential amino acids in diet, poor sanitary condition, etc.</td>
</tr>
<tr>
<td>24</td>
<td>Exophthalmia</td>
<td>Deficiency of vitamin E in the diet</td>
</tr>
<tr>
<td>25</td>
<td>Anaemia and cloudy lens</td>
<td>Deficiency of riboflavin in the diet</td>
</tr>
<tr>
<td>26</td>
<td>Scoliosis and lordosis</td>
<td>Deficiency of ascorbic acid in the diet</td>
</tr>
</tbody>
</table>

The above table is compiled from Gopalakrishnan (1961 a and b); Snieszko (1972, 1973 & 1974); Wedemeyer, Meyer and Smith (1976); Chun (1976); Pal (1976); Pal & Tripathi (1978) and Schlotfeldt (1980).

**Other parameters**

Apart from the above mentioned diseases, mortalities of fishes due to algal toxicosis have also been reported by Matida et al. (1967); Sarig (1971); etc. Sewage has also been reported as a stress factor by several workers Collins, 1970; Heuschmann-Bruner, 1970 and Shotts et al. 1972).

Burrows (1964), Burrows and Combs (1968), Larmoyenex and Piper (1973), Meyer and Bullock (1973), etc. have reported about the stress caused by the fish metabolites.

The effects of industrial pollution in outbreaks of infectious diseases of fishes have been reported by Snieszko et al. (1964); Simidu and Egushi (1972); Kusuda and Miura (1972); Kusuda and Yamaska (1972); Burton et al. (1972); Starr and Jones
(1957); Skidmore (1970); Pippy and Hare (1969); Perkins et al. (1972); Van Valin et al. (1968). Similarly effects of pesticides on the outbreaks of infectious diseases of fishes have also been reported by Couch (1974), Mohoney et al. (1973) and Collins (1970).

Before closing this chapter it is made clear that “Disease is the end result of an interaction between a noxious stimulus and a biological system and to understand disease is to understand all aspects of the biology of the species”—Mawdesley Thomas (1972).

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<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Source</th>
<th>Family</th>
<th>Genus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ichthyobodo paraensei</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
</tr>
<tr>
<td>Ichthyophonus elongatus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
</tr>
<tr>
<td>Ichthyophonus dohrni</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
</tr>
<tr>
<td>Ichthyophonus tubuliferus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
</tr>
<tr>
<td>Ichthyophonus mytilus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
<td>Ichthyophonus</td>
</tr>
</tbody>
</table>
**CHAPTER III**

**Infectious bacterial diseases of fishes**

Bacterial diseases of fishes are often recorded from natural resources as well as man-made impoundments particularly from waters polluted with organic load. Bacterial fish pathogens recorded from the teleosts are tabulated below:

<table>
<thead>
<tr>
<th>Family</th>
<th>Genus</th>
<th>Species</th>
<th>Habitat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cytophagaceae</td>
<td>Flexibacter</td>
<td><em>F. columnaris</em> (Bergey, 1974)</td>
<td>Mucus of fish</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>F. psychrophila</em> (Davis, 1946)</td>
<td>Fish body</td>
</tr>
<tr>
<td>Pseudomonadaceae</td>
<td><em>Pseudomonas</em></td>
<td><em>P. fluorescens</em> (Plehn, 1924)</td>
<td>Ambient soil &amp; water; decaying fish</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-oxidizing Pseudomonads (Park, 1962)</td>
<td></td>
</tr>
<tr>
<td>Enterobacteriaceae</td>
<td>Edwardsiella</td>
<td><em>E. tarda</em> (Meyer &amp; Bullock, 1973)</td>
<td>Polluted waters; Human excreta</td>
</tr>
<tr>
<td></td>
<td>Unclassified</td>
<td>Unclassified (Bullock <em>et al.</em>, 1971)</td>
<td>Unknown</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Red mouth disease (Ross <em>et al.</em>, 1966)</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>Vibrio</em></td>
<td><em>V. anguillarum</em> (Cannestrini, 1893)</td>
<td>Marine benthos &amp; invertebrates</td>
</tr>
<tr>
<td></td>
<td><em>Aeromonas</em></td>
<td><em>A. salmonicida</em> (Emerich &amp; Weibel, 1894)</td>
<td></td>
</tr>
</tbody>
</table>
## TABLE: BACTERIAL PATHOGENS OF FISHES

