SAPROLEGNIA INFECTIONS OF SALMONID FISH

A. D. PICKERING & L. G. WILLOUGHBY

The colonization of teleost fish by aquatic fungi is a serious problem affecting both wild fish and those under conditions of intensive cultivation. Fungi belonging to the class Oomycetes are the most important colonists of freshwater fish. To the untrained eye, such infections are the most obvious of all the fish diseases yet the instant recognition of cotton-wool-like growth on the integument (Plate 1a) is, paradoxically, largely responsible for the dearth of critical investigations on such conditions. The simplistic approach of classifying all fungal infections of freshwater fish as "Saprolegniasis" (i.e. belonging to the genus *Saprolegnia*) and then dismissing them without further study is a mistake. We found as many as four different genera of fungi growing together in lesions of the perch (*Perca fluviatilis* L.), so the composition of such lesions can be very complex and the identification of fungi growing on fish needs considerable care (Willoughby 1970; Pickering & Willoughby 1977; Bucke et al. 1979).

One characteristic of fungal infections in both wild and hatchery-reared fish is the sporadic nature of such outbreaks. The interrelations between a pathogen, its host and the environmental conditions are complex, and it is generally accepted that overt diseases occur only when a susceptible host is exposed to a virulent pathogen under certain environmental conditions. Consequently, it is necessary to consider all three components (pathogen, host and environment) if we are to fully understand the nature of such outbreaks and, ultimately, find out how to control them. To this end, we will deal firstly with the identification and characteristics of fungal pathogens that colonize salmonids and then consider the relative importance of the condition of the host fish and the environmental factors which may influence the interaction between pathogen and host. Our prime consideration will be the work that has been undertaken at the Windermere laboratory of the FBA during the past decade, work which has been partly funded since 1974 by a contract with MAFF.

Identification of the salmonid pathogen

Common species of *Saprolegnia* in fresh water include the *S. diclina* complex, *S. ferax* and *S. hypogyna*. Other species such as *S. australis*, *S. glomerata* and *S. subterranea* are more rare. At the present time we have over 100 strains of fungi in pure culture and these are used extensively in our experimental work. The majority of these isolates have been obtained from infected fish and it is clear that the most important pathogens comprise part of the *S. diclina* complex. These frequently occur on infected salmonids and have also been found on cyprinids such as carp, *Cyprinus carpio* L., orfe, *Leuciscus idus* (L.), and tench, *Tinca tinca* (L.), as

well as on the pike, *Esox lucius* L., and the eel, *Anguilla anguilla* (L.). Indeed, we have even isolated members of the *S. diclina* complex from a non-teleost 'fish', the river lamprey, *Lampetra fluviatilis* (L.). Our experience over the years has shown that many of the morphological and some of the physiological characters of these fungi are stable and this has enabled us to examine aspects of their biology which we believe may account for their undoubted pathogenicity.

Of the isolates from salmonid fish, 75% have been identified as *Saprolegnia diclina* Humphrey Type 1, synonymous with the previously-named *S. parasitica* Coker (Willoughby 1978 a,b). The remaining isolates have all failed to produce sexual structures under laboratory conditions and hence cannot be identified by classical criteria. These will be referred to as *Saprolegnia* sp. but it seems likely from ultrastructural studies (see below) that they are closely related to, if not identical with, *Saprolegnia diclina* Type 1.

Special features of the Saprolegnia of salmonids

S. diclina is not an obligate parasite on fish and can best be described as a facultative necrotroph (i.e. normally saprophytic on dead animal and plant tissue but also capable of a parasitic existence). Each growing colony bears numerous sporangia, releasing large numbers of motile zoospores; these are believed to be the main dispersive and infective agents in the fungal life cycle. Infected fish may release in excess of 190000 spores per minute (Willoughby & Pickering 1977). Primary zoospores are released from the zoosporangia but these quickly encyst to release a more active, secondary zoospore stage. Little is known about the factors that influence the migration of the zoospores, but it seems probable that chemotropism towards a suitable food source is an important mechanism. Once the secondary zoospore finds itself near suitable food it again encysts to form a secondary zoospore cyst. Interestingly, the ultrastructure of the secondary zoospore cysts of *S. diclina* Type 1 and *Saprolegnia* sp. differs markedly from *S. diclina* Type 3, the purely saprophytic strain of the same species (Pickering et al. 1979). The secondary zoospore cysts of pathogenic strains bear numerous bundles of long hairs, each terminating in a pair of recurved hooks (Plate 1b). By comparison, the saprophytic strains of *S. diclina* together with all other *Saprolegnia* species that we have so far examined have much shorter hooked hairs which are not aggregated into bundles (Plate 1c). It seems likely that the hooked hairs are attachment structures and we believe that their greater development in the pathogenic strains represents an important adaptation to their mode of life in which firm attachment to the host fish is a prerequisite for a parasitic existence.

