

Recent achievements in studies on diseases of common carp (*Cyprinus carpio* L.)

Zs. Jeney*, Galina Jeney

Fish Culture Research Institute, PO Box 47, H-5541 Szarvas, Hungary

Abstract

Parasitic, fungal, bacterial and viral diseases of common carp (*Cyprinus carpio* L.) are reviewed. Besides a general overview of parasites of carp, swimbladder inflammation, caused by *Sphaerospora* spp., is discussed in detail. *Saprolegnia* spp. is the most important fungal pathogen. *Aeromonas hydrophila* and the atypical *Aeromonas salmonicida*, as well as *Flexibacter columnaris*, are described as the major bacterial pathogens of carp. Spring viraemia of carp caused by *Rhabdovirus carpio* is presented as the main viral infection of common carp. Details on methods of treatment and prevention are presented together with a description of the given diseases. The role of environmental stress, including “normal” culture practice and pollution, in the outbreak of diseases of common carp is discussed. Prospective methods to minimize the risk of diseases as well as their limitations are presented.

Keywords: Diseases and their control – fish; Parasites; Fungi; Bacteria; Viruses; Diagnostic parameters

1. Introduction

Carps (common carp, Indian major carps and Chinese carps) are the biggest group of cultured fish. Their aquaculture production was 6212000 mt in 1989. Among them common carp (*Cyprinus carpio* L.) is probably the most widely “spread” species. Its annual production was 1112726 mt in 1990 (FAO, 1992). This figure does not contain the production of three countries in Asia (Bangladesh, India and Vietnam), producing 1170000 mt of freshwater fish, including a large proportion of common carp. In a recent review on aquaculture in Asia, New and Csavas (1993) gave a corrected figure (850530 mt) for common carp production in 1990 as well as a forecast for Asia for the year 2000: 1073704 mt.

Common carp is cultured on almost all continents under a wide range of geographic, climatic and technological conditions. As a result, there is a large group of pathogens

* Corresponding author.

parasitising on carp and/or causing diseases. This is especially true for parasitic diseases. Due to limited space, only selected aspects of diseases of cultured common carp can be reviewed. General overviews of parasitic, fungal, bacterial and viral diseases, as well as environmental stress are followed by a detailed description of the most important diseases. Nutritional diseases are not discussed in this review.

2. Parasitic diseases

Representatives of almost all major systematic groups of parasites have been found in carp. Altogether 226 parasite species were described (Landsberg, 1991). The most important ones that cause disease are listed in Table 1. As can be seen, with few exceptions they have a world-wide geographical distribution.

It should be emphasised that environmental stress appears to play a major role in the development of epizootics caused by parasites of carp. This is especially true for ectoparasitic infestations, where mortality rates are typically high in habitats where fish become overcrowded, such as in overwintering storage ponds or in densely populated growing ponds and reservoirs (Paperna, 1991). Other environmental factors such as inadequate oxygenation, accumulation of metabolites (primarily the highly toxic ammonia), handling, fish concentrations during netting or harvesting etc. may also play a role in the development of epizootics with ectoparasitic protozoa.

Swimbladder inflammation (aerocystis)

Swimbladder inflammation (SBI) is a common, economically important disease of the common carp. First observed in Hungary by Szakolczai (1967) among three-summer-old carps, it had long been known in neighbouring countries (Hofer, 1904; Roth, 1922). Here we list the main steps in the long history of this disease.

There are divergent opinions on the aetiology of SBI. Certain authors have regarded it as a viral disease (Arshanica, 1969; Ahne, 1973; Bachmann and Ahne, 1973), while others (Neciporenko et al., 1963; Kanaev et al., 1967; Szakolczai, 1967; Markiewicz, 1966; Mattheis and Kulow, 1967; Kocylowski et al., 1970) have advocated the primary aetiological responsibility of bacteria, because of the isolation of many bacterial strains from carp with SBI. Szakolczai (1967) detected bodies reminiscent of unicellular parasites (*Coccidia*, *Pleistophora*) in squash preparations of affected swimbladder and suggested they might be the causal agent of the disease. Kanaev and Kuzmin's (1970) report on experimental reproduction of SBI by feeding carp organs containing *Myxobolus* spores agreed well with Szakolczai's protozoan hypothesis. Otte (1966) implied that the blood flagellate *Cryptobia cyprini* may be responsible for SBI. Molnár (1980a) observed during studies on carp renal sphaerosporosis that the latter condition was frequently developed by fish with clinical SBI, and he postulated a relationship between the prevalence of SBI, renal sphaerosporosis and the condition caused by the C-blood protozoan originally described by Csaba (1976). Waluga and Budzinska (1980) detected the developmental stages of *Sphaerospora carassii*, the causal agent of gill sphaerosporosis in carp, in visceral blood vessels, but did not pursue the possible identity of these stages with the C-blood protozoan. Indirect proof of a relationship between SBI and renal sphaerosporosis has emerged from Grishchenko's (1967)

Table 1
Parasites of common carp

Parasite	Geographical range	Gross pathology
Protozoa		
<i>Ichthyobodo</i> spp.	C	dpbc
<i>Cryptobia</i> spp.	C	circ
<i>Eimeria</i> spp.	C	dig
<i>Ichthyophthirius multifiliis</i>	C	dpbc
<i>Chilodonella</i> spp.	C	dpbc
<i>Trichodina</i> spp.	C	dpbc
Myxozoa		
<i>Myxidium</i> spp.	C	vm, c, resp
<i>Sphaerospora</i> spp.	As, E, NA	vm, c, resp
<i>Myxobolus</i> spp.	C	vm, c, resp
<i>Henneguya</i> spp.	C	vm, c, resp
Helminths		
Monogenea		
<i>Dactylogyrus</i> spp.	C	dpbc, resp
<i>Gyrodactylus</i> spp.	C	dpbc, resp
Trematoda		
<i>Diplostomum</i> spp.	C	so
<i>Posthodiplostomum</i> spp.	C	vm, c
<i>Sanguinicola</i> spp.	C	circ
<i>Clonorchis sinensis</i>	As	vm, c
<i>Opistochis felineus</i>	As	vm, c
Cestoda		
<i>Caryophyllaeus</i> spp.	C	dig
<i>Ligula intestinalis</i>	C	vm, f
<i>Bothriocephalus acheilognathi</i>	As, E, NA, SU	dig
<i>Khawia sinensis</i>	C	dig
<i>Triaenophorus</i> spp.	C	vm, c
<i>Philometroides</i> spp.	E, SU	dig
Nematoda		
<i>Anisakis</i> spp.	As, E	vm, c
<i>Contracaecum</i> spp.	C	dig
<i>Camallanus</i> spp.	C	dig
<i>Philometra</i> spp.	C	vm, c, f,
<i>Acanthocephala</i> spp.	C	dig
Annelida		
<i>Hirudinae</i>	C	dpbc, resp
Mollusca		
<i>Glochidia</i>	C	resp
Arthropoda (Crustacea)		
<i>Ergasilus</i> spp.	C	resp
<i>Lernaea</i> spp.	C	dpbc
<i>Tracheiastes</i> spp.	C	dpbc
<i>Argulus</i> spp.	C	dpbc

Abbreviations: Geographical range — C = cosmopolitan; As = Asia; E = Europa; NA = North America; SU = former Soviet Union. Gross pathology — dpbc = decaying and puriginous branchio-cutaneous; resp = respiratory; so = sensory organs; circ = ciculatory; vm = viscero-muscular; f = free parasite; c = cystic; dig = digestive.

From De Kinkelin and Hattenberger (1986) and Molnár and Szokolczai (1981) with modification.

observation that hosts with SBI harboured spore-like bodies in the renal tubules, and from a photograph published by Schäperclaus (1979) as the image of the swimbladder wall, although it actually shows a renal tubule packed with sphaerospores.

Kovács-Gayer has regularly detected developmental stages of parasitic protozoa in the wall of the swimbladder of carp fry with SBI (Molnár, 1980b; Kovács-Gayer et al., 1982).

Dykova and Lom (1982) have described the parasite which frequently occurs in the kidney of the common carp and which was formerly regarded as *Sphaerospora angulata* Fujita, 1912, as a new species, *Sphaerospora renicola*.

Since the works of Molnár (1980), Kovács-Gayer et al. (1982), Körting (1982) and Csaba et al. (1984) led to the identification of parasites designated as K-protozoa causing swimbladder inflammation in the common carp, with the presporonic developmental stages of the kidney parasite *Sphaerospora renicola* Dykova and Lom 1982, a vast amount of knowledge has accumulated on the complex developmental cycle of *Sphaerospora*-type parasites. It has become known that the early developmental stages described by Csaba (1976) and designated as ‘‘C blood protozoa’’ by Molnár (1980) and as ‘‘UBO’’ by Lom et al. (1983) consistently appear in the blood of carp before renal sphaerosporosis develops. The works of Lom et al. (1985) have revealed that the blood stages can also be demonstrated in the blood of other *Sphaerospora*-infected cyprinid fishes. Moreover, in the blood of these cyprinids Baska and Molnár (1988) found circulating forms morphologically resembling the K-stages of the common carp. In common carp, the K-stages were earlier detected first of all in the swimbladder wall (Kovács-Gayer et al., 1982; Körting, 1982; Csaba et al., 1984; Landsberg, 1986; Odening, 1987). The possible occurrence of K-stages in other locations has only been suggested by Körting (1982), Körting et al. (1984) and the Ter Höfte et al. (1984), who found the developmental stages in the integument of the head, in the orbit and in hepatic granules of the common carp. Recently, Lom et al. (1991) have found *Sphaerospora* stages resembling K-protozoa in the rete mirabile of non-cyprinid fish (*Gasterosteus aculeatus*), although they failed to clearly identify these forms with the developmental stages of *S. elegans* found in the kidney.

Molnár (1993) found that *S. renicola* K-stages, previously considered to be typical swimbladder parasites, are blood stages just like C-parasites; however, they are frequently caught in the capillaries. These stages can also produce lesions in organs other than the swimbladder, and are directly responsible for the exophthalmos regarded as a sign of swimbladder inflammation, as well as for the intraorbital haemorrhages and tissue necrosis. The following detailed information is based on the above literature as well as on the review by Molnár and Szakolczai (1992).