<table>
<thead>
<tr>
<th>Family</th>
<th>Genus</th>
<th>Species</th>
<th>Habitat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncertain</td>
<td>Flavobacterium</td>
<td>A. hydrophila</td>
<td>Freshwater fish with organic load</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Popoff &amp; Veron, 1976)</td>
<td></td>
</tr>
<tr>
<td>Streptococcaceae</td>
<td>Streptococcus</td>
<td>Str. faecalis</td>
<td>Faecal matter of warm-blooded animals</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Hashina, et al., 1958)</td>
<td></td>
</tr>
<tr>
<td>Bacillaceae</td>
<td>Clostridium</td>
<td>Cl. betulinum</td>
<td>Soil, faeces, decaying organic matter</td>
</tr>
<tr>
<td>Corynbacteriaceae</td>
<td>Corynbacterium</td>
<td>Gr+, non-sporing rods (Bolding &amp; Merrill, 1935)</td>
<td>Unknown</td>
</tr>
<tr>
<td>Mycobacteriaceae</td>
<td>Mycobacterium</td>
<td>Myco. marinum</td>
<td>Unknown</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Bataillon &amp; Terre, 1897)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Myco. fortuitum, (Ross &amp; Broncate, 1959 ; Gordon &amp; Smith, 1955)</td>
<td>Unknown</td>
</tr>
</tbody>
</table>
For the presumptive identification of bacteria pathogenic to fishes, Bullock (1971) has provided a schematic outline. However, the following scheme prepared from Bullock (1961) and Shotts and Bullock (1975) and Shotts (1976) will be useful.

- **Nocardiaceae**
  - **Nocardia**
    - *N. asteroides* (Valdez & Conrey, 1963) Unknown
    - *N. kampachi* (Kariya *et al.*, 1968) Unknown
  - Epitheliocystis organisms (Hoffman *et al.*, 1969) Unknown

- **Chlamydiaceae**
### Bacteria (Primary Isolation on TSA Plates & Slants)

<table>
<thead>
<tr>
<th>Gr+ rods</th>
<th>Gr- rods</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Numerous short Diplobacilli, isolated mainly from kidney</strong></td>
<td><strong>Fairly long thin acid-fast rods</strong></td>
</tr>
<tr>
<td><em>Corynebacterium</em></td>
<td><em>Myxobacterium</em></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Non-motile</th>
<th>Motile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short rods</td>
<td>Long thin rods</td>
</tr>
</tbody>
</table>

#### Columnaris disease
- *Chondrococcus columnaris*

#### Bacterial Gill disease
- *Myxobacterium*

### Gr- Short Rods on TSI (Triple-Sugar Iron Agar)

<table>
<thead>
<tr>
<th>Acid / Acid &amp; gas</th>
<th>Acid / Acid gas &amp; H₂S</th>
<th>Alkali / Acid</th>
<th>Alkali / No change</th>
<th>Alkali / Acid-gas</th>
<th>Alkali / Acid gas &amp; H₂S</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>(2)</td>
<td>(3)</td>
<td>(4); (5)</td>
<td>(6)</td>
<td>(7)</td>
</tr>
</tbody>
</table>

**Oxidase Test**
- \(+\)

**Indole Test**
- \(-\)

**Citrate Test**
- \(+\)

**Motility Testes**
- \(+\)

**Testes**

(2) Acid/Acid: May be treated as a special case; Gram staining may be repeated
(3) Acid/Acid gas H₂S
Phenyl-alanine Test