The factors which trigger both secondary zoospore encystment and subsequent germination and growth require further study, but already we

have found marked differences between the pathogenic and the purely saprophytic isolates (Willoughby et al. 1982). The pathogenic strains have the ability to germinate and grow in water containing very low concentrations of nutrients. This has been observed in sterile lake water but is best seen in water from the effluent of our own fish hatchery. In both situations, the secondary zoospores of *S. diclina* Type 1 or *Saprolegnia* sp., isolated as pathogens from fish, encyst within a few minutes and then germinate to produce a thread-like mycelium. In contrast, secondary zoospores of saprophytic strains of *S. diclina* Type 3, *S. ferax* and *S. hypogyna* are totally unresponsive under these conditions and continue to swim vigorously for periods in excess of 24 h. The subsequent mode of growth of the pathogenic isolates is strongly dependent upon the concentration of nutrients in the surrounding water (Willoughby et al. 1982). We have shown, by means of experiments involving the serial dilution of a standard growth medium, that at high nutrient levels the mycelium is in cytoplasmic continuity with the attached cyst. At low nutrient concentrations, however, the cytoplasm of the developing mycelium migrates apically and is repeatedly walled-off from the attached zoospore cyst case. The empty portion of the mycelium may be over 400 μm in length after 20 h at 10 °C and have as many as 20 cross-walls between the cytoplasm-filled apical portion and the cyst case. This is the kind of germination seen in pathogenic isolates growing in our fish hatchery effluent; it also occurs when they grow in solutions of fish mucus. One can speculate that this may be a mechanism which enables the fungus to extend its area of search for new nutrient sources with a minimal use of the cytoplasmic reserves.

If the colony continues to survive under these conditions of half-starvation it may abbreviate its life cycle by producing a 'minisporangium' which releases a single zoospore (Willoughby 1977). Thus, the pathogenic strains can not only germinate and grow under conditions which would not even trigger the encystment of saprophytic strains, but they also have the ability to revert back to the dispersive phase should the environment ultimately prove to be unsuitable for further growth.

Our preliminary examination of physiological differences between strains has, with few exceptions, segregated the fungi from salmonid fish, which cannot grow at high temperatures (33 °C), from those from non-salmonid (coarse) fish that show vigorous growth at 33 °C (Willoughby & Copland in press). Moreover, in the coarse fish isolates, zoospore production and release – an essential preliminary to the pathogenic cycle – can occur at 29 °C whereas the salmonid isolates are unable to produce zoospores at such high temperatures. Conversely, the fungi from salmonid fish are more competent at zoospore production at low temperatures (3 °C) than are those from coarse fish. Fungal cultures obtained during an outbreak of fungal infection on elvers in an intensive cultivation unit