Causative agent. The early developmental stage of *Sphaerospora renicola*, the causative agent of swimbladder inflammation, can be detected in the acute stage from the wall of the swimbladder. In the fibrous connective tissue of infected swimbladder, groups of protozoa with a diameter of 17–30 μm can be visualized, that are parasitic cells developing with gemmation, as well as their progenies. In the plasma of the primary developmental form, occurring among the tissues, 20–40 secondary forms will develop, and later they will serve as a site for development of 2–2 tertiary forms. After destruction of the primary form the so-called tertiary developmental stages, which include one secondary form and two tertiary forms, through the bloodstream will reach the renal tubules where spores of *S. renicola* will

develop from them. Following the acute phase of SBI the sphaerosporosis will always be developed in the renal tubules.

SBI is always preceded by the appearance of so-called Csaba protozoa. These are the earliest developmental stages of *S. renicola*; they flow freely in the blood and propagate by endogen gemmation, resulting in 8 daughter cells in each mother cell.

The parasitic SBI finishes in 3–4 weeks, and if no complication occurs, the disease is over. In most cases the facultative bacteria (*Aeromonas* spp., *Pseudomonas* spp.) will cause complications.

Pathogenesis. On the basis of the observed changes the disease can be divided into 5 stages: (1) In the first stage the blood vessels are dilated, and beside the hyperaemia only pitechiae can be observed in the swimbladder. (2) In the second stage the hyperaemia decreases; in the wall of the swimbladder brown or black spots with sharp borders can be seen. (3) In the third stage the wall of the swimbladder becomes thickened, and an exudate appears as a sign of inflammation. Peritonitis, as well as adherence of the peritoneum covering the intestine and the abdomen, is not unusual. (4) The fourth stage is characterised by exacerbation of the above processes, and certain layers of swimbladder are necrotised. (5) In the fifth stage cysts are formed in the wall of the swimbladder; the cavity of the swimbladder is filled with serous-purulent matter. Inflammation spreads to other organs, and secondary peritonitis develops.

The first two stages are typical of parasitic swimbladder inflammation, while in the third the bacterial complication probably plays a role. The fourth and fifth stages appear only when there are complications.

Clinical symptoms. In the first two stages the fish do not show any typical signs. The changes listed (haemorrhages, hyperaemia) can be detected only at necropsy. In the chronic form (the last three stages) sick individuals lose their balance, and swim on their back or side, or with the head downwards. Their caudal fin stands out of the water; from time to time they try to dive but without success.

The abdomen of sick fish increases markedly in size. This enlargement expands into the whole abdomen or appears only in the region of the anus, depending on whether both parts or only the second part of the swimbladder is affected.

In the course of autopsy the thickened wall of the abdomen can be detected. Abdominal organs generally do not show pathological signs. Due to functional disturbances of the kidneys some fish will show general oedema, but without haemorrhagic signs referring to spring viraemia.

The swimbladder loses its typical form, and either both or only one of the two chambers will enlarge. The glossy silver-white colour of the outer wall becomes mat or whitish grey. On cutting through the wall 1–2 cm thick parts are seen that include holes of different sizes with serous-purulent matter. In the exudate very often green-bean-sized fibrin nodules can be found. The real cavity of the swimbladder is strongly contracted, and contains a minimal amount of a water-like serous exudate. The inner epithelium is healthy, with a glossy silver-white colour.

Some 10–20% of fish will show clinical signs, but mortality is only occasional. These fish are very sensitive to any kind of handling (harvesting, transport etc.). Other fish species

kept together with carp (bream, crucian carp, pike-perch) do not show these signs of swimbladder inflammation.

In the early stages of the disease protozoa can be easily detected in the blood and swimbladder by impression smears or histological preparations. Sphaerospores in the kidney are visualized on native preparations. In chronic forms a diversified bacterial flora can be isolated from the swimbladder.

The course of the disease is rapid in fingerlings, the parasitic form easily recovers, the swimbladder regenerates within a few weeks, and only the pigmented spots will indicate the past infection. In the case of complications the greater part of the population will die. In the chronic form, only occasional mortality is observed.

Treatment. No treatment is available. For prevention of sphaerosporosis fumagillin is the only effective medicament. In the course of development of different forms of swimbladder inflammation 1–2 weeks feeding with medicated food containing 0.1% fumagillin will prevent the development of disease. This very expensive procedure can be avoided by preventing bacterial complication by feeding antibiotics, as well as by destroying the infective spores by drying and disinfecting the ponds.

3. Fungal diseases

Fungal infections of common carp are serious problems of both natural fisheries and intensive aquaculture. Fungal infections are also a persistent problem in warmwater hatcheries as any dead eggs quickly become a focus of proliferation which can then spread to adjacent healthy eggs. In spite of their importance our knowledge about the fungal infections is still poor, for two basic reasons: difficult identification of pathogenic fungi (Pickering and Willoughby, 1982) and the prolific growth of saprophytic fungi once the fish is dead (Willoughby, 1971). The potential fish pathogens of the family Saprolegniaceae was reviewed by Pickering and Willoughby (1982) (Table 2).

Saprolegnia spp., *Achlya* spp. and *Branchiomyces* spp. are probably the major pathogenic fungi in the common carp. Here we give a short description of the genus *Saprolegnia*, probably the most, but still insufficiently, studied of these pathogens, as well as saprolegniasis of common carp, the disease caused by them.

Table 2
Fungi and fungal diseases of carp

Fungi	Disease	Reference
Oomycetes		
<i>Achlya</i> spp.	Water mould	Tiffney (1939)
<i>Saprolegnia</i> spp.	Cotton wool disease, saprolegniosis	Tiffney (1939)
Branchiomyces		
<i>Branchiomyces sanguinis</i>	Gill rot, branchiomycosis	Plehn (1912)

Saprolegniasis

Causative agent. The genus *Saprolegnia* is characterised by the production of biflagellata zoospores which swim away after emptying of the zoosporangia. New zoosporangia often form in the old ones. *Saprolegnia parasitica* Coker (in Willoughby's system of classification syn. *Saprolegnia diclina* Type 1) is most frequently associated with fish, and produces sexual organs only in special cases (Oláh and Farkas, 1978). Zoosporangia are 300–600 μm long, cylindrical, often irregular in shape, and appear on the 2nd–3rd day of culture. Ripening and release of zoospores soon begins. The zoospores are spherical with two flagella, motile and 10 μm in diameter.

Clinical signs. Gross pathology of *Saprolegnia* infection is characterised by conspicuous fungal colonies growing on the body surface of the fish. These cotton-wool-like lesions are normally white in colour, or discoloured by the accumulation of debris between the fungal hyphae or as a result of a simultaneous bacterial infection. The fungus can colonise almost any area of the external surface of the fish.

Histopathological changes of the *Saprolegnia* infection are characterised as follows. The penetration of fungal hyphae is restricted to the epidermis and dermis. Muscular lesions are uncommon but occasionally develop when fungal and bacterial penetration to the muscle surface occurs. In case of small fish, hyphae may invade the deeper tissues of the fish and penetrate the vital organs (Bootsma, 1973). Degenerative changes in the epidermis and dermis include focal oedema and ultimate sloughing of the epidermis (Pickering and Willoughby, 1982). Inflammatory responses are normally absent or weakly developed (Wolke, 1975; Pickering and Richards, 1980) unless bacterial infection also develops. According to Pickering and Willoughby (1982) there is no evidence that pathogenic *Saprolegnia* strains produce any toxins that might be transmitted systemically.

Once infected, fish do not normally recover from saprolegniasis unless treated. Death of the fish is due to massive osmoregulatory problems caused by destruction of the superficial tissues over large areas of the body surface although the survival time is influenced by the precise location of damaged tissue.

Saprolegniasis has been considered to be a secondary condition with fungal hyphae normally colonising existing lesions on the fish. Although fungal infections are often secondary to other diseases and to physical damage, there is also evidence that *Saprolegnia diclina* Type 1 (syn. *S. parasitica*) can also act as a primary pathogen on undamaged, healthy fish (Pickering and Christie, 1980). The following conditions can increase the susceptibility of fish to infection: wounds are potential sites for *Saprolegnia* colonisation; physical damage (Tiffney, 1939; Egusa, 1965); high stocking density; any environmental conditions and manipulations that result in physical damage; sexual maturation; other infections, especially those that cause ulceration of, or damage to, the superficial tissues of the fish; stress-mediated increase of plasma corticosteroid (Nord et al., 1977; Neish and Hughes, 1980).

Treatment. Malachite green has been and still is the treatment of choice for *Saprolegnia* infections during all stages of the life cycle of carp and other commercially produced fish species (Foster and Woodbury, 1936; Scott and O'Bier, 1962; Nelson, 1974; Alderman,

1985, 1991). Recently, stabilised chlorine dioxide was suggested as a control agent for fungal infections, first of all saprolegniasis (Hiney and Smith, 1993). General measures to improve water quality should also be considered.

4. Bacterial diseases

According to the most recent review, altogether only 37 bacteria have been reported to be pathogenic for fish, which is surprisingly low considering the large number of fish species and their diverse environments (Fryer and Rohovec, 1993). Of these 37 bacteria, 8 were isolated from and associated with diseased carp (Table 3).

Motile Aeromonas septicaemia

The term “motile *Aeromonas septicaemia*” (MAS) is used to describe motile aeromonad infections of warmwater fish, including common carp. The motile aeromonads are characteristically bacteria of fresh water. The motile species are often ubiquitous members of the aquatic ecosystem, but all can be components of the microbial flora of aquatic animals and may be pathogens of poikilotherms, homiotherms and even man (Fraire, 1978; Salton and Schnick, 1973). Motile aeromonads show very considerable heterogeneity and their taxonomy is confused. This group comprises *A. hydrophila* (syn. *A. liquefaciens*), *A. sobria* and *A. caviae* (syn. *A. hydrophila* subsp. *anaerogenes*). Of these *A. hydrophila* is of great importance for carp culture. According to Austin and Austin (1987), *Aeromonas hydrophila* may include several distinct species. *A. punctata* is no longer a recognised species and isolates previously identified as *A. punctata* would now be classified as *A. hydrophila* (Frerichs, 1993).