Proteus
Indole +
& citrate test

Escherichia
Citrobacter

(4) Alkaline/Acid
Oxidase Test
Citrate Test
Malonate Test

(4) Alkaline/Acid
Oxidase Test

Lysine Test
Lysine Test

Shigella
Aeromonas

Shigella
Y. ruckerii
Pseudomonas
Aeromonas

(5) Alkaline/No reaction
Cotrimido Test

(5) Alkaline/No reaction
Cotrimido Test

Pseudomonads
Flourescent test for P. fluorescent

Consider as a special case

(5) Alkaline/No reaction
Cotrimido Test

Pseudomonads
Flourescent test for P. fluorescent

Consider as a special case

(6) Alkaline/Acid gas
Phenyl-alanine Test

Proteus
Oxidase Test

Indole Test
Aeromonas

V-P Test
Escherichia

Salmonella
Enterobacter

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Alkaline/Acid gas & $H_2S$

- Phenyl-alanine test
  - + Proteus Oxidase Test
  - - ONPG Test
    - + Aeromonas
      - + Citrobacter
      - - Motility & citrate Test
        - + Edwardsiella
          - + A. hydrophila
            - + Arizona
            - - Salmonella
          - - A. salmonicida
        - - Malonate Test
    - - Indole Test
      - + Citrobacter
      - - Motility & citrate Test
Temperature for growth

*Aeromonas salmonicida* is a gram negative nonmotile rod and prefers a temperature between 20 and 25°C, whereas *A. hydrophila* is motile and prefers 30°C for growth. *Pseudomonas fluorescens* is usually grown at 20—22°C but *Vibrio anguillarum* prefers a temperature between 25 and 30°C. Both *A. hydrophila* and *Y. ruckerii* can even be grown at 37°C, as both of them are heat labile.

*Chondrococcus columnaris* and *Cytophaga psychrophila* are long thin rods and the former prefers 14°C for growth, whereas the growth of the latter occurs at 12°C.

Clinical pathology

1. *F. columnaris*: Raised whitish plaques on head, back and gills. Lesions (may be yellow/orange) on gills are often necrotic.
2. *P. psychrophila*: Ulcerative necrosis of skin and epithelial hyperplasia.
3. *P. fluorescens*: Haemorrhagic septicaemia; Cyprinids show ascites.
4. *E. tarda*: Small cutaneous lesions; peritonitis and necrosis of hepatic and renal tissue.
5. E. R. M. bacterium: Erosion of the lower jaw, necrosis of intestinal mucosa, deep seated haemorrhage of the tissues of head.
9. Flavobacterium: Haemorrhagic septicaemia or chronic granulomatous disease.
10. *H. piscicium*: Small white epidermal spongiotic hyperplasia; followed by an ulcer with a white rim.
11. *Pasteurella*: Acute haemorrhagic septicaemia; lesions on haematopoietic tissue.
12 Streptococcus faecalis : General septicaemia.
13 Clostridium betulinum : Nervous imbalance.
14 KD corynbacterium : Darkening; Exophthalmos and small haemorrhages.
15 M. marinum : Darkening and swelling of abdomen.
16 M. fortuitum : Darkening and hypersensitivity.
17 Nocardia : Anorexia, emaciation, and distension of the mouth.

Control measures

Control measures generally adopted against bacterial diseases of fishes are sanitation and chemotherapy (Shotts & Snieszko, 1976). Sulfonamides (Snieszko et al., 1950), Oxytetracyclines (Snieszko et al., 1952) and Nitrofurans (Post, 1959) are the drugs commonly used for disease treatments. Immunization and genetic selection are also practised against infection of Vibrio anguillarum (Rohovec, et. al., 1975, Gjodram and Aulestad, 1974). No treatment method is available against the infections of Flavobacterium, Streptococcus Lancefield group B, Mycobacterium spp. and Nocardia.