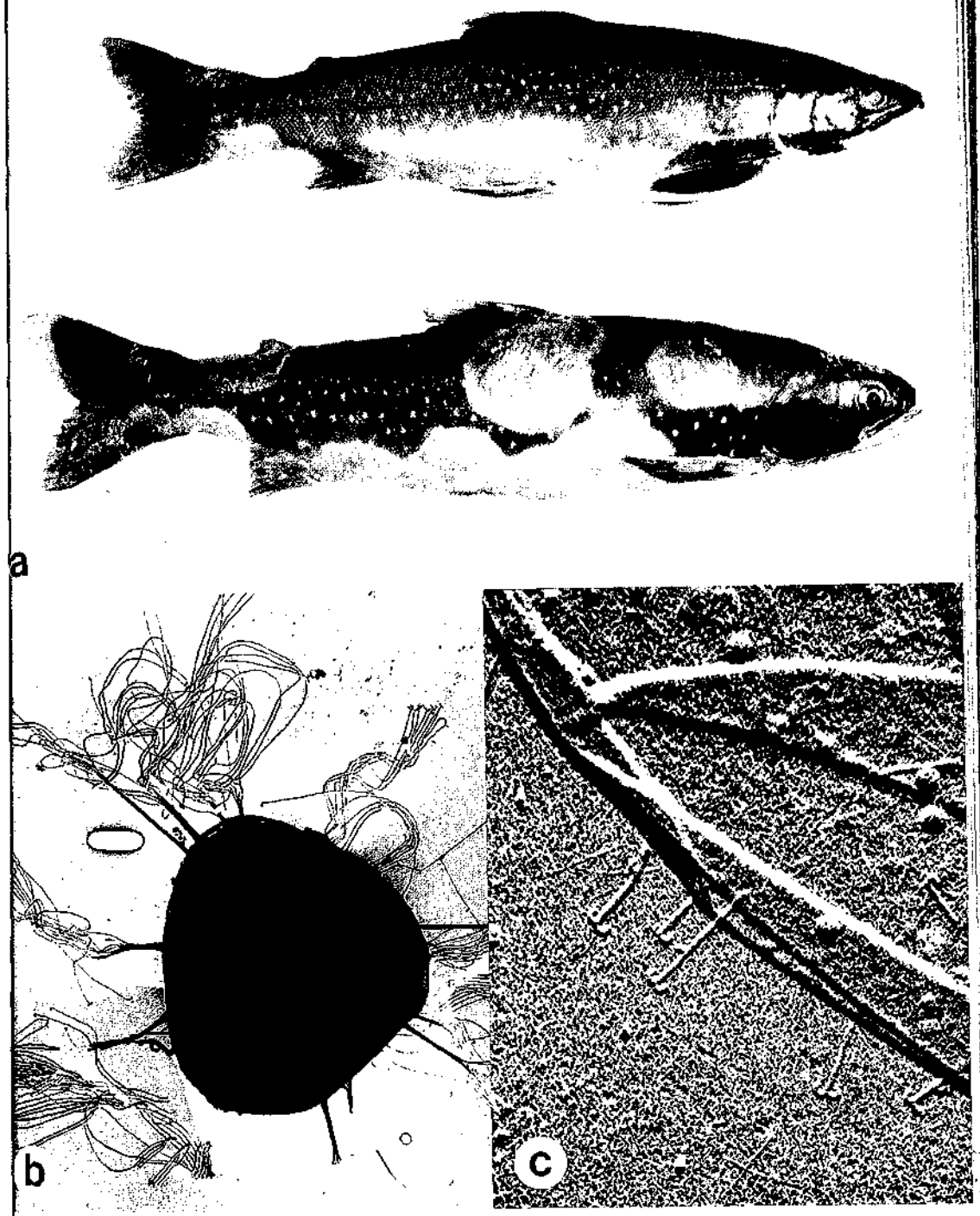

PLATE 1. (a) *Saprolegnia*-infected char from Windermere.
(b) Secondary zoospore cyst of *S. diclina* Type 1 (×4300)
(c) Part of a secondary zoospore cyst case of *S. diclina* Type 3 (× 30 000).

which uses the heated effluent of a power station (25 °C) have been found to stand apart from this basic division; they can both grow and produce zoospores over a wide temperature range. A series of sub-culture experiments has shown that these differences in temperature tolerance are consistent and reproducible and, therefore, may be of ecological importance. The suggestion that the temperature preference of the parasite is matched to that of its host is of great interest.

Effects of the fungus on the host fish

Saprolegnia infection can occur on almost any external part of the fish although there is evidence that some areas are much more easily infected than others (Richards & Pickering 1978). Hatchery-reared fish are most frequently infected on the fins and in both wild and hatchery-reared trout there is a marked sexual difference in the pattern of fungal infection during the spawning season. Male fish are most vulnerable along the dorsal surface whereas females are more frequently infected on the caudal and ventral fins. One unexplained feature of the lesions caused by *Saprolegnia* on salmonid fish is the occurence of lesions in the form of a delicate ring of fungus surrounding an apparently uninfected area (Willoughby 1971). This may represent some form of temporary inhibition or restriction of growth during the early stages of infection from a point source, but we have not yet done experiments to test this idea.