Although motile aeromonads may occur in relatively unpolluted water, they are much more abundant in waters with a high organic load (Hazen et al., 1978). Because the “normal” conditions of a carp-rearing pond are close to these, and *Aeromonas hydrophila* is a normal inhabitant, carp are constantly exposed to infection. Disease outbreaks are common when carp are under stress from crowding, low oxygen, high temperatures, etc., and significant losses from such outbreaks occur annually.

Table 3
Bacteria and bacterial diseases of carp

Bacterium	Disease	Reference
<i>Aeromonas salmonicida</i>	Carp erythrodermatitis	Bootsma et al. (1977); Fijan et al. (1977)
<i>achromogenes</i>		
<i>Aeromonas hydrophila</i>	Motile <i>Aeromonas septicaemia</i>	Fijan (1972)
<i>Edwardsiella tarda</i>	Edwardsiellosis	Bullock and Herman (1985)
<i>Pseudomonas fluorescens</i>	<i>Pseudomonas septicaemia</i>	Inglis and Hendrie (1993)
<i>Flexibacter columnaris</i>	Flexibacteriosis	Bootsma and Clerx (1976)
<i>Flavobacterium branchiophila</i>	Bacterial gill disease	Farkas (1985)
<i>Streptococcus</i> sp.	Streptococcal septicaemia	Farkas and Oldh (1986)
<i>Mycobacterium</i> sp.	Mycobacteriosis	Reichenbach-Klinke (1972)

Causative agent. *A. hydrophila* is characterised by active motility, achieved by means of single polar flagellum. It is a Gram-negative bacilli measuring $0.5 \times 1.0\text{--}1.5\ \mu\text{m}$. *A. hydrophila* is aerobic and facultatively anaerobic, fermenting carbohydrates with the formation of acid and producing 3-butanediol. It is cytochrome-oxidase-positive, reduces nitrates, and is resistant to the vibriostat 0/129 (Roberts, 1993). As to extracellular production, *A. hydrophila* produces haemolysin, cytotoxins and enterotoxins (Boulanger et al., 1977; Wretling et al., 1971; Donta and Haddow, 1978) as well as gelatinase, caseinase, elastase, lectithinase and desoxyribonuclease (Nord et al., 1975).

The pathogenicity of *A. hydrophila* often seems to be associated with stressed or compromised hosts. The principal feature of the pathogenesis is generalised dissemination in the form of a bacteraemia, followed by elaboration of toxins, tissue necrosis and bacterial haemorrhagic septicaemia. In most cases the *A. hydrophila* infection has a secondary character and the disease is associated with primary viral or parasitic infection, with sudden change in environmental or nutritional status, or with husbandry changes. There is some doubt, therefore, as to whether *Aeromonas hydrophila* should even be considered as a primary pathogen. There is little doubt, however, that once it has invaded a host which has been compromised for whatever reason, it rapidly makes the condition worse and can be responsible for ultimate death. The complexity of the situation makes the diagnosis and treatment difficult. Unless the primary cause has been excluded, treatment of aeromonosis will be of little value in the longer term (Roberts, 1993).

Clinical signs. Clinical signs of the disease are rupture of minor blood vessels and haemorrhages caused by haemolysin, which may be associated with ulcerative skin lesions. The size of external lesions may vary. The ulcers are usually shallow. Secondary infection of ulcers by fungi is common. In carp the condition may be pre-acute, with few signs, acute, with the typical syndrome described above, or chronic with large, long-standing ulcers. It is often associated with abdominal oedema or dropsy. Internally the principal feature is hyperaemia of the viscera with haemorrhage over mesenteritis and within the visceral and parietal peritoneum. There may be an excess of clear or reddish ascitic fluid in clinical dropsy. The spleen and the kidney are enlarged.

Treatment. Control of *A. hydrophila* infection should be linked to the control of the underlying factors which have facilitated its invasion of the host. However, when an outbreak occurs, it is very difficult to determine the underlying stress factors or primary infections and it is not possible to change water flows or stocking densities with any ease. Thus treatment is limited to the use of antibacterial compounds in the feeds. Generally there are two basic problems with the application of medicated feeds. The first is the development of antibiotic resistance (Mitchell and Plumb, 1980). Such a plasmid-related drug resistance has been described for most of the antibiotics used to treat aeromonad infections (Chang and Bolton, 1987; Ansary et al., 1992). The second problem is that the efficacy of such treatment could be very low in some cases due to the low appetite of the sick fish.

Efforts to develop vaccines against MAS have not been successful, mainly because of the wide serological diversity among *A. hydrophila* strains (Wolf, 1988).

Carp erythrodermatitis

Ulcerative erythrodermatitis of carp is a subacute to chronic disease which varies in morbidity and mortality, and is generally associated with ulcerative inflammatory lesions in the skin and muscle. Before 1971 it was considered as the chronic form of disease called “dropsy of carp”. Fijan et al. (1971) isolated a virus (*Rhabdovirus carpio*) from the acute form of infectious dropsy and differentiated the acute form as “spring viraemia of carp” and the chronic form as “carp erythrodermatitis”. Bootsma (1975) isolated a bacterium from the ulcerative form. Later Bootsma and Blommaert (1978) classified the bacterium as an atypical *Aeromonas salmonicida*.

Causative agent. The disease is caused by an atypical *Aeromonas salmonicida* (CE bacterium) which is a Gram-negative, non-motile bacterium measuring $0.4\text{--}0.6 \times 0.8\text{--}1.5\text{ }\mu\text{m}$. Atypical *Aeromonas salmonicida* from carp belong to the subspecies known as *Aeromonas salmonicida* subspecies *nova* according to biochemical characteristics (Bohm et al., 1986) and DNA/DNA re-association studies (Belland and Trust, 1988). The bacterium can be isolated from the hyperaemic surroundings of the ulcers of sick fish in most cases. On blood agar after 24 h it will produce small colonies, under which B haemolysis can be observed after 48 h. The bacterium does not produce pigment. It ferments glucose and maltose with the formation of acid, but without gas production. The CE bacterium is cytochrome-positive and hydrolyses gelatine and casein. It is resistant to penicillin and polymyxin B, but sensitive to medicines like oxytetracycline, neomycin, nitrofurantoin, Sumetrolim (sulfamethoxazole + trimethoprim) (Molnár and Szakolczai, 1992). It seems that the CE bacterium is able to infect carp in the region of mechanical injuries, such as wounds caused by blood-sucking parasites.

Clinical signs. At an early stage, small spherical nodules can be observed on the fins; later at these sites the finrays break down. The scales are surrounded by inflamed rings; afterwards the scales are lost, and the epithelium and corium become necrotized. The resulting ulcers with markedly hyperaemic surroundings often spread into muscle. Such ulcers can be found everywhere on the body except the head.

Histologically, there is a lack of epithelium, and necrosis of the corium and muscle, as well as the presence of large numbers of inflammatory cells, can be observed. Inside the ulcer is filled with granular tissue. In internal organs neither macroscopic nor microscopic pathological signs can be seen, but sometimes slight fatty degeneration is visible.

At the beginning the appetite of the fish is normal, but later it decreases due to exacerbation of ulceration. The course of the disease is slow, mortality rarely reaching 20–25%. Other economic losses are caused by the slow growth of the sick fish. In the presence of favourable environmental conditions the erythrodermatitis is curable. The sites of healed ulcers show strong pigmentation, which differs from healthy skin.

Treatment. Treatment is successful with the use of antibacterial compounds in the feeds. Oxytetracycline, neomycin, nitrofurantoin and Sumetrolim (sulfamethoxazole + trimethoprim) are usually effective, and although resistance to them was not found in Hungary, standard bacteriological and resistance investigations are highly recommended before starting the treatment course (Molnár and Szakolczai, 1992). In prevention of erythrodermatitis

of carp, good rearing practice and improvement in environmental conditions (firstly by removing blood-sucking parasites) play the primary role.

Columnaris disease

Columnaris disease is distributed worldwide. Many species have been effected by columnaris disease, including the common carp. Gill necrosis of common carp, caused by unfavourable environmental conditions (ammonia, pH and temperature) and *Flexibacter columnaris* infection, was suggested to count as a separate disease (Farkas and Oláh, 1986). These authors described the gill necrosis as a three-stage, complex disease. The first stage is initiated and maintained by environmental stress, caused most typically by ammonia intoxication. The second stage starts when *F. columnaris* invades the damaged gill. The third stage occurs if the fish survives the bacterial invasion. In the present review we give a general description for columnaris disease of common carp.

Causative agent. *Flexibacter columnaris* syn. *Cytophaga columnaris* is the causative agent of this disease. Carp is susceptible to columnaris under environmental conditions favourable to the bacterium and stressful to the fish. Outbreaks occur when water temperatures reach 20°C and above (Farkas and Oláh, 1986). *F. columnaris* primarily attacks the external tissues, and uninjured tissue also appears to be attacked. The first indication of the infection is generally the appearance of a white spot on some part of the head, gills, fin or body. This is usually surrounded by a zone with a distinct reddish tinge, leading to under-running of adjacent skin. Lesions on the end of gills or fins extend principally from the distal end towards the base, and the tissues are eroded and destroyed. Lesions are covered with a yellowish white mucoid exudate consisting largely of swarms of *F. columnaris* (Wakabayashi, 1993). The bacteria are not usually found systemically until a relatively large amount of external skin or gill damage has taken place; thus it would appear that the bacteria enter the bloodstream through the external lesions and are probably not directly involved in causing death (Wood, 1974).

In wet preparations *F. columnaris* shows a slow gliding movement and gather into characteristic column-like masses. The filamentous cells on the columns also display an active flexing movement. The cells are Gram-negative, slender and rather long bacilli, 0.3–0.5 µm long. The bacteria grows well on low nutrient cytophaga agar, producing pale yellow rhizoid colonies, which can vary markedly in size and shape. The colonies tend to spreading with irregular margins and they adhere to the agar (Wakabayashi, 1993). According to Bernardet and Grimont (1989), growth occurs in cytophaga broth supplemented with 0.1 or 0.5% NaCl and at 10–33°C, strictly aerobic. Catalase and cytochrome oxidase are produced; nitrate is reduced to nitrite; hydrogen sulphide is produced. Cellulose, carboxymethyl cellulose, chitin, starch, aesculin and agar are not hydrolysed. No acid is produced from carbohydrates in ammonium salt/sugar medium. Gelatin, casein and tyrosine are hydrolysed. Lysine, arginine and ornithine are not decarboxylated.