Before closing this chapter it is suggested that one should bear in mind that fishes are poikilothermal animals, and the bacteria isolated from fishes as such should be grown according to their ambient temperature. Often a common mistake is made by incubating the bacteria at 37°C which is the average human body temperature. At this temperature most of the fish pathogenic or symbiotic bacteria cannot be isolated because the difference of temperature between ±5°C (compared to ambient) may be lethal for the bacteria desirable for isolation. Further, even recent edition of “Bargey’s Manual” is not a full proof book for identifying ‘Fish Bacteria’, as the same incorporates mostly the bacteria isolated from warm blooded animals (perhaps the only exception is Aeromonas salmonicida). Recent advancement in sero-dignosis of fish diseases is presented in the last chapter for use.

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CHAPTER IV
Infectious viral diseases of fishes

A comprehensive account of fish diseases of viral origin was first made available by Jensen (1978), but a more comprehensive and systematic account of fish viruses has been recently presented by Mc Allister (1979). It is needless to mention that fish virology is a most modern subject and with the advent of fish cell-lines and tissue culture in the latter half of the present century a great advancement has been made. However, more studies are yet to be made as studies on this branch of science are mostly confined in countries like U. K., U. S. A. and Japan.

All fish viruses have not been studied thoroughly; as such some are well characterized while others need isolation. However, fish viruses can be tentatively classified as follows:

1. **Herpesviridae**
   
   - Channel catfish virus
   - *Herpesvirus salmonis*
   - Nerka virus

   **Herpesvirus like agents**
   
   - Epithelioma papillosum of cyprinus
   - *Herpesvirus scophthalmi*

2. **Reoviridae**
   
   - Infectious pancreatic necrosis virus
   - Eel virus european
   - Golden shiner virus

   **Isolated in cell-culture**

   - Wolf and Darlington, 1971; Fijan, 1968
   - Wolf, et al., 1975a, b;
   - Wolf & Taylor, 1975
   - Sano, 1976
   - Bauer et al., 1969
   - Buchanan & Medley, 1978
   - Wolf ei. al., 1960
   - Mitchell & Plumb, 1980
3. *Iridoviridae*

Isolated in cell-culture

- a Lymphocystis virus
- b Piscine erythrocytic necrosis virus

4. *Rhabdoviridae*

Isolated in cell-culture

- a Infectious haematopoetic necrosis virus
- b Egtved virus
- c Rhabdovirus carpio
- d Pike-fry rhabdovirus
- e Eel virus-american
- f Rhabdovirus of European eel

5. *Retroviridae*

- a Atlantic salmon fibrosarcoma
- b Esox lymphosarcoma
- c Esox epidermal hyperplasia
- d Walleye dermal sarcoma
- e Walleye epidermal hyperplasia
- f White sucker epidermal papilloma

6. Unclassified fish viruses causing neoplasia

- a Stomatopapilloma of eel
- b Brown bullhead papilloma
- c Atlantic Salmon papilloma
- d Pleuronoctid epidermal papilloma

Dunbar & Wolf, 1966
Laird & Bulloek, 1969
Amend, *et al.*, 1999
Jensen, 1963
Fijan, *et al.*, 1971
deKinkelin, *et al.*, 1973
Mc Allister, *et al.*, 1977
Duncan, 1978
Papas, *et al.*, 1976;
Winqvist *et al.*, 1973
Winqvist *et al.*, 1968;
Sonstegard, 1976;
Walker, 1961, 1969
Walker, 1969,
Yamamoto, *et al.*, 1976
Sonstegard, 1977a, b
Mc Allister, *et al.*, 1977
Edwards, *et al.*, 1977
Carlisle, 1976, 1977;
Wiren, 1971
Brooks, *et al.*, 1969
7 Unclassified putative fish viruses

Isolated in cell-culture

a Bluegill virus  Hoffman, et al., 1969
b Grunt-fin agent  Clem, et al., 1965
c Ulcerative dermal necrosis virus  Roberts, 1972
d Gill necrosis of carp virus  Popkova & Shohel-kunov, 1978