On most fish, *Saprolegnia* infections rarely penetrate deep into the body tissues and are usually restricted to the epidermis, dermis and, possibly, the superficial musculature. The microscopic structure of these lesions has been fully described (Pickering & Richards 1980) and it is suggested that the degenerative changes observed in the tissues may represent damage caused by proteolytic enzymes secreted by the fungal hyphae. Typical inflammatory responses by the fish tissues are usually absent or only weakly developed unless the lesions are also infected by bacteria. The relatively superficial nature of *Saprolegnia* infections and the lack, as far as we are aware, of any evidence of a toxin that may be transmitted by the bloodstream of the fish, pose the question "How does the fungus exert its undoubted, debilitating effects on the host fish?"

The answer to this question lies in a study of the osmoregulatory systems of the fish. In fresh water, teleost fish are constantly subjected to an osmotic influx of water and a diffusional loss of ions, mainly across the membranes of the gills. Under normal circumstances these fluxes are counter-balanced by the active uptake of salts at sites located in the gills and by the production of copious amounts of urine. This system can remain balanced only as long as the skin of the fish forms a relatively impermeable layer. In *Saprolegnia*-infected fish it has been shown that the osmotic pressure and salt content of the blood is inversely related to the severity of the infection (Richards & Pickering 1979). It seems clear

from this study that the fungus destroys the essential waterproofing properties of the skin and ultimately results in a lethal dilution of the body fluids of the fish. From our own experience with hatchery-reared brown trout, survival time may be less than three days from the first visible signs of infection.

Defence systems of the fish

Because *Saprolegnia* infections of salmonid fish are normally restricted to the superficial tissues, the defence systems of the potential host fish can be broadly divided into two categories: (1) removal of the infective agent from the body surface before growth of the colony has become established, and (2) inhibition or restriction of the growth of an attached colony. Much of our work to date concerns the first of these two possibilities.

The epidermis of the salmonid fish contains numerous mucus-secreting goblet cells which continuously replace the layer of slime on the surface of the fish (Pickering 1974). Radioisotope tracer studies on the kinetics of the secretion of mucus indicate that the life span of each cell in the epidermis is of the order of one week (Pickering 1976). We have shown that *S. diclina* Type 1 spores readily adhere to the surface of both the brown trout and the char, although most of the spores are either removed or inactivated within a few hours (Willoughby & Pickering 1977). In subsequent studies, it was shown that the rate of loss of viable spores from the surface of the fish was similar to that of inert particles of a similar size (Pickering & Willoughby in press) and from this we conclude that the physical removal of fungal spores by the continuous secretion of mucus from the epidermis is a major component of the fish's defence to this form of challenge.

The second component of the defence system, i.e. the restriction or inhibition of fungal growth, has not been satisfactorily demonstrated. Antibiotic properties of the mucous secretions of certain teleosts have been recognized for a considerable period of time and some of the active principles have now been isolated and characterized. However, to our knowledge, no antifungal properties of salmonid mucus have been reported. Moreover, epidermal mucus taken from the surface of salmonid fish acts as an effective growth medium for *S. diclina* Type 1 (Willoughby 1971; Willoughby & Pickering 1977; Willoughby et al. in press). Nevertheless, in view of the marked plasticity of epidermal structure and function in salmonids (Pickering & Richards 1980) we cannot yet discount the possibility of changes in time in the antibiotic properties of their mucous secretions.

Little of the extensive literature on teleost immunology concerns the responses of fish to fungal antigens, although the early infiltration of lymphocyte-like cells to the sites of *Saprolegnia* infection in brown trout may indicate a possible direct immune response (Pickering & Richards 1980). It is apparent that much more work is required if we are to elucidate the relative roles of specifically induced and natural antibodies in the defences of fish against *Saprolegnia* infection.

Predisposing factors

We have now seen how several of the morphological and physiological characters of the pathogenic strains of *Saprolegnia diclina* Type 1 and *Saprolegnia* sp. may be adaptations to a parasitic mode of life, and we have outlined what is known about the defence of the fish against such pathogens. Outbreaks of disease occur when the equilibrium between the pathogenic challenge and the fish's resistance is disturbed in favour of the pathogen. This may be a result of an increase in numbers or virulence of the pathogen in the water or an increase in the susceptibility of the fish.