Epizootology. In the epizootology of the disease the following environmental factors play an important role: water temperature, water quality and the density of fish.

Flexibacter columnaris attacks fish only at comparatively high water temperatures. In experimental infection studies, carried out with different fish species, below the range of

9.4–12.8°C no effect of bacteria was reported (Fish and Rucker, 1943; Holt et al., 1975). Wakabayashi and Egusa (1972) studied the effect of water temperature on columnaris disease. No mortality occurred in experimentally infected fish at 5 or 10°C, 25% of those held at 15°C died and all of the exposed fish held at 20–35°C died. The mean time to death was 7.0, 3.0, 1.8, 1.0 and 1.0 days at 15, 20, 25, 30 and 35°C, respectively. Pacha and Ordal (1970) reported a clear relationship between the pathogenicity of *F. columnaris* and the water temperature: high-virulence strains were found to infect fish and produce disease at lower temperature than low-virulence strains. Mortalities were produced at 12.8°C by high-virulence strains, whereas low-virulence strains were able to initiate infection only when the temperature was increased to 20°C.

Water quality is the other important environmental factor because the long-term survival of the pathogen in pond water is thought to provide a reservoir of infection. Fijan (1968) indicated that *F. columnaris* could persist for longer periods in water of high hardness and organic matter content, but survival time was reduced significantly in water with a pH of 6.0. Soft water of about 10 ppm CaCO₃, especially when acid or of low organic matter content, did not provide a favourable environment for the organism. Chowdhury and Wakabayashi (1988) showed that *F. columnaris* survived for the longest period in water with hardness of 73.7 mg/l CaCO₃. Arsenic and nitrite (5 ppm) enhanced, while copper decreased the *F. columnaris* infection (MacFarlane et al., 1986; Hanson and Grizzle, 1985). *F. columnaris* grows very well on the particles derived from the break-up of pelleted diets in water (Sugimoto et al., 1981).

Density of fish has a clear effect on the *F. columnaris* infection: at higher stocking densities, higher rates of infection as well as mortality occur (Becker and Fujihara, 1978). It also seems that crowding not only reduces resistance of fish to *F. columnaris* but also increases the chances of the organisms coming into contact with the fish. The results of the study carried out by Wakabayashi and Egusa (1972) suggested that the higher the initial density of *F. columnaris* or the concentration of fish, the more certainly infection occurred and the earlier mortalities started to occur.

Treatment. Control of columnaris disease should be based firstly on good rearing practice and on improvement of environmental conditions. Some of the possible measures have practical implications, such as avoidance of overfeeding and reduction of wild fish in the water supply. Others like cooling the water temperature (Wood, 1974), or reduction of *F. columnaris* through adding significant numbers of competitive bacteria (e.g., *Citrobacter freundii*) to susceptible fish ponds (Chowdhury and Wakabayashi, 1989) do not sound very practical for a typical carp pond farm. Although positive results were published on the feasibility of vaccination against columnaris disease in channel catfish (Moore et al., 1990), a commercially available vaccine is not likely in the near future.

Amend (1970) reviewed the chemotherapy of columnaris disease. For systemic infections, sulphonamides or antibiotics are added to the food. Sulphamerazine and oxytetracycline are administered therapeutically in the feed in a two-stage regime: 220 mg/kg/day for 10 days followed by 50–75 mg/kg/day for 10 days.

4. Viral diseases

In the most detailed work on fish viruses and viral diseases 23 viruses that were isolated and the resulting diseases in fish are listed (Table 4). Two of them are from carp: *Rhab-*

Table 4
Viruses and viral diseases of carp

Virus	Disease	Reference
<i>Rhabdovirus carpio</i>	Spring viraemia of carp	Fijan (1972)
Herpesvirus	Carp pox	Fijan et al. (1983)
Iridovirus	Gill necrosis	Shchelkunov and Shchelkunova (1981)
Carp coronavirus		Wolf (1988)

dovirus carpio (causative agent of spring viraemia of carp) and *Herpesvirus cyprini* (causative agent of carp pox). Carp iridovirus is one of the 11 listed viral infections of indeterminate pathogenicity. Of 17 viruses that have been visualised but not yet isolated, one is the carp coronavirus (Wolf, 1988).

Spring viraemia of carp

Causative agent. Spring viraemia of carp (SVC) is an acute haemorrhagic and contagious viral infection of the common carp in Europe. Fijan et al. (1971) isolated a virus (*Rhabdovirus carpio*), proved its aetiological role in SVC and called the disease “spring viraemia of carp”. Before this time the disease was known as “infectious dropsy of carp” (IDC) and was considered to have either a bacterial or a viral aetiology. Two forms of IDC became recognised: an acute or ascitic form and a chronic or ulcerative form. Fijan et al. (1971) in their already cited work showed that *Rhabdovirus carpio* produced the acute form of what had been termed “IDC”. The chronic or ulcerative form was later named “carp erythrodermatitis” and was shown to be caused by a non-motile bacterium of the genus *Aeromonas* (Bootsma et al., 1977). The initial isolation of *Rhabdovirus carpio* was followed by confirming isolations in Czechoslovakia (Macura et al., 1973; Tesarcik, 1977) in Hungary (Békési and Szabo, 1977, 1979) and in Great Britain (Bucke and Finlay, 1979). During the 1980s, the protozoan aetiology of swimbladder inflammation (SBI) was proved (Csaba et al., 1984; Körting, 1982; Kovács-Gayer, 1983) and the possible role of SVCV in SBI was no longer considered.

Clinical signs. SVCV has the characteristics of Rhabdoviruses and the agent is pathogenic to carp of all ages. The disease becomes manifest when the water temperatures rise in the springtime. Moribund fish show dark colouration, low respiration, exophthalmia, inflamed and oedematous vent, pale gills and haemorrhages in the skin. Internally there are frequently haemorrhages in the viscera and airbladder, and peritonitis with serous or haemorrhagic exudate is mostly present in acute cases.

A clinical diagnosis of SVC is possible if serious mortality occurs among carp during spring or early summer and affected specimens show the key features of the behavioural changes and the above-described signs. For confirmative diagnosis the *Rhabdovirus carpio* should be isolated and identified serologically, or the viral antigen should be identified serologically in tissue sections.

Epizootology. Under natural conditions, most transmission is probably horizontal (Wolf, 1988), although vertical transmission may also take place in some situations (Békési and

Csontos, 1985). During outbreaks and several months thereafter, virus is shed by faeces and possibly urine (Ahne, 1979; Baudouy et al., 1980; Pfeil-Putzien, 1977). Blood-sucking parasites, like the carp louse (*Argulus foliaceus*) and the leech (*Piscicola geometra*) are vectors for SVCV. They both carried the virus passively, and multiplication did not occur (Ahne, 1978). The virus is transmitted by the water route and the gills were the primary portal of entry for SVCV.

Water temperature plays an important role both in the outbreak of the disease and the immunity acquired. Cases of SVC occur in ponds in springtime, mostly at water temperatures between 12 and 18°C and mortality ceases with the increase in water temperature. Mortality may sometimes continue up to 22°C and this temperature was therefore mentioned by Fijan (1972) as the upper thermal range of the disease. In experimentally infected carp kept under various controlled temperature conditions Baudouy et al. (1980) found the SVC mortality to be most severe at temperatures below 10–11°C; at 18°C and above the defence mechanisms were able to protect the fish from mortality. According to Ahne (1980) and Fijan and Matasin (1980) the infection of susceptible carp with SVCV at 20°C does not result in mortality: at these and higher temperatures the defence mechanisms are able to restrict virus replication and to eliminate it.

Treatment. Theoretically there are 5 basic control measures for SVC: (1) avoidance of sources of virus; (2) development of specific-pathogen-free (SPF) broodstock; (3) temperature manipulation (rearing fish above 20°C); (4) genetic selection for resistance to SVC (Kirpichnikov et al., 1972); (5) immunoprophylaxis. Practically all these control measures have their limitations: no methods to detect carrier state, unpracticality of control, lack of methods of chemical control, and quality problems with the only tested SVC vaccine (Tesarcik et al., 1984).

5. Environmental stress

As is known, most of the pathogens of common carp are facultative pathogens. This means that they are almost always present in the water surrounding the fish, but they can

Table 5
Environmental stress factors predisposing to diseases of carp

Disease	Environmental stress factors predisposing to disease
Parasite infestations (Costia, Trichodina)	Overcrowding of fry and fingerlings; low oxygen; excessive size variation among fish in ponds
Bacterial gill disease	Crowding; unfavourable environmental conditions such as chronic low oxygen, elevated ammonia, particulate matter in water
Columnaris disease	Crowding or handling during warm (above 15°C) water periods if carrier fish are present in the water supply; temperature increase to about 30°C; if the pathogen is present, even if it not crowded or handled
Haemorrhagic septicaemia (<i>Aeromonas</i> and <i>Pseudomonas</i> spp.)	Pre-existing protozoan infestations such as Costia, Trichodina; increased bacterial load in water; particulate matter in water; handling (especially after overwintering at low temperatures); crowding; low oxygen; chronic sublethal exposure to heavy metals; pesticides or PCBs
Spring viraemia of carp	Handling after overwintering at low temperatures, rapid warming of water temperature

After Wedemeyer and McLeay (1981) with modification.

