

progress in order to show that too facile dismissal of fungal involvement in previously reported work was premature.

We did not set out to answer "whether *S. parasitica* is a fresh-water fungus or a fungus capable of growing and sporulating in both fresh-water and estuarine environments". We merely recorded two facts: (a) that *Saprolegnia* has been isolated in a number of cases from fish that we can prove to be freshly arrived in an estuary; and (b) that *S. parasitica* can grow in saline conditions.

At this stage of the investigation, these are facts important enough to warrant recording and are not vitiated by Dick's comments. For *Saprolegnia* to be involved at all in estuarine conditions it is not essential, as Dick states, to find out whether the fungus is capable of sporulating and so on equally well in both fresh-water and estuarine conditions (Q1-6), however desirable it may be to have further evidence along these lines. We do not imply that the estuary is the sole locus for infection, and indeed believe that it is much more likely that *Saprolegnia* is more active in fresh water.

Finally, we state that "the apparent correlation between the spread of the fungus in ulcerative dermal necrosis and water temperature merits investigation". This is a general observation and nowhere do we quote "temperature data for asexual sporulation" as Dick states.

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¹ Dick, M. W., *Nature*, **217**, 875 (1968).

² Stuart, M., and Fuller, H., *Nature*, **217**, 90 (1968).

Resistance of Rainbow Trout to Ulcerative Dermal Necrosis

THE susceptibility of rainbow trout to ulcerative dermal necrosis¹, an infectious disease of some salmonids, has become a matter of importance to fish farmers. We have shown that the aetiological agent of ulcerative dermal necrosis is filterable² although some workers have suggested that specific bacteria^{3,4} or fungi⁵ may be the primary pathogens. Preliminary experiments indicated that rainbow trout exposed to a salmon affected by ulcerative dermal necrosis did not show symptoms of the disease whereas a healthy salmon in the same tank as the trout did. The siphoning technique successfully used to reproduce ulcerative dermal necrosis in salmon² was adapted to the experiments described here, which were designed to ascertain the susceptibility of rainbow trout to the disease. Further experiments, also described, were carried out to show whether rainbow trout can act as symptomless carriers.

In experiment *A*, ten mature hatchery-reared rainbow trout obtained from the Inland Fisheries Trust farm at Roscrea were divided into two equal groups. One group was kept as a control and the other was placed in a tank with a salmon obtained from the Boyne, a river free from ulcerative dermal necrosis. Water was siphoned into the tank containing the trout and salmon from a tank containing a salmon obtained during a natural outbreak, and showing clinical signs of ulcerative dermal necrosis¹. Both tanks (and all tanks used in these experiments) were disinfected with a chlorine solution before use.

The mean water temperature was 10.8° C, range 7.5-13.0° C. On the fifteenth day of the experiment, the Boyne salmon died showing lesions of ulcerative dermal necrosis. The five trout in the tank with the salmon showed no lesions, and they and the controls remained

clinically healthy until the end of the series of experiments, more than 4 months later.

In experiment *B*, the five rainbow trout exposed to infection in experiment *A* were placed in one disinfected tank and a healthy salmon from the Boyne was placed in another. Water was siphoned from the tank containing the trout to that containing the salmon. The mean water temperature was 7.2° C, range 5.0-8.0° C. On the forty-seventh day of the experiment the salmon was in excellent health and showed no signs of ulcerative dermal necrosis. The usual incubation period of the disease is 7-17 days².

In experiment *C*, the salmon used in experiment *B* was placed in a disinfected tank, and water was siphoned to it from a tank containing a salmon affected in a natural outbreak of ulcerative dermal necrosis. The mean water temperature was 6.3° C, range 3.0-10.0° C. The recipient salmon died showing lesions of ulcerative dermal necrosis.

The results of experiment *A* confirmed previous findings that rainbow trout do not show clinical signs of ulcerative dermal necrosis. Trout do not act as symptomless carriers of the disease, for the salmon which had been exposed to possible infection from the trout in experiment *B* was in fact susceptible, as shown in experiment *C*, so the result of experiment *B* was obtained because the trout were not infective.

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¹ Carbery, J. T., and Strickland, K. L. (in the press).

² Strickland, K. L., and Carbery, J. T. (in the press).

³ Brown, Margaret E., *Atlantic Salmon Journal* (summer, 1966).

⁴ Brown, M. E., and Collins, V. G., *Salmon Trout Mag.*, No. 178, 180 (1966).

⁵ Stuart, Mary R., and Fuller, Hubert T., *Nature*, **217**, 90 (1968).

Imprinting: Drug-produced Isolation and the Sensitive Period

IMPRINTING as a phenomenon in early behavioural development owes much of its importance to the fact that it occurs only during a limited period of an animal's life. Although there is some disagreement as to the exact nature and duration of this sensitive period, there is general agreement that such a period exists.

Because of its importance in distinguishing imprinting from other forms of stimulus-response association, the sensitive period has been the subject of considerable theoretical interest. This interest in turn has centred on the factors which influence the length and offset of the period. Bateson¹ has recently reviewed empirical studies of the role of these factors and has concluded that the imprinting response will usually continue to occur to a particular stimulus unless the bird concerned has had an opportunity to develop a preference for something else. This is, of course, equivalent to saying that imprinting as a process is inevitable and it will happen to either a class of stimuli in the bird's regular environment, or to a stimulus presented in an experimental imprinting situation.

I have developed this argument by assuming that the tendency to imprint on experimental objects declines as a result of the bird imprinting on some stimuli in its housing environment. If this imprinting on the general environment is made less effective by procedures which are known to make experimental imprinting less effective, then the time during which the bird will remain responsive to test objects will be prolonged. While it would be desirable to test directly the strength of attachment to aspects