(a) *Concentration of pathogenic organisms in the water*

From our laboratory studies on a large number of fungi it would appear that many of the characters which we believe may be responsible for their pathogenicity are of a relatively stable nature. Thus, one also has to consider the possibility of an increase in *numbers* of infective organisms in the water as a factor influencing the outbreak of disease. Hitherto, a major drawback in the estimation of the number of propagules in a water-body has been the difficulty of identifying the pathogenic strains. On the basis of their sexual structures it is possible to distinguish *S. diclina* Type 1 from the numerous purely saprophytic species of *Saprolegnia* which inevitably occur in such water samples. This approach is extremely labour-intensive and time-consuming, however, and cannot be used to identify strains which are reluctant to produce the sexual structures under laboratory conditions. Because of these difficulties, relatively few critical investigations have been undertaken concerning the changes in abundance of spores of pathogenic fungi (determined to species) in relation to the outbreak of disease. Willoughby (1962) estimated the total concentration of Saprolegniales in Windermere to be between 25 and 5200 propagules per litre in the centre of the lake, with some evidence of a peak during the autumn period, but it was not possible to distinguish pathogenic from non-pathogenic fungi in this work. In a later study (Willoughby 1969) it was shown that *S. diclina* Type 1 accounted for approximately half the total Saprolegniaceae in water samples from the River Leven (the outflow of Windermere). This was a time when disease was rampant in the river. Under hatchery conditions and in the presence of infected fish, the concentration of fungal spores in the water may exceed 20000 spores per litre (Willoughby & Pickering 1977). We hope that the relatively rapid identification of pathogenic isolates by means of their secondary zoospore cyst ultrastructure (Pickering et al. 1979) will do much to stimulate studies in this area.

(b) *Epidermal integrity*

The initial defence of the fish against pathogenic fungi relies heavily upon the physical removal of infective spores by copious mucous secretion from the epidermal goblet cells. Thus, any impairment of mucous secretion or any breach in the integrity of the epidermis is likely to predispose the fish to fungal infection. Small abrasions can be covered by migrating epidermal cells and by mucus produced from adjacent areas of the epidermis. Large wounds, on the other hand, are less easily protected and act as points where fungi can colonize. It is likely that the increased susceptibility of the fins of hatchery-reared brown trout to *Saprolegnia* infection (Richards & Pickering 1978) is partly the result of physical damage to these areas under conditions of unnaturally high stocking density. In this case the problem may be exacerbated by the relatively low concentration of mucus-secreting goblet cells on the fins compared with the rest of the body (Pickering 1974). The association between damage and infection is further borne out by the pattern of infection on mature, female brown trout in which the tail and anal fins are particularly vulnerable (Richards & Pickering 1978). It is precisely these areas that are used by the fish to excavate the gravel of the spawning redds and, consequently, physical damage is to be expected. Many of the routine hatchery procedures involving handling of fish will also increase the probability of fungal infection.

Other kinds of damage can also predispose salmonid fish to *Saprolegnia* infection, for example ulceration of the superficial tissues. Perhaps the most publicized case of ulceration associated with fungal infection is that of ulcerative dermal necrosis (UDN). In many ways, however, UDN is a poor example to cite because the primary cause of the ulcerated condition has not yet been identified. In the major outbreak of salmon disease during the period 1966-69 three conditions were recognized: ulceration only (UDN), ulceration followed by *Saprolegnia* infection (UDN), *Saprolegnia* infection only. The third of these (i.e. *Saprolegnia* only) was the most prevalent condition in the local rivers Leven and Lune (see Willoughby 1968, 1969, 1971, 1972). The consensus of opinion at the time was that fish could recover from the ulcerated condition, but should the fish become infected by *S. diclina* Type 1 or *Saprolegnia* sp. the probability of survival was markedly reduced.

From our own experience with hatchery-reared brown trout it is clear that both bacterial fin rot and furunculosis (*Aeromonas salmonicida*) can also result in *Saprolegnia* infections.