Table 6
Typical changes in diagnostic parameters of carp under the effect of environmental factors

FISH		Adrenaline	Noradrenaline	Cortisol	Glucose	Hematocrit	Leukocrit	Hemoglobin	Total protein	Calcium	Chlorid	GOT	GPT	GLDH	ACHE	ATP	Growth	Mortality	References
ENVIRONMENT																			
Natural - factors	heating, 5 oC	nt	nt		▲							▲	nt	nt	▲				Jeney G. et al., 1984a
	cooling, 5 oC	nt	nt		▲			nt		▲		nt	nt	▲				Jeney G. et al., 1984a	
	heating, 15 oC	▲			▲			nt				▲	▲					Jeney Z. et al., 1984	
	cooling, 15 oC	nt	nt		▲			nt					nt					Jeney Z. et al., 1984	
	gradual cooling	▲			▲	nt	nt	nt	nt	▲		▲						Jeney Z. et al., 1985	
	hyperoxia				nt		▲	▲	nt			▲	▲					Jeney, 1989	
	hypoxia				▲		▲	▲	▲			▲	▲					Jeney, 1989	
	anoxia				▲	nt		▲				▲				▲		Jeney, 1989	
	ammonia, sublethal	▲							▲				▲	▲				Jeney et al., 1992	
	Antropogenic factors																		
handling	anaesthetic	▲	▲	▲	▲			nt	nt										Jeney, 1989
	asphyxia	nt	nt	▲	▲	▲	▲	nt	nt	▲									Jeney et al., 1986
	propagation	▲	▲	▲	▲	▲	▲	nt	nt	▲	▲	▲	▲				▲		Jeney and Jeney, 1992
	insecticide	▲	▲	▲	▲	▲	▲	nt	nt			▲	▲						Jeney and Jeney, 1986
	bacterial infection	▲	▲	▲	▲	▲	nt	nt	▲			▲	nt						Jeney et al., 1984b
	viral infection					▲		▲	▲			▲	▲						Jeney et al., 1990

Remarks:

▲ increase
 ▼ decrease
 nt non typical change

cause disease only under certain circumstances, namely when the resistance of fish is decreased by adverse changes in the aquatic environment. The theory of Snieszko (1974) on the primary role of environmental stress in the outbreak of fish diseases is especially important in warmwater aquaculture, including carp culture, where mainly surface waters are used and the basic water quality parameters are subject to a wide range of fluctuations, as well as where the level of management is generally low.

Due to this phenomenon it is not surprising that several diseases of carp are typically “stress-mediated”: certain parasite infestations (costiosis, trichodinosis), bacterial gill disease, flexibacteriosis and spring viraemia of carp. Environmental stress factors predisposing to diseases in carp are listed in Table 5.

For a better understanding of the role of environmental stress factors in the outbreak of fish diseases it is essential to study the response of the fish organism. In the scheme suggested for the stress response of fish (Peters, 1979; Mauzeaud and Mauzeaud, 1981) the most important physiological and biochemical changes occurring at the primary, secondary and tertiary levels were summarised. Certain of these changes can also be employed to detect diseases at early stages or sublethal effects of environmental contaminants. A summary of the physiological tests for stress assessments of salmonids was published by Wedemeyer and Yasutake (1977). A similar system for tilapia (*Sarotherodon aureus*) and silver carp (*Hypophthalmichthys molitrix*) was presented by Bejerano (1984). Data are available on the “normal” ranges or values of some of these parameters for common carp (Svobodova, 1987). The limits of the “normal” values of these parameters in fish can only be overcome by a description of the typical changes under the effect of different environmental factors, compared to the changes in “non-treated” groups. For the assessment of the effects of environmental stress on common carp a system of clinical diagnostic parameters was set up and tested (Jeney and Jeney, 1986, 1987; Jeney, G. et al., 1992; Jeney, Z. et al., 1992a,b). Using this system, different environmental stresses of both natural and antropogenic origin were investigated. Typical changes (increase or decrease, compared to the control) in these parameters are shown in Table 6. As can be seen, most of the natural and anthropogenic environmental factors studied, even though typical for “normal” technological conditions, causing marked changes at all three levels of stress response of the carp.

6. Prospective methods of prevention and treatment of diseases of common carp

Further development of carp culture will be accompanied by several changes that have well-established incidences of pathological conditions: increase in culture densities, frequent degradation of environmental quality, mixing of populations of different origin and manipulations etc. All these factors are increasing the probability of the advent and seriousness of diseases.

Research on fish pathology has allowed the progressive setting-up of a rather large variety of possible interventions: specific diagnostic methods, sanitary prophylaxis and disinfection, vaccination, chemotherapy, immunomodulation and genetics of disease resistance will continue to supply new possibilities to breeders (Fig. 1). However, several constraints often make such interventions difficult and expensive; thus prophylaxis based on strict sanitary isolation, which is relatively easy in terrestrial species, is often more difficult in the aquatic

Limitations

Few pathogens
Limited market

Limitations

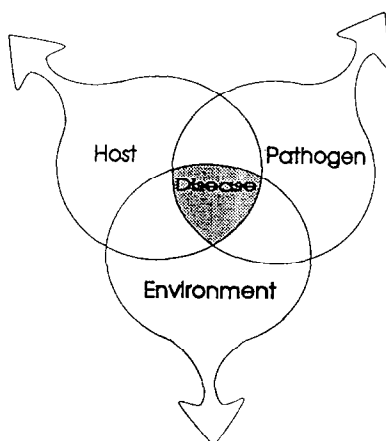
Specific diagnostic methods
Registered drugs
Improper use of drugs
Resistance
Environmental issues
Human health concerns

Possibilities

Vaccines
Immunostimulants
Genetic resistance

Possibilities

Antibiotics
Drugs



Possibilities
Quality control

Limitations

Water quality
Management

Fig. 1. Possibilities and limitations to minimize the risks of diseases of fish. After Anderson (1992) with modification.

environment where the water, other fish species or even invertebrates or other vertebrates constitute potential contaminating agents which are difficult to eliminate (Chevassus and Dorson, 1990; Anderson, 1992).

Antibiotic therapy, mostly applied orally, has more and more to cope with resistance in selected bacteria due to repeated treatments. In theory, vaccination is the most valuable approach, but, so far, effective vaccines are available only for a limited range of pathogens and not for those infecting common carp. In the case of viral diseases, attenuated vaccines are not authorised and killed vaccines are expensive to produce. Both kinds of vaccines also prevent any serological differentiation between vaccinated and contaminated fishes. Research on a recombinant subunit vaccine against spring viraemia of carp aimed at the production of a low-cost, completely safe and effective vaccine could provide a possible solution (Rossius and Thiry, 1993).

The small-scale market is a special challenge for any products to be introduced into aquaculture (De Kinkelin and Michel, 1984).

These limitations raise a question about the future of chemotherapy. Although chemotherapeutic agents are available against a wider range of pathogens than vaccines, they also have limitations in terms of the pathogen groups against which they are effective. Chemotherapy is also a palliative rather than a final solution to problems of disease and its use in the aquatic environment is not free from practical problems. For example, attention is being drawn increasingly to the potential hazards that intensive use of drugs could represent for the environment and for human health.

Use of immunomodulators in fish culture offers a wide range of attractive methods for inducing and building up protection against diseases. Immunostimulants may be used alone, inducing elevated activities in the non-specific defence mechanisms (Anderson, 1992; Jeney and Anderson, 1993). During recent years several substances have been tested in carp to enhance non-specific defence mechanisms and to build up protection against a wide range of pathogens. Levamisole treatment of carp fingerling stimulated growth and reduced natural mortality due to losses (Siwicki and Korwin-Kossakowski, 1988). Vitamin C is a popular immunostimulant added to diets of certain animals. Glucans, long-chain polysaccharides extracted from yeasts, are good stimulators of non-specific defence mechanisms in fish. Schizophyllan, scleroglucan, and lentinan were evaluated in carp for their ability to enhance protection against *E. tarda* and *A. hydrophila* (Yano et al., 1989, 1991). In fish injected with these polysaccharides increased phagocytic activity and protection against the pathogen were observed. Research on immunostimulants for use in food fish, including carp is in progress.

Due to the above-mentioned situation, fish geneticists and pathologists are interested in the search for intrinsic resistance factors protecting the individual during part or the whole of its biological cycle in the course of aquaculture development (Chevassus and Dorson, 1990). Promising data are available on selective breeding of carp with high tolerance to particular diseases (Kirpichnikov and Faktorovich, 1972; Kirpichnikov et al., 1972; Kirpichnikov, 1986; Sövényi et al., 1989; Houghton et al., 1991), or to stressful environmental conditions (Jeney et al., 1995), as well as on selecting for traits which have proven to be genetically correlated to disease resistance: cortisol – in Atlantic salmon and rainbow trout (Refstie, 1986; Fevolden et al., 1991, 1993).

Acknowledgements

The authors express their thanks to Dr. Kálmán Molnár and Dr. József Szokolczai for critical discussions and comments on the manuscript.

References

- Ahne W., 1973. Zellkulturen an verschiedenen Süßwasserteleostengeweiben und Untersuchungen über die Ätiologie der Schwimmblasenentzündung der Karpfen. Thesis, Ludwig Maximilian University, Munich.
- Ahne, W., 1978. Uptake and multiplication of spring viremia of carp virus in carp, *Cyprinus carpio* L. J. Fish Dis., 1: 265–268.