8 Susceptible cell system

Infectious pancreatic necrosis virus
Eel virus european

a Rainbow trout

Herpesvirus salmonis
Eel virus american

Infectious haematopoetic necrosis virus
Egtrved virus
Stomatopapilloma of eel virus
Eel viruses of Japan

b Brown bullhead

Channel catfish virus

c Channel catfish ovary

Infectious pancreatic necrosis virus
Herpesvirus salmonis

Infectious haematopoetic necrosis virus

Golden shiner virus
Pike-fry rhabdovirus
Eel virus japan (I & II)
Rhabdovirus carpio
Stomatopapilloma of eel virus
f Bluegill fry Lymphocystis virus

g Other cell-lines (certified) with their abbreviations

<table>
<thead>
<tr>
<th>Cell-line/Species</th>
<th>Abbreviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bluegill</td>
<td>BGL</td>
</tr>
<tr>
<td>Channel catfish ovary</td>
<td>CCO</td>
</tr>
<tr>
<td>Coho salmon embryo</td>
<td>CSE</td>
</tr>
<tr>
<td>Common carp hyperplasia</td>
<td>EPC</td>
</tr>
<tr>
<td>Bluestripped grunt</td>
<td>GF</td>
</tr>
<tr>
<td>Large-mouth bass</td>
<td>LBF</td>
</tr>
<tr>
<td>Rainbow trout fry</td>
<td>RTF</td>
</tr>
<tr>
<td>Sockeye salmon</td>
<td>SSE</td>
</tr>
<tr>
<td>Steelhead trout embryo</td>
<td>STE-137</td>
</tr>
<tr>
<td>Red-sword tail</td>
<td>SWT</td>
</tr>
<tr>
<td>Carp sac fry</td>
<td>CSF</td>
</tr>
</tbody>
</table>

9 Optimum temperature for cell-culture and cytopathetic effect (site of replication)

<table>
<thead>
<tr>
<th>Virus Type</th>
<th>Temperature</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>a Infectious haematopoetic necrosis</td>
<td>12-18°C</td>
<td>Lytic</td>
</tr>
<tr>
<td>virus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pike-fry rhabdovirus</td>
<td>21-28°C</td>
<td></td>
</tr>
<tr>
<td>Egived virus</td>
<td>14-15°C</td>
<td></td>
</tr>
<tr>
<td>Rhabdovirus carpio</td>
<td>20-22°C</td>
<td></td>
</tr>
<tr>
<td>Eel virus american</td>
<td>20-27°C</td>
<td>(cytoplasm)</td>
</tr>
<tr>
<td>Rhabdovirus of European eel</td>
<td>15°C</td>
<td></td>
</tr>
<tr>
<td>b Channel catfish virus</td>
<td>25-30°C</td>
<td>Syncetium</td>
</tr>
<tr>
<td><em>Herpesvirus salmonis</em></td>
<td>4-10°C</td>
<td>Nucleus</td>
</tr>
<tr>
<td><em>Herpesvirus scophthalmi</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epithelioma papillom of cyprinus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nerka virus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>c Infectious pancreatic necrosis</td>
<td>15°C</td>
<td>Lytic</td>
</tr>
<tr>
<td>virus</td>
<td></td>
<td>Cyto-</td>
</tr>
<tr>
<td>Eel virus european</td>
<td>20°C</td>
<td>Pyknosis</td>
</tr>
<tr>
<td>Golden shiner virus</td>
<td>30°C</td>
<td>Syncetium</td>
</tr>
</tbody>
</table>
d Piscine erythrocytic necrosis virus
Lymphocystis virus 25°C Hypertrophy

e Eel virus (Stomatopapilloma) 16-20°C Lytic
f Eel virus japan (II) 20-25°C Syncetium (Cytoplasm)

Most of the above mentioned viral agents generally affect fry and fingerlings of fishes. Spring virus of carps (SVS) and fish pox virus (EV) infect even adults. Same is true for PENV (Piscine erythrocytic necrosis virus) as well.