(c) *Sexual maturation*

Sexual maturation in brown trout (*Salmo trutta* L.) is associated with an increase in susceptibility to a variety of skin ectoparasites, including *S. diclina* Type 1 (Pickering & Christie 1980). As we have already seen, physical damage of the female during the process of spawning may partly

account for this phenomenon, as might physical damage of the male fish during aggressive territorial encounters. However, the increase in susceptibility to fungal infections develops in lacustrine male brown trout before the fish move into the spawning streams (Richards & Pickering 1978), and the fact that maturing fish are also more susceptible to other ectoparasites (e.g. *Ichthyophthirius*, *Costia*, *Gyrodactylus*, *Scyphidia*) indicates a more general increase in vulnerability. It has been found that mature male fish are more prone to such infestations than are mature female fish (Richards & Pickering 1978; Pickering & Christie 1980) an observation supported by the work of Paling (1965) who demonstrated a sexual difference in the incidence of infestation of the gills of the brown trout in Windermere by the trematode *Discocotyle sagittata* (Leuckart). Like us, he found that the mature male fish were more susceptible than the mature females or the immature fish. Thus, there is good evidence that sexual maturation (particularly in the males) is associated with changes in the defence mechanisms which may ultimately increase the susceptibility of the fish to a wide range of parasites, including *Saprolegnia*.

Sexual maturation in the brown trout is also accompanied by dramatic changes in the structure and mucification of the epidermis. A marked loss of mucous cells occurs in the male fish towards the end of the spawning period (Pickering 1977; Pickering & Richards 1980), and from the preceding section on the role of mucus in the defence systems of the fish, the temptation to conclude that this loss is responsible for the changes in susceptibility to fungal infection is almost irresistible. However, it should be pointed out that the demucification often occurs *after* the increase in susceptibility (Pickering & Christie 1980), not before it. Perhaps the loss of epidermal mucous cells in mature male brown trout exacerbates an existing increased vulnerability to skin ectoparasites. Because major changes occur in the endocrine system when teleost fish mature, we are now looking to see if these hormones, especially the corticosteroids and androgens, might not affect the defence systems of salmonid fish.

(d) *Stress*

Activation of the pituitary-interrenal axis is an almost ubiquitous component of the response of teleost fish to a wide variety of different stimuli, most of which can be described as stressful (see Pickering 1981). For example, we have shown an increase in the amount of circulating cortisol (a major corticosteroid in salmonid fish) in response to disturbance, physical handling and disease (Pickering & Christie 1981; Pickering et al. in press). Moreover, the use of formalin or malachite green when treating ectoparasitic infestations and fungal infections can, in itself, act as an acute stress and elevate blood cortisol levels (Pickering & Pottinger in press). Neish & Hughes (1980) postulate that there is a direct link between stress-mediated increases in plasma corticosteroid levels and a fish's susceptibility to *Saprolegnia* infection. The mechanisms involved

have not been elucidated although immunosuppression, decreased inflammatory response and impaired wound healing all seem worth looking at. Convincing demonstrations of the predisposing effects of elevated blood corticosteroids are, however, somewhat limited. In a recent investigation into methods of administering physiological levels of cortisol to brown trout without the stress of physical handling, we found that the incorporation of cortisol into the diet resulted in a significant increase in mortality rate. The cause of death was primary *Saprolegnia* infection. In subsequent studies involving the implantation of hormones, we found that cortisol also decreased the resistance of brown trout to *Aeromonas salmonicida*, the causative agent of furunculosis. The evidence from these preliminary experiments is sufficient to suggest that further work along these lines is justified. Particular attention will be given to experimental challenge of the fish by pathogenic strains of *S. diclina* Type 1 and *Saprolegnia* sp.

Conclusions

Fungal infections of salmonid fish are almost invariably associated with strains either identical to *S. diclina* Type 1 or very similar to it. Ultrastructural evidence indicates that the sexually sterile isolates (*Saprolegnia* sp.) are closely related to *S. diclina* Type 1. These pathogenic isolates are able to encyst and grow under environmental conditions (with a low nutrient status) which would not promote encystment and growth of related saprophytic species of *Saprolegnia*. Moreover, there are mechanisms in the pathogenic isolates for reverting to zoospore motility under conditions which are unsuitable for the subsequent growth of the colony. The temperature tolerances of isolates from a range of teleost fish reflect, to a certain extent, the tolerances of the host fish.