- Ahne, W., 1979. Untersuchungen über die acute Form der infektiösen Bauchwassersucht bei Cypriniden (*Cyprinus carpio*, *Ctenopharyngodon idella*). Habilitationsschrift zur Erlangung der Venia Legendi. Ludwig-Maximilian University, Munich.
- Ahne, W., 1980. *Rhabdovirus carpio* – Infektion beim Karpfen (*Cyprinus carpio*): Untersuchungen über die reaktionen des Wirtsorganismus. Fortschr. Vetrinärmed., 30: 180–183.
- Alderman, D.J., 1985. Malachite green: a review. J. Fish Dis., 8: 289–298.
- Alderman, D.J., 1991. Malachite green and alternatives as therapeutic agents. In: N. De Pauw and J. Joyce (Editors), Aquaculture and Environment. Eur. Aqua Spec. Publ., 16. Ghent, Belgium.
- Amend, D.F., 1970. Myxobacterial infections of salmonids: prevention and treatment. In: S.F. Snieszko (Editor), Proceedings, Symposium on Diseases of Fishes and Shellfishes American Fisheries Society, Washington, DC, pp. 258–265.
- Anderson, D.P., 1992. Immunostimulants, adjuvants, and vaccine carriers in fish: applications to aquaculture. Annu. Rev. Fish Dis., 2: 281–307.
- Ansary, A., Haneef, R.M., Torres, J.L. and Yadov, M., 1992. Plasmids and antibiotic resistance in *A. Hydrophila*. J. Fish Biol., 15: 191–196.
- Arshanica, N.M., 1969. Data about epizootiology, diagnosis and prevention of swimbladder inflammation in carp. Izv. Gosniorch, 69: 15–46 (in Russian).
- Austin, B. and Austin, D.A., 1987. Bacterial Fish Pathogens. Ellis Horwood, Chichester, UK, pp. 11–195.
- Bachmann, P.A. and Ahne, W., 1973. Isolation and characterisation of agent causing swimbladder inflammation in carp. Nature, 244: 235–237.
- Baska, P. and Molnár, K., 1988. Observations on the blood stages of *Sphaerospora* spp. Myxosporea in cyprinid fishes. Dis. Aquat. Org., 5: 23–28.
- Baudouy, A.M., Dantin, M. and Merle, G., 1980. Experimental infection of susceptible carp fingerlings with spring viremia of carp virus under wintering environmental conditions. In: W. Ahne (Editor), Fish Disease. Third COPRAQ Session. Springer-Verlag, Berlin, pp. 23–27.
- Becker, C.D. and Fujihara, M.P., 1978. The bacterial pathogen *Flexibacter columnaris* and its epizootology among Columbia river fish. Am. Fish. Soc. Monograph 2: 92.
- Bejerano, I., 1984. Detection and control of stress conditions in warm water aquaculture. In: H. Rosental and S. Sarig (Editors), Research on Aquaculture. Europ. Maricult. Soc. Spec. Publ. No. 8, pp. 73–92.
- Békési, L. and Szabo, E., 1977. The first isolation of *Rhabdovirus carpio* from Hungarian carps. Magy. Allatorv. Lapja., 32: 369–371 (in Hungarian).
- Békési, L. and Szabo, E., 1979. A rhabdovirus isolated from carps in Hungary. Experimental infection of carps and resistance of the virus. Acta Microbiol. Acad. Sci. Hung., 26: 193–197.
- Békési, L. and Csontos, L., 1985. Isolation of spring viraemia of carp virus from asymptomatic broodstock carp, *Cyprinus carpio* L. J. Fish Dis., 8: 471–472.
- Belland, R.J. and Trust, T.J., 1988. DNA: DNA reassociation analysis of *Aeromonas salmonicida*. J. Gen. Microbiol., 134: 307–315.
- Bernardet, J.F. and Grimont, P.A.D., 1989. Deoxyribonucleic acid relatedness and phenotypic characterisation of *Flexibacter psychrophilus* sp. nov., nom. rev., and *Flexibacter maritimus* Wakabayashi, Hikida and Masumura, 1986. Int. J. Syst. Bacteriol., 39: 346–354.
- Bohm, K.H., Fuhrmann, H., Schlotfeldt, H.J. and Korting, W., 1986. *Aeromonas salmonicida* from salmonids and cyprinids – serological and cultural identification. J. Vet. Med., 33: 777–783.
- Bootsma, R., 1973. Infections with *Saprolegnia* in pike culture. Aquaculture, 2: 385–394.
- Bootsma, R., 1975. Studies on the aetiology of carp erythrodermatitis. Paper presented at: FAO, EIFAC Meet. Fish. Dis., Sept.29–Oct. 2, Zagreb, Yugoslavia. Draft report, FAO, Rome, p. 6.
- Bootsma, R. and Clerx, J.P.M., 1976. Columnaris disease of cultured carp, *Cyprinus carpio* L.: characteristics of the causative agent. Aquaculture, 7: 371–384.
- Bootsma, R. and Blommaert, J., 1978. Ätiologie der Erythrodermatitis beim Karpfen, *Cyprinus carpio* L. Fish Umwelt, 5: 25–36.
- Bootsma, R., Fijan, N.N. and Blommaert, J., 1977. Isolation and preliminary identification of the causative agent of carp erythrodermatitis. Vet. Arh. Zagreb., 47: 291–302.
- Boulanger, Y., Lallier, R. and Cousineau, G., 1977. Isolation of enterotoxigenic *Aeromonas* from fish. Can. J. Microbiol., 23: 1161–1164.
- Bucke, D. and Finlay, J., 1979. Identification of spring viraemia in carp (*Cyprinus carpio* L.) in Great Britain. Vet. Rec., 104: 69–71.

- Bullock, G.L. and Herman, R.L., 1985. *Edwardsiella* infections of fishes. US Fish Wildl. Dis. Leaflet, 71 pp.
- Chang, B.J. and Bolton, S.M., 1987. Plasmids and resistance to antimicrobial agents in *Aeromonas sobria* and *Aeromonas hydrophila* clinical isolates. Antimicrob. Agents Chemother., 31: 1281–1282.
- Chevassus, B. and Dorson, M., 1990. Genetic resistance to disease in fishes. Aquaculture, 85: 83–107.
- Chowdhury, M.B.R. and Wakabayashi, H., 1988. Effects of sodium, potassium, calcium and magnesium ions on the survival of *Flexibacter columnaris* in water. Fish Pathology, 23: 231–235.
- Chowdhury, M.B.R. and Wakabayashi, H., 1989. Effects of competitive bacteria on the survival and infectivity of *Flexibacter columnaris*. Fish Pathol., 24: 9–15.
- Csaba, G., 1976. An unidentifiable extracellular sporozoan parasite from the blood of the carp. Parasitol. Hung., 9: 21–24.
- Csaba, G., Kovacs-Gayer, E., Békési, L., Bucsek, M., Szakolczai, J. and Molnár, K., 1984. Studies into the possible protozoan aetiology of swim bladder inflammation in carp fry. J. Fish Dis., 7: 39–56.
- De Kinkelin, P. and Hattenberger, A.M., 1986. Les dominantes pathologiques chez la carpe et les Cyprinides d'élevage. In: R. Billard and J. Marcel (Editors), Aquaculture of Cyprinids. INRA, Paris, pp. 413–428.
- De Kinkelin, P. and Michel, C., 1984. Symposium on Fish Vaccination, 20–22 February 1984, OIE, Paris, 248 pp.
- Dykova, I. and Lom, J., 1982. *Sphaerospora renicola* n.sp., a myxosporean from carp kidney, and its pathogenicity. Z. Parasitenkd., 68, 259–268.
- Donta, S.T. and Haddow, A.D., 1978. Cytotoxic activity of *Aeromonas hydrophila* Infect. Immun., 21: 989–993.
- Egusa, S., 1965. The existence of primary infectious disease in the so-called "fungus disease" in pond-reared eels. Bull. Jpn. Soc. Sci. Fish., 31: 517–526.
- FAO, 1992. Aquaculture production (1985–1990). FAO Fisheries Circular No. 815, Revision 4, FIDI/C 815 Rev. 4, Statistical Tables. 206 pp.
- Farkas, J., 1985. Filamentous *Flavobacterium* sp. isolated from fish with gill disease in cold water. Aquaculture, 44: 1–10.
- Farkas, J. and Oláh, J., 1986. Gill necrosis – a complex disease of carp. Aquaculture, 58: 17–26.
- Fevolden, S.E., Refstie, T. and Roed, K.H., 1991. Selection for high and low stress response in Atlantic salmon (*Salmo salar*) and rainbow trout (*Oncorhynchus mykiss*). Aquaculture, 95: 53–65.
- Fevolden, S.E., Nordmo, R., Refstie, T. and Roed, K.H., 1993. Disease resistance in Atlantic salmon (*Salmo salar*) selected for high or low responses to stress. Aquaculture, 109: 215–224.
- Fijan, N.N., 1968. The survival of *Chondrococcus columnaris* in waters of different quality. Bull. Off. Int. Epizoot., 69: 1158–1166.
- Fijan, N., 1972. Infectious dropsy in carp – a disease complex. In: L.E. Mawdesley-Thomas (Editor), Diseases of Fish. Symposium of the Zoological Society of London. Academic Press, London, pp. 39–51.
- Fijan, N. and Matasin, Z., 1980. Spring viraemia of carp: preliminary experiments on vaccination by exposure to virus in water. Vet. Arch., 50: 215–220.
- Fijan, N., Petrinc, Z., Sulimanovic, D. and Zwillenberg, L.O., 1971. Isolation of the viral causative agent from the acute form of infectious dropsy of carp. Vet. Arch., 41: 125–138.
- Fijan, N., Petrinc, Z., Stancl, Z., Dorson, M. and Le Berre, M., 1977. Hyperimmunization of carp with *Rhabdovirus carpio*. Bull. Off. Int. Epizoot., 87: 439–440.
- Fijan, N., Sulimanovic, D., Berzotti, M., Muzinic, D., Zwillenberg, L.O., Chilmonczyk, S., Vautherot, J.F. and De Kinkelin, P.D., 1983. Some properties of the *Epithelioma papulosum cyprini* (EPC) cell line from carp *Cyprinus carpio*. Ann. Virol. (Paris), 134E: 207–220.
- Fish, F.F. and Rucker, R.R., 1943. Infectious disease of Pacific salmon. Trans. Am. Fish. Soc., 83: 297–312.
- Foster, F.J. and Woodbury, L., 1936. The use of malachite green as a fish fungicide and antiseptic. Prog. Fish-Cult., 18: 7–9.
- Fraire, A.J., 1978. *Aeromonas hydrophila* infection. J. Am. Med. Assoc., 239: 192.
- Frerichs, G.N., 1993. Mycobacteriosis: nocardiosis. In: V. Inglis, R.J. Roberts and N.R. Bromage (Editors), Bacterial Disease of Fish. Blackwell Scientific Publications, Oxford, pp. 219–234.
- Fryer, J.L. and Rohovec, J.S., 1993. Bacterial diseases of fish. In: J.A. Couch and J.W. Fournie (Editors), Pathology of Marine and Estuarine Organisms. CRC Press, Boca Raton, FL, pp. 53–83.
- Grishchenko, L.I., 1967. The swimbladder inflammation of carps. Veterinariya (Moscow), 43, 78–79 (in Russian).
- Hanson, L.A. and Grizzle, J.M., 1985. Nitrite-induced predisposition of channel catfish to *Flexibacter columnaris* infection. Prog. Fish-Cult., 47: 98–101.