10. Clinical signs

I RNA virus diseases

a IPNV :— Darker in colour, tail chasing, exophthalmia, distended abdomen ;
b VHS :— Darker in colour, haemorrhages at the base of fins and in gills, lethargy ;
c IHNV :— Lethargy-cum-sporadic hypersensitivity, darker in colour, exophthalmia, distended abdomen, long white faecal casts ;
d SVC :— Darker in colour, petechial haemorrhages, loss of balance, gathering near outflows, exophthalmia, dropsy ;
e PFR :— Loss of balance, swimming near the surface, haemorrhages of skin and gills, cranial distension and exophthalmia.

II DNA virus diseases

a CCV :— Loss of balance, hanging vertically in the water, abdominal distension and haemorrhages of skin and gills ;
b Herpesvirus salmonis :— Exophthalmia, darker in colour, distended abdomen and anaemia.
Other viruses generally cause papillomatosis. Carp pox lesions are raised white nodules. **PENV** infects both mature and immature erythrocytes resulting in anaemia of the diseased fish.

Control measures generally taken against viral diseases of fishes are avoidance of an infection and chemotherapy with synthetic polynucleotides (Roberts, 1978). Raising the temperature of ambient water controls IHNV infection (Amend, 1970). Immunization of the stocking material with sonicated antigens or avirulent strains (alive antigen) may also be used. Mass vaccination can be done by immersing the stocking material in hyperosmotic solutions containing the antigen (Amend and Fender, 1976). Recently, more efforts are being made to produce bacterins and vaccines on commercial scale to control infectious diseases of fishes (Fryer et al., 1977).

Before close it must also be mentioned that guidelines for virological examination of fishes have been suggested by Wolf (1970). Wolf and Quimby (1977 & 1978) have provided much information on—(a) primary monolayer culture of fish cells initiated from minced and trypsinized tissues, (b) procedures for subculturing fish cells and propagating fish cell-lines, and (c) systematic management of animal cell-lines.

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CHAPTER V

Serodiagnosis of fish diseases

Recent advent of serodiagnosis fish diseases has helped much for standardization of the diagnosis. Being more specific these biological tests are most subjective and certain Serotechniques generally used in the identification of fish diseases are:

a Precipitin test;
b Agglutination test;
c Immune diffusion test;
d Passive haemagglutination test;
e Fluorescent antibody test (FAT);
f Serum-viral neutralization test.

However, all these antibody-antigen reactions are subject to pH, temperature and time of incubation.

Serodiagnoses applied in diseases of fishes

A Infectious pancreatic necrosis virus (IPNV)

a New Zealand Rabbit—IPNV antiserum has no cross reaction with rabbit anti VHS (Jørgensen, 1969);
b Complement fixation test for IPNV (Finlay & Hill, 1975);
c Fluorescent antibody test (Tu et al., 1974);
d Cross reaction between Sp and Ab strains of IPNV in direct FAT (Jørgensen, 1972);
e Specific neutralizing antibody (Wolf & Quimby, 1969);
f Anti-IPNV antibodies are Ig M like immunoglobulins (Jørgensen, 1973);
g Plaque morphology in RTG2 cells (Wolf, 1973);
h Five strains belonging to forty-two IPNV isolants (Livents & Springer, 1973) and
i Detection of IPNV (McCarthy, 1975).
B  *Infectious haematopoetic necrosis virus (IHNV)*

a  No antigenic relationship between IPNV and Egtved virus (McAllister *et al.*, 1974a);
b  Low cross reaction among IHNV, PFR, and SVC (Hill *et al.*, 1975);
c  FAT and indirect fluorescent antibody staining (McAllister *et al.*, 1974b);
d  Identification of IHNV (Wolf *et al.*, 1973);
e  Haematological and blood chemical changes (Amend & Smith, 1975) and
f  Auto-interference of IHNV (McAllister & Pilcher, 1974).

C  *Channel catfish virus (CCV)*

a  Virus neutralizing activity (Plumb, 1973);
b  Quantifying CCV or antibody (Gratzek *et al.*, 1973);
c  Immune response and antibody characterization (Heartwell, 1975)—Acrylamide gel electrophoresis of serum and

D  *Viral haemorrhagic septicaemia virus (VHSV)*

a  Fluorescent antibody technique (FAT) to demonstrate antigens (Jørgensen and Meyling, 1972);
b  Specific neutralizing antibody (Jørgensen, 1972) and
c  No antigenic relationship with IHNV and IPNV (McAllister *et al.*, 1974a).

E  *Bluegill virus*

a  No cross reaction with viruses causing influenza, mumps, and lymphocytic chorion meningitis (Backwith, 1974).

F  *Swim-bladder infection of carp*

a  Cross reaction with Rhabdovirus carpio (Backman & Ahne, 1973).
**G  Aeromonas salmonicida**

a. Positive-agglutination with rabbit anti- *A. salmonicida* (Rabb et al., 1964);
b. Auto-agglutination of virulent strain (Bullock, 1976);
c. McCarthy’s latex agglutination to identifying *Aeromonas salmonicida*;
d. Indirect FAT for identification (Klontz and Anderson, 1970) and

e. Precipitin and agglutinin tests (Bullock, 1966).

**H  Aeromonas hydrophila**


**I  Pseudomonas fluorescens**

a. Fluorescent test (not a serological test).

**J  Enteric red mouth (Yersinia ruckerii)**

a. Indirect FAT for confirmatory identification (Bullock, 1976);
b. Slide agglutination for confirmatory identification (Bullock, 1976) and


**K  Vibrio anguillarum**

a. Two serotypes cause fish diseases (Bullock, 1976);
b. Slide agglutination for identification (Bullock, 1976) and


**L  Edwardsiella tarda**

a. Availability of diagnostic antiserum (Bullock, 1976).
Corynbacterium kidney disease

a Double diffusion test for identification (Bullock et al., 1974);
b Indirect FAT for confirmatory identification (Bullock, 1976);
c FAT for identification (Bullock & Stuckey, 1975)
and
d Serological diagnosis (Chen et al., 1974).

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Infectious protozoan diseases

Infectious protozoan diseases are caused mainly by two parasites viz., *Ceratomyxa shasta* and *Myxosoma cerebralis*; the former infects the viscera and musculature of the host while the latter parasitizes the branchial cartilage of a fish.

A *Ceratomyxa shasta*

Considerable work on myxosporidians has already been done by Schafer (1968), but very little is known yet about its life cycle and mode of transmission. A more comprehensive account of this parasite has recently been made available by Johnson *et al.* (1978).

1 Geographical distribution

The parasite has a restricted geographical distribution and has not yet been recorded from India. Zinn *et al.* (1977) have proved beyond doubt that most salmonid species are susceptible to the disease caused by *C. shasta* and it is no wonder that trouts cultured in India might also be suffering from the attack of this parasite.

2 External and internal signs of infection

External symptoms of *Ceratomyxa* infection are variable; infected fishes lose appetite, are darker in colour, lethargic and prefer shallow waters. Distended abdomen due to accumulation of ascitic fluid and exophthalmia are the other signs develop later in the disease (Schafer, 1968); large pustules filled with fluid containing blood, necrotic tissue, etc., are the other external symptoms (Conrad & Decew, 1966; Johnson, 1975).

Internal symptoms of the infection are:

a Mucoid intestinal contents; posterior intestine swollen and haemorrhagic (Conrad & Decew, 1966);

b All layers of hind gut are swollen (Wales & Wolf, 1955);
c Abscessed lesions on the musculature (Wood, 1979);

3 Identification of the parasite

Noble (1950) described the parasite. The dimensions of the binucleate spores vary from 14-23 µm in length and 6-8 µm in width at the sutural line. Smear preparations of intestine and gall bladder and stained by Ziehl-Neelsen method (without heating carbol fuchsin) help in most reliable identification of the parasite (Hoffman and Meyer, 1974).

4 Effect of temperatures on the parasite

At low temperature (below 6.7°C) the infection of C. shasta can be suppressed but at higher temperature (between 14 and 23.3°C) mortalities of the fish may occur from the 12th day onwards (Udey et al., 1975).