S. diclina Type 1 and *Saprolegnia* sp. frequently occur as secondary infections although they can also act as primary pathogens. Infections are normally restricted to the superficial tissues, resulting in a breakdown of the fish's osmoregulatory mechanisms. Much of the defence system of salmonid fish to *Saprolegnia* infections appears to depend upon the removal of infective spores and cysts by means of the continual secretion of mucus by the epidermis. Once an infection is established it is unusual for a fish to recover naturally. Integumental damage (by physical means or as a result of other primary infections) frequently results in fungal infections and as salmonid fish become sexually mature they also become more susceptible. Some evidence suggests that activation of the pituitary-interrenal axis as part of the stress response in teleosts predisposes the fish to Saprolegniasis.

This work could not have been undertaken without the help and cooperation of many colleagues at the FBA to whom our thanks are due. They have been individually acknowledged in the original research papers on which this review article is based. We are particularly grateful to MAFF for financial support of this project since 1974, and to Mr T. I. Furnass (FBA) for assistance with the preparation of photographic material for this article.

References

Bucke, D., Cawley, G. D., Craig, J. F., Pickering, A. D. & Willoughby, L. G. (1979). Further studies of an epizootic of perch, *Perca fluviatilis* L., of uncertain aetiology. *J. Fish Dis.* **2**, 297-311.

Neish, G. A. & Hughes, G. C. (1980). *Fungal diseases in fish.* New Jersey. T. F. H. Publications.

Paling, J. E. (1965). The population dynamics of the monogenean gill parasite *Discocotyle sagittata* Leuckart on Windermere trout, *Salmo trutta* L. *Parasitology*, **55**, 667-94.

Pickering, A. D. (1974). The distribution of mucous cells in the epidermis of the brown trout *Salmo trutta* (L.) and the char *Salvelinus alpinus* (L.) *J. Fish Biol.* **6**, 111-18.

Pickering, A. D. (1976). Synthesis of N-acetyl neuraminic acid from [^{14}C] glucose by the epidermis of the brown trout, *Salmo trutta* L. *Comp. Biochem. Physiol.* **54B**, 325-28.

Pickering, A. D. (1977). Seasonal changes in the epidermis of the brown trout *Salmo trutta* L. *J. Fish Biol.* **10**, 561-66.

Pickering, A. D. (ed.) (1981). *Stress and fish.* London. Academic.

Pickering, A. D. & Christie, P. (1980). Sexual differences in the incidence and severity of ectoparasitic infestation of the brown trout, *Salmo trutta* L. *J. Fish Biol.* **16**, 669-83.

Pickering, A. D. & Christie, P. (1981). Changes in the concentrations of plasma cortisol and thyroxine during sexual maturation of the hatchery-reared brown trout, *Salmo trutta* L. *Gen. comp. Endocr.* **44**, 487-96.

Pickering, A. D. & Pottinger, T. G. (in press). Factors influencing blood cortisol levels of brown trout under intensive culture conditions. *Proc. 9th int. Symp. comp. Endocr.*

Pickering, A. D., Pottinger, T. G. & Christie, P. (1982). Recovery of the brown trout, *Salmo trutta* L., from acute handling stress: a time-course study. *J. Fish Biol.* **20**, 229-44.

Pickering, A. D. & Richards, R. H. (1980). Factors influencing the structure, function and biota of the salmonid epidermis. *Proc. R. Soc. Edinb.* **79B**, 92-104.

Pickering, A. D. & Willoughby, L. G. (1977). Epidermal lesions and fungal infection on the perch, *Perca fluviatilis* L., in Windermere. *J. Fish Biol.* **11**, 349-54.

Pickering, A. D. & Willoughby, L. G. (in press). *Saprolegnia* infections of salmonid fish. *Symp. Soc. gen. Microbiol.*

Pickering, A. D., Willoughby, L. G. & McGrory, C. B. (1979). Fine structure of secondary zoospore cyst cases of *Saprolegnia* isolates from infected fish. *Trans. Br. mycol. Soc.* **72**, 427-37.

Richards, R. H. & Pickering, A. D. (1978). Frequency and distribution patterns

of *Saprolegnia* infection in wild and hatchery-reared brown trout *Salmo trutta* L. and char *Salvelinus alpinus* (L.) *J. Fish Dis.* 1, 69-82.