- Hazen, T.C., Fliermaus, C.P., Hirsch, R.P. and Esch, G.W., 1978. Prevalance and distribution of *Aeromonas hydrophila* in the United States. *Appl. Environ. Microbiol.*, 36: 731–738.
- Hiney, M.P. and Smith, P.R., 1993. Initial investigations into the fungicidal activity of stabilized chlorine dioxide against *Saprolegnia* in a freshwater hatchery. *Bull. Eur. Assoc. Fish Pathol.*, 13: 123–126.
- Hofer, B., 1904. *Handbuch der Fischkrankheiten*. Munich.
- Holt, R.A., Sanders, J.E., Zinn, J.L., Fryer, J.L. and Pilcher, K.S., 1975. Relation of water temperature to *Flexibacter columnaris* infection in steelhead trout (*Salmo gairdneri*), coho (*Oncorhynchus kisutch*) and chinook (*O. tshawytscha*) salmon. *J. Fish. Res. Board Can.*, 32: 1553–1559.
- Houghton, G., Wiergertjes, A., Groeneveld, A. and Van Muiswinkel, W.B., 1991. Differences in resistance of carp, *Cyprinus carpio* L., to atypical *Aeromonas salmonicida*. *J. Fish Dis.*, 14, 33–341.
- Inglis, V. and Hendrie, M.S., 1993. *Pseudomonas* and *Alteromonas* infections. In: V. Inglis, R.J. Roberts and N.R. Bromage (Editors), *Bacterial Disease of Fish*. Blackwell Scientific Publications, Oxford, pp. 169–174.
- Jeney, G. and Anderson, D.P., 1993. Glucan injection or bath exposure given alone or in combination with a bacterin enhance the non-specific defence mechanisms in rainbow trout (*Oncorhynchus mykiss*). *Aquaculture*, 116: 315–329.
- Jeney, G., Péteri, A., Jeney, Z. and Oláh, J., 1984a. Effect of artificial propagation following thermal stress on the activity of serum transaminases (GOT, GPT) of carp (*Cyprinus carpio* L.). *Acta Biochim. Biophys. Acad. Sci. Hung.*, 19: 137.
- Jeney, G., Nemcsók, J. and Oláh, J., 1984b. Transaminase enzyme activity of cyprinid fish depending on environmental factors and bacterial infection. *Symp. Biol. Hung.*, 23: 201–207.
- Jeney, G., Jeney, Z. and Oláh, J., 1990. Effect of Rhabdovirus infection on selected blood parameters of wells (*Silurus glanis* L.). *Aquacult. Hung.*, 6: 153–160.
- Jeney, G., Nemcsók, J., Jeney, Z. and Oláh, J., 1992. Acute effect of sublethal concentration of NH₃ on common carp (*Cyprinus carpio* L.). II. Effect of ammonia on GOT, GPT, GIDH enzymes activities and ATP level in blood. *Aquaculture*, 104: 149–156.
- Jeney, Z., 1989. Effects of stress factors of fish culture on some biochemical and hysiological parameters of carp. Ph.D. Thesis, University of Eötvös, Budapest, 147 pp.
- Jeney, Z. and Jeney, G., 1986. Studies on the effect of trichlorphon on different biochemical and physiological parameters of common carp (*Cyprinus carpio*). *Aquacult. Hung.*, 5: 79–90.
- Jeney, Z. and Jeney, G., 1987. A system of diagnostic parameters for detection of environmental impact of fish. In Abstracts, 3rd International Conference of EAAP, p. 45.
- Jeney, Z. and Jeney, G., 1992. Primary and secondary stress responses of common carp (*Cyprinus carpio* L.) caused by artificial propagation. In: Z. Adamek and M. Faljshans (Editors), *Proceedings of the Scientific Conference Fish Reproduction '92*, pp. 37–41.
- Jeney, Z., Jeney, G., Oláh, J., Nemcsók, J. and Horváth, J.S., 1984. Effect of thermal stress on noradrenaline level in different organs, serum glucose level and serum GOT, GPT, LDH and cholinesterase activity of common carp (*Cyprinus carpio* L.). In: Abstracts, 16th Meeting of the Federation of European Biochemical Societies, Moscow, USSR, p. 440.
- Jeney, Z., Jeney, G., Oláh, J. and Nemcsók, J., 1985. Thermal pollution influences physiological status of fish. Summary of Papers of 12th Scientific Session on Environment Analysis, p. 35.
- Jeney, Z., Jeney, G., Oláh, J., Siwicki, A. and Dankó, I., 1986. Propanidid, a new anaesthetic for use in fish propagation. *Aquaculture*, 54: 149–156.
- Jeney, Z., Jeney, G. and Maule, A.G., 1992a. Cortisol measurements in fish. In: J.S. Stolen, T.C. Fletcher, D.P. Anderson, S.L. Kaatari and A.R. Rowley (Editors), *Techniques in Fish Immunology*. SOS Publ., Fair Haven, NJ.
- Jeney, Z., Nemcsók, J., Jeney, G. and Oláh, J., 1992b. Acute effect of sublethal concentration of NH₃ on common carp (*Cyprinus carpio* L.). I. Effect of ammonia on level of adrenaline and noradrenaline in different organs of carp. *Aquaculture*, 104: 139–149.
- Jeney, Z., Papp, Z., Jeney, G. and Gorda, S., 1995. Stress sensitivity of four genetically different strains of common carp (*Cyprinus carpio* L.). *Aquaculture*, 129: 203 (Abstract).
- Kanaev, A.I. and Kuzmin, G.G., 1970. The swim bladder inflammation of carp and the control measures against the disease. *Russelchozisdar* (Moscow), pp. 1–42 (in Russian).
- Kanaev A.I., Lobuncov, K.A. and Naumova, A.M., 1967. Microflora and parasitofauna of carps diseased in swimbladder inflammation. *Rybn. Chosj.* (Moscow), 43: 16–18 (in Russian.).

- Kirpichnikov, V.S., 1986. Genetic Basis of Fish Selection. Springer-Verlag, Berlin, 127 pp.
- Kirpichnikov, V.S. and Faktorovich, K.A., 1972. Increase in the resistance of carp to dropsy by means of breeding. II. The course of the selection and evaluation of the breeding groups selected. Sov. Genet., 8: 592–600 (in Russian).
- Kirpichnikov, V.S., Faktorovich, K.A. and Suleymanian, V.S., 1972. Increasing the resistance of carp to dropsy by breeding. I. Methods of breeding for resistance. Sov. Genet., 8: 306–312 (in Russian).
- Körting, W., 1982. Protozoan parasites associated with swimbladder inflammation (SBI) in young carp. Bull. Eur. Assoc. Fish Pathol., 2: 25–28.
- Körting, W., Hoffman, R., Neukirch, M. and Fuhrman, H., 1984. Myxosporidien bedingte Schwimmblasenentzündung bei Karpfenbrut. Berl. Münch. Tierärztl. Wochenschr., 97: 99–104.
- Kocylowski, B.J., Antychowicz, J. and Zelazny, J., 1970. Studies on the etiology and pathogenesis of carp swimbladder inflammation. Riv. Ital. Piscicult. Ittiopatol. 3: 59–63.
- Kovács-Gayer, E., 1983. Histopathological studies on protozoan swimbladder inflammation of common carp fry. Parasitol. Hung., 16: 39–42.
- Kovács-Gayer, E., Csaba, G., Békési, L., Bucsek, M., Szokolczai, J. and Molnár, K., 1982. Studies on the protozoan aetiology of swim-bladder inflammation in common carp fry. Bull. Eur. Assoc. Fish. Pathol., 2, 22–24.
- Landsberg, J.H., 1986. Swimbladder inflammation and kidney sphaerosporosis in carp (*Cyprinus carpio*) in Israel. First report. Bull. Eur. Assoc. Fish Pathol., 6: 36–37.
- Landsberg, J.H., 1991. Parasites and associated diseases in fish in warm water aquaculture, with special emphasis on intensification. In: M. Shilo and S. Sarig (Editors), Fish Culture in Warm Water Systems: Problems and Trends. CRC Press, Cleveland, OH, pp. 195–252.
- Lom, J., Dykova, I. and Palaskova, M., 1983. Unidentified mobile protozoans from the blood of carp and some unsolved problems of myxosporean life cycles. J. Protozool., 30: 497–508.
- Lom, J., Palaskova, M. and Dykova, I., 1985. Notes on kidney infecting species of the genus *Sphaerospora thelohan* (Myxosporidia), including a new species *S. gobionis* sp. nov., and on myxosporean life cycle stages in the blood of some freshwater fish. J. Fish Dis., 8: 221–232.
- Lom, J., Pike, A.W. and Feist, S.W., 1991. Myxosporean vegetative stages in the choroidal rete mirabile of *Gasterosteus aculeatus* infected with *Myxobolus gasterostei* and *Sphaerospora elegans*. Dis. Aquat. Org., 11: 67–72.
- MacFarlane, R.D., Bullock, G.L. and McLaughlin, J.J.A., 1986. Effects of five metals on susceptibility of striped bass to *Flexibacter columnaris*. Trans. Am. Fish. Soc., 115: 227–231.
- Macura, B., Dedek, L. and Tesarcik, J., 1973. The positive cytopathic effect from case of spring viremia of carp. Bul. VURH Vodn., 9: 7–14 (in Czech).
- Markiewicz, F., 1966. Swimbladder inflammation – a new disease of carp. Gospod. Rybn., 18: 8–9 (in Polish).
- Mattheis, T.H. and Kulov, H., 1967. Die Schwimmblasenerkrankung des Karpfens. Dtsch. Fisch.-Z., 14: 77–86.
- Mauzeaud, M.M. and Mauzeaud, F., 1981. In: A.D. Pickering (Editor), Adrenergic Responses to Stress in Fish. Academic Press, London–New York, pp. 49–75.
- Mitchell, A.J. and Plumb, J.E., 1980. Toxicity and efficacy of furanase on channel catfish infected experimentally with *Aeromonas hydrophila*. J. Fish Dis., 3: 93–100.
- Molnár, K., 1980a. Renal sphaerosporosis in the common carp, *Cyprinus carpio* L. J. Fish Dis., 3: 11–19.
- Molnár, K., 1980b. Sphaerosporosis, a new kidney disease of the common carp. In: W. Ahne (Editor), Fish Diseases, Third COPRAQ-Session, pp. 156–164. Springer Verlag, Berlin–Heidelberg–New York.
- Molnár, K., 1993. The occurrence of *Sphaerospora renicola* K-stages in the choroidal rete mirabile of the common carp. Folia Parasitol., 40: 175–180.
- Molnár, K. and Szokolczai, J., 1981. The Book of Fish Diseases. Mg. Kiado, Budapest, pp. 283 (in Hungarian).
- Molnár, K. and Szokolczai, J., 1992. Fish Diseases. Lectures. Veterinarian University, Budapest, 163 pp. (in Hungarian).
- Moore, A.A., Eimers, M.E. and Cardell, M.A., 1990. Attempts to control *Flexibacter columnaris* epizootics in pond-reared channel catfish by vaccination. J. Aquat. Anim. Health, 2: 109–111.
- New, M.B. and Csavas, I., 1993. Aquafeeds in Asia – a regional overview. In: M.B. New, A.G.J. Tacon and I. Csavas (Editors), Farm-Made Aquafeeds, Regional Expert Consultation on Farm-Made Aquafeeds. Prayurawong Printing, Bangkok, pp. 1–24.
- Neciporenko, J.D., Rudenko, A.P., Karpenko, J.M. and Advosed, B.S., 1963. The swimbladder inflammation of carp as the complication of dropsy. Thesis IV. Vsesajuz. Societs. po Boleznjam Ryb., Moscow, pp. 74–75 (in Russian).

- Neish, G.A. and Hughes, G.C., 1980. Fungal Diseases in Fish. T.F.H. Publ., Neptune, NJ.
- Nelson, N.C., 1974. A review of the literature on the use of malachite green in fisheries. U.S. Fish Wildl. Serv. Lit. Rev., 74–111.
- Nord, C.J. Sjöberg, L., Neish, G.A., 1977. Observations on saprolegniasis of adult sockeye salmon. J. Fish Biol., 10: 513–522.
- Nord, C.J., Sjöberg, L., Wadstrom, T. and Wretling, B., 1975. Characterization of three *Aeromonas* and nine *Pseudomonas* species by extracellular enzymes and haemolysins. Med. Microbiol. Immunol., 161: 79–87.
- Odening, K., 1987. *Sphaerospora renicola* (Myxosporidia) der Erreger der protozoaren Schwimmblasenentzündung des Jungkarpfens. Angew. Parasitol., 28: 16.
- Oláh, J. and Farkas, J., 1978. Effect of temperature, pH, antibiotics, formalin and malachite green on the growth and survival of *Saprolegnia* and *Achlya* parasitic on fish. Aquaculture, 13: 273–288.
- Otte, E., 1966. Die eitrige Schwimmblasenentzündung des Karpfens. Wien. Tierärztl. Monatsschr., 53: 401–403.
- Pacha, R.E. and Ordal, E.J., 1970. Myxobacterial diseases of salmonids. In: S.F. Snieszko (Editors), A Symposium on Diseases of Fish and Shellfish. American Fisheries Society, Washington, DC, pp. 243–247.
- Paperna, I., 1991. Disease caused by parasites in the aquaculture of warm water fish. Annu. Rev. Fish Dis., 1: 155–194.
- Peters, G., 1979. Zur Interpretation des Begriffs "Stress" beim Fisch. Fisch Umwelt, 7: 23–45.
- Pfeil-Putzien, C., 1977. New results in the diagnosis of spring viraemia of carp caused by experimental transmission of *Rhabdovirus carpio* with carp louse (*Argulus foliaceus*). Bull. Off. Int. Epizoot., 87: 457.
- Pickering, A.D. and Christie, P., 1980. Sexual differences in the incidence and severity of ectoparasitic infestation of the brown trout. J. Fish Biol., 16: 669–683.
- Pickering, A.D. and Richards, R.H., 1980. Factors influencing the structure, function and biota of the salmonid's epidermis. Proc. R. Soc. Edinb., 79B: 93–104.
- Pickering, A.D. and Willoughby, L.G., 1982. *Saprolegnia* infections of salmonid fish. In: R.J. Roberts (Editor), Microbial Diseases of Fish, Academic Press, London, pp. 271–298.
- Plehn, M., 1912. Eine neue Karpfenkrankheit und ihr Erreger: *Branchiomyces sanguinis*. Zbl. Bakteriell., 62: 129–134.
- Refstie, T., 1986. Genetic differences in stress response in Atlantic salmon and rainbow trout. Aquaculture, 57: 374 (Abstract).
- Reichenbach-Klinke, H.H., 1972. Some aspects of mycobacterial infections in fish. Symp. Zool. Soc. London, 30: 17–24.
- Roberts, R.J., 1993. Motile aeromonad septicaemia. In: V. Inglis, R.J. Roberts and N.R. Bromage (Editors), Bacterial Disease of Fish. Blackwell Scientific Publications, Oxford, pp. 143–156.
- Rossius, T.M. and Thiry, M., 1993. Towards a recombinant subunit vaccine against spring viraemia of the carp virus. In: Symposium on the Carp, Budapest, Sept. 6–9, 1993, p. 112.
- Roth, W., 1922. Krankheiten der Aquarienfische und ihre Bekämpfung. Stuttgart.
- Salton, R. and Schnick, S., 1973. *Aeromonas hydrophila* peritonitis. Cancer Chem. Rep., 57: 489–491.
- Schäperclaus, W., 1979. Fischkrankheiten. Akademie-Verlag, Berlin.
- Scott, W.W. and O'Bier, A.H., 1962. Aquatic fungi associated with disease fish and fish eggs. Prog. Fish-Cult., 24: 3–15.
- Shchelkunov, I.S. and Shchelkunova, T.I., 1981. Results of virological studies of gill necrosis diseased carps. In: J. Oláh, K. Molnár and Z. Jeney (Editors), Proceedings, International Seminar on Fish, Pathogens and Environment in European Aquaculture. Kellas, Budapest, pp. 466–482 (in Russian).
- Siwicki, A.K. and Korwin-Kossakowski, M., 1988. The influence of levamisole on the growth of carp (*Cyprinus carpio* L.) larvae. J. Appl. Ichthyol., 4: 178–181.
- Snieszko, S.F., 1974. The effects of environmental stress on outbreaks of infectious diseases of fishes. J. Fish Biol., 6: 197–208.
- Sővényi, J.F., Bercsényi and M., Bakos, J., 1988. Comparative examination of susceptibility of two genotypes of carp (*Cyprinus carpio* L.) to infection with *Aeromonas salmonicida*. Aquaculture, 70: 301–308.
- Sugimoto, N., Kashiwaga, S. and Matsuda, T., 1981. Pathogenic relation between columnaris disease in cultured eel and the formula feeds. Bull. Jpn. Soc. Sci. Fish., 47: 716–725.
- Svobodova, Z., 1987. Toxicology of Aquatic Animals. MZVZ CSR a Cesky rybarsky svaz. Prague, 231 pp. (in Czech).
- Szokolczai, J., 1967. Untersuchungen der Schwimmblasenentzündung bei Karpfen anhand von zwei Fällen in Ungarn. Zeitschr. Fisch., 15: 139–151.

- Ter Höfte, B.B., Körting, W. and Lehman, J., 1984. "C- und K-Protozoen". Endoparasiten unsicherer systematischer Zuordnung bei jungen Karpfen (*Cyprinus carpio* L.). Fisch Umwelt, 13: 89–99.
- Tesarcik, J., 1977. Studies in the relations between air bladder inflammation and infectious dropsy in carps. Pr. VURH Vodn., 10: 75–90.
- Tesarcik, J., Macura, B., Rehulka, J., Hrdonka, M. and Konasova, V., 1984. Summarized results of pilot vaccination of carp against spring viremia in the Czech Socialist Republic. Pr. VURH Vodn., 14: 68–74.
- Tiffney, W.N., 1939. The identity of certain species of the Saprolegniaceae parasitic to fish. J. Elisha Mitchell. Sci. Soc., 55: 134–151.
- Wakabayashi, H., 1993. Columnaris disease. In: V. Inglis, R.J. Roberts and N.R. Bromage (Editors), Bacterial Disease of Fish. Blackwell Scientific Publications, Oxford, pp. 23–39.
- Wakabayashi, H. and Egusa, S., 1972. Preliminary experiments on environmental factors influencing the prevalence of columnaris disease. Fish Pathol., 7: 58–63.
- Waluga, D. and Budzynska, H., 1980. *Sphaerospora* invasion as the cause of a massive mortality in carp populations. Gospod. Rybn., 7: 5–7 (in Polish).
- Wedemeyer, G.A. and McLeay, D.J., 1981. Methods for determining the tolerance of fishes to environmental stressors. In: A.D. Pickering (Editor), Stress and Fish. Academic Press, London–New York–Toronto–Sidney–San Francisco, pp. 247–276.
- Wedemeyer, G.A. and Yasutake, W.T., 1977. Clinical methods for the assessment of the effects of environmental stress of fish health. US Fish Wildl. Leaflet, 89.
- Willoughby, L.G., 1971. Observations of fungal parasites of Lake District salmonids. Salmon Trout Mag., 192: 152–158.
- Wolf, K., 1988. Fish Viruses and Fish Viral Diseases. Cornell University Press, Ithaca, NY.
- Wolke, R.E., 1975. Pathology of bacterial and fungal diseases affecting fish. In: W.E. Ribelin and G. Migaki (Editors), The Pathology of Fishes pp. 33–116. The University of Wisconsin Press, Madison, WI.
- Wood, J.W., 1974. Diseases of Pacific Salmon: Their Prevention and Treatment, 2nd edn. State of Washington Department of Fisheries, Hatchery Division, 82 pp.
- Wretling, B., Molby, R. and Wadstrom, T., 1971. Separation of two haemolysins from *Aeromonas hydrophila*. Infect. Immun., 4: 503–505.
- Yano, T., Mangindaan, R.E.P. and Matsuyama, H., 1989. Enhancement of resistance of carp *Cyprinus carpio* to experimental *Edwardsiella tarda* infection, by some β -1,3-glucans. Nippon Suisan Gakkaishi, 55: 1815–1819.
- Yano, T., Matsuyama, H. and Mangindaan, R.E.P., 1991. Polysaccharide-induced protection of carp *Cyprinus carpio* L. against bacterial infection. J. Fish Dis., 14: 577–582.