5 Mode of transmission

Direct transmission of C. shasta from fish to fish never occurs but salmonids get infected when their ambient waters contain infectious stage of the parasite. Under laboratory condition at 20°C Ceratomyxosis did occur when bottom sediments of a lake, where the disease outbreaks every year, were used (Fryer, 1971). In winter susceptible salmonids could not be infected with the parasite (Johnson, 1975).

6 Prevention and control

No treatment to control the disease is yet known (Needham & Wootten, 1978). Sanders et al. (1972) have suggested a combination of MicroFloc filtration followed by exposure to chlorine (2.2-5.3 ppm for 60 min.) for hatchery management. Ultraviolet radiation has also been found to eradicate the pathogens (Bedell, 1971). Genetic selection and production of resistant strains of rainbow and steelhead trouts and chinook salmon have also been reported against Ceratomyxosis (Johnson, 1975; Zinn et al., 1977).

B Myxosoma cerebralis

Of all the myxosporidian parasites invading the cartilage of a fish Myxosoma
cerebralis is recognized as the most important. Myxosoma cartilaginis, encountered from the cartilage at the base of fin rays and gill arches of centrachids (Hoffman et al., 1965), and Myxobolus aeglefini, causing erosion or hypertrophy of the cranial cartilages of plaice, hake and haddock (Sindermann, 1970) are found to be less harmful than M. cerebralis. As such a bulk of literature is already available on the last named parasite. However, the comprehensive account of M. cerebralis has been made available by Halliday (1976).

1 Geographical distribution of M. cerebralis


2 External and internal signs of infection

The most conspicuous sign that develops after 2 or 3 months of infection is tail-chasing; as such, the disease is known commonly as “whirling disease”.

Head cartilage, particularly around the auditory capsule, is fed upon by the vegetative forms of the parasite resulting in extensive damage and deformities of the cartilage of trouts. With the advancement of the disease the parasite invades the spinal chord causing severe deformities. The fish looks darker in colour.

3 Identification of the parasite

Morphology of the spores of M. cerebralis (Hofer, 1903) was first compared with M. cartilaginis (Hoffman, Putz, and Dunbar, 1965) in 1971 (Lom and Hoffman, 1971). The dimensions of spore of the former vary between 7.4 and 9.7 µ in length and 6.2 and 7.4 µ in breadth. The polar capsules 5-6 X 3-3.5 µ. Markiw & Wolf (1974 & 1978) have evolved spore detection methods and recognized M. cerebralis using fluorescent antibody techniques. In vitro sporulation of the parasite was done by Wolf & Markiw (1976). Rydlo (1975) quantitatively studied the spores of M. cerebralis. Biological
properties of the invasive stage of the parasite have been made available by Uspenskaya (1978). Wolf & Markiw (1979) have used silver nitrate for staining spores and other stages of *M. cerebralis*.

4 Effect of temperature on the parasite

Spores of *M. cerebralis* remain viable even at a very low temperature (−20°C) for a considerable period (Putz, 1970; Hoffman & Putz, 1971), but are killed when kept for 10 minutes at a temperature of 60 to 100°C (Hoffman & Putz, 1969).

5 Mode of transmission of the parasite

Transmission of *M. cerebralis* is effected by shipments of live and frozen trouts and their eggs or alevins. Birds and human agencies may help in the transmission of the parasite (Halliday, 1974a).

6 Prevention and control

Schaperclaus (1954), Rasmussen (1965) and Bogdanova (1968) have suggested destruction of the affected trouts to control the infection of *M. cerebralis*. However, disinfection, husbandry, and water treatment have been advocated to prevent “whirling disease” caused by *M. cerebralis* (Rasmussen, 1958, 1961, and 1965; Hoffman *et al.*, 1962; Brierly & Scott, 1969; Hoffman and Putz, 1969; Ghittino, 1970a; Hoffman, 1970b & c; Hoffman and Hoffman, 1972).

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