Richards, R. H. & Pickering, A. D. (1979). Changes in serum parameters of *Saprolegnia*-infected brown trout, *Salmo trutta* L. *J. Fish Dis.* 2, 197-206.

Willoughby, L. G. (1962). The occurrence and distribution of reproductive spores of Saprolegniales in freshwater. *J. Ecol.* 50, 733-59.

Willoughby, L. G. (1968). Atlantic salmon disease fungus. *Nature, Lond.* 217, 872-73.

Willoughby, L. G. (1969). Salmon disease in Windermere and the River Leven: the fungal aspect. *Salm. Trout Mag.* 186, 124-30.

Willoughby, L. G. (1970). Mycological aspects of a disease of young perch in Windermere. *J. Fish Biol.* 2, 113-16.

Willoughby, L. G. (1971). Observations on fungal parasites of Lake District salmonids. *Salm. Trout Mag.* 192, 152-58.

Willoughby, L. G. (1977). An abbreviated life cycle in the salmonid fish *Saprolegnia*. *Trans. Br. mycol. Soc.* 69, 133-66.

Willoughby, L. G. (1978a). Saprolegnias of salmonid fish in Windermere: a critical analysis. *J. Fish Dis.* 1, 51-67.

Willoughby, L. G. (1978b). *CMI descriptions of pathogenic fungi and bacteria, No. 598: Saprolegnia diclina.* Kew. Commonwealth Mycological Institute.

Willoughby, L. G. & Copland, J. W. (in press). Temperature-growth relationships of *Saprolegnia* pathogenic to fish, especially eels grown in warm water. *Trans. Br. mycol. Soc.*

Willoughby, L. G. & Pickering, A. D. (1977). Viable Saprolegniaceae spores on the epidermis of the salmonid fish *Salmo trutta* and *Salvelinus alpinus*. *Trans. Br. mycol. Soc.* 68, 91-95.

Willoughby, L. G., McGrory, C. B. & Pickering, A. D. (in press). Zoospore germination in *Saprolegnia* pathogens of fish. *Trans. Br. mycol. Soc.*

POPULATION DYNAMICS OF WINDERMERE PERCH

J. F. CRAIG

The history of the long-term studies of Windermere perch (*Perca fluviatilis* L.) is well recorded by Bagenal (1977) and there is no necessity to repeat it here. The main areas of interest throughout these studies have been factors which control recruitment and abundance (Le Cren 1965; Kipling 1976; Bagenal 1977; Le Cren et al. 1977; Craig et al. 1979; Craig 1980). This review looks briefly at some of the more recent analyses and interpretations of the changes that have occurred, and at the present level of understanding. The dynamics of a population of fish depend on how the individuals grow, reproduce and survive. The capacity of any organism to grow, reproduce and survive in a changing environment will depend on its ability to respond to these changes. This ability is limited by the organism's genetically determined powers of adaptation (both physiological and behavioural). Extensive knowledge about the responses of populations can be gained by studies conducted over a period of time. In this situation the changes in environmental conditions are brought about naturally although some conditions can be influenced by man. So far we have followed this system in Windermere. Initially in our study, the population of perch was severely perturbed by a fishery which removed a large proportion of the adult stock of fish (Worthington 1950). Since then the population has been monitored while it responded to the natural year-to-year changes in temperature and other factors. We are now in a position to make some fairly accurate predictions about the response of the population to certain environmental conditions. But these predictions are based largely on empirical correlations, and to make further progress we need to know more about the causal mechanisms involved. This will require experimental studies on the responses of individual fish; in particular their responses to temperature, the bioenergetics of their food use, and how they respond to the density of their own species.

Marking experiments have shown that there is little movement of perch between the north and south basins of Windermere, and perch do not migrate out of the lake so we are able to ignore immigration and emigration as factors in their population dynamics. In the analysis of population dynamics we are concerned with birth and mortality rates. Growth rates are also of importance in so far as they have a direct influence on the former two rates.

Birth rate is the number of offspring produced per female per unit of time. The *absolute fecundity*, i.e. the total number of eggs laid by a female perch, is related to the size of the fish. A regression of logarithm of absolute fecundity (F) on the logarithm of length (L) from data gathered in the years 1979, 1980 and 1981 gave: