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OBSERVATIONS ON VISCERAL GRANULOMATOSIS AND DERMAL NECROSIS IN POPULATIONS
OF NORTH SEA COD (Gadus morhua)

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Abstract

There have recently been reports and concern that large numbers of North Sea cod showing unusual gross signs of disease were being landed at English east-coast ports. MAFF investigations revealed that there were two diseases of significance affecting, particularly, the larger fish in two distinct cod populations. In the southern Bight, from 800 cod examined in November 1988, approximately 8% showed gross abnormalities of the visceral organs, including large cysts, nodules and discolouration (i.e. visceral granulomatosis). The second disease, which has been reported in the UK for the past 10 years, occurs in cod caught to the north of the Dogger Bank. The fish exhibited large haemorrhagic, blistering lesions on the skin near the anterior dorsal fin (i.e. dermal necrosis). A desk study revealed that at least one fish in every 6 boxes of cod (>60 cm length) landed at two ports in north-east England during 1988 were affected. The pathology of both conditions is described, and suggestions are proposed for their aetiologies. The significance of the disease is discussed in respect of any pollution involvement and the effect on the cod populations.

Résumé

On a récemment remarqué que des quantités importantes de morues de la mer du Nord présentant des signes pathologiques majeurs étaient apparues dans les ports de la côte est de l'Angleterre, ce qui a entraîné l'inquiétude que l'on devine. Le ministère (MAFF) a enquêté, ce qui a révélé que les plus gros individus de deux populations distinctes de morues étaient touchés par deux maladies principales. Dans la zone Bight du sud, sur 800 morues examinées en novembre 1988, environ 8 % présentaient des anomalies marquées des viscères, entre autres de larges cystes et nodules et une décoloration (c. -à-d. une granulomatose viscérale). La seconde maladie, signalée en Grande-Bretagne depuis dix ans, se rencontre chez les morues pêchées au nord du banc de Dogger. Les poissons présentent d'importantes lésions hémorragiques vésicantes de la peau à proximité de la nageoire dorsale antérieure (c. -à-d. une nécrose dermique). Une étude sur dossier a révélé qu'au moins un poisson toutes les six caisses de morues (de longueur > 60 cm) débarquées dans deux ports au nord-est de l'Angleterre durant 1988 était ainsi touché. La pathologie de ces deux états est décrite et des hypothèses étiologiques proposées. L'importance de ces maladies est discutée dans la perspective d'une cause liée à la pollution et au point de vue des effets sur les populations de morues.

Introduction

Two diseases occurring in cod, Gadus morhua, were recently brought to our attention. The first involved abnormalities of the visceral organs, and the second an unusual skin necrosis. Lesions of the viscera, previously described in cod from European waters, were diagnosed as "Ichthyophonosis" (Moller, 1974) and "presumptive mycobacteriosis" (van Banning, 1987), respectively. In 1988, a significant number of large cod, caught in the Southern Bight showed unusual visceral lesions. The skin necrosis syndrome in cod does not appear to have been officially documented, although there are ad hoc descriptions on our files of a similar disease going back at least 10 years. The condition appears to be restricted to large cod and only to those caught from a fairly small area to the north of the Dogger Bank, North Sea. This report describes currently-known prevalence rates of both diseases, as well as descriptions of their pathologies.

Case history A; visceral granulomatosis in cod

During summer 1988, an unusually-high number of reports were received about large cod being caught with internal abnormalities, including discoloured livers, lumps and cysts on hearts and other visceral organs. Initially, the reports came from fish processors who noticed growths on hearts in situ in eviscerated cod. Subsequently, reports of the problem were received from commercial fishermen and pleasure anglers. All reports suggested that it was only cod that were affected, which amounted to 30% of diseased fish being found in catches. Most reports referred to inshore landings, particularly off the south-east coast, including the outer Thames Estuary. However, there were occasional reports of diseased cod being caught all along the south English coast as far as Dorset in the south-west. Outward

appearances of the fish did not necessarily indicate internal problems, although there were reports suggesting that affected fish showed signs of emaciation and were covered in small skin ulcers. However, when eviscerating affected cod it was difficult not to see the varying numbers of small-to-large grey/brown nodules and cysts on livers, spleens, kidneys and hearts. In some fish, all visceral organs were affected; in others perhaps only one organ, usually the liver, showed signs of change. In the most affected cases, the appearance was quite severe, but even in those specimens their fillets appeared unaffected.

In order to obtain first-hand information on the situation we sampled cod in the Southern Bight on MAFF's R.V. CORYSTES in October/November 1988.

In all, 800 cod were examined and of those a mean prevalence rate of 8% fish with visceral granulomas was found, peaking to 10.7% at one inshore station. Microbiological examinations of tissue samples did not reveal any evidence of micro-organisms on selective media (Dorset egg agar, Lowenstein's medium and seawater agar). A comprehensive histological examination of the visceral organs was made, which showed that the nodules and other lesions were all in varying stages of granuloma development (Fig.4). Such lesions are typical for mycobacterial infections in fish (Bucke, 1980), and have been linked with other non-specific causes (Balouet & Baudin-Laurencin, 1986). Each granuloma consisted of aggregates of histiocytes forming into whorls. In the older lesions the cellular centres had degenerated into caseous necrotic masses. Bacteria were not demonstrated in any stages of the granuloma or in surrounding tissues. However, certain staining techniques (e.g. PAS, Grocott-Gomori's methenamine silver method) positively reacted to substances in the early lesions. These substances were identified as being 10-15 μ m diameter bodies (Fig. 5), but it was unclear, from light microscopical examination,

whether they represented a stage or remains of a protistan or, in fact, were endogenous tissue substances within host cells.

Case history B: skin necrosis in cod

This most unusual, if not unique, anomaly only appears to affect cod. Reports of the condition referred to affected fish with length size >60 cm. Even more remarkable is the fact that affected fish have almost entirely been caught in one area of the North Sea to the north of the Dogger Bank (see Fig. 1). Most reports of the disease have come by way of MAFF Fishery Inspectors and workers handling fish landed in the ports of Scarborough, North Shields and Whitby, north-east England. The gross clinical appearance of this condition, at its worst, is of a large, haemorrhagic "blistering" lesion, which either occurs singularly or bilaterally, and is usually situated adjacent to the anterior dorsal fin. The lesions have the appearance of "skin burns" rather than acute ulcers (Fig. 6). Our field investigations have indicated that, as the lesions develop, the general health status of the fish declines and it appears debilitated (slink). The lesions have been mostly superficial and, therefore, rarely is the underlying skeletal muscle affected and, as cod are skinned before sale, they are considered as being fit for consumption. Nevertheless, it has been reported that the most affected fish are destroyed before processing, or discarded at sea. Histological examination of the skin lesions by light and electron microscopy has revealed stages of epidermal necrosis, without any obvious signs of associated micro-organisms or parasites. Other organs and tissues in affected fish have appeared normal. The significance of this disease is unknown, although one would expect the more debilitated fish to die. Fishermen have reported prevalence rates as high as 20-30% in large cod and market surveys on cod catches showed that approximately 1-2 fish in every 6

boxes (12-15 fish per box) landed at two or three ports on the north-east coast of England were affected. The fact that, so far, an infectious agent has not been implicated has led to much speculation about the aetiology of this disease. The most popular hypothesis amongst fishermen has been a direct toxicity effect caused when large cod feed close to gas pipe-lines in this area of the North Sea, and as they swim near the pipes some form of toxic substance, emitting from the pipes, affects the skin. Other theories include: abrasions caused when fish are being hauled in trawl nets; injuries caused when large cod escape from static monofilament nets; seal bites; toxic substances in the water originating from burning waste products at sea; toxic effects of oil wastes near drilling rigs; and other pollution-related situations.

It seems unusual that only cod over a certain size become affected and only in a limited area, as there are also pipelines, seals, fishing activities, etc, elsewhere in the North Sea. Commercial fishermen suggest that this skin condition is present at a low level all of the time in this one cod population, but that it only draws attention to itself when large cod move into the area and are caught. The disease is not easy to investigate, because affected cod are not caught on routine research cruises. Therefore, surveys and the collection of diseased materials can only be made from commercial fishing vessels because they tend to fish near pipelines and wrecks which are favourite sites for large cod.

General discussion

A. Visceral granulomatosis in cod

Visceral granulomatosis is a condition common to many fish species. It has frequently been diagnosed as mycobacteriosis (syn. piscine tuberculosis) (Majeed et al., 1981). This diagnosis was given because the

typical histological characteristics involving the granulomatous lesions are often identical for mycobacterial infections in higher animals. In some of these instances, acid alcohol-fast bacilli have been demonstrated in the granulomas (Bucke, 1980). From those observations there is justification to interpret the diagnosis of "presumptive mycobacteriosis", and unless Koch's postulates are fulfilled the diagnosis should not be more than "presumptive". Early workers (Alexander, 1913; Johnstone, 1927), describing mycobacteria in cod, may have been mistaken in their diagnosis unless they fulfilled the above criteria. However, as in this current case, the majority of visceral granulomas in fish tissues may not be the result of mycobacterial infections, but a host response to any non-specific pathogen. Morrison et al. (1984) described the development of visceral granuloma in cod after they had been experimentally injected with the fish pathogen Aeromonas salmonicida. Furthermore, formation of granulomas in the cod may be a host-response to pathogenic toxins liberated from infectious agents which have themselves disappeared from the fish's system. Balouet & Baudin-Laurencin (1986) experimentally induced such granuloma in turbot, Scophthalmus maximus, after injection with immunogenic (BCG) material.

Visceral granulomatosis in cod appears to have the characteristics of an infectious disease rather than those of a chemically-induced condition. The suggestion that such diseases are triggered-off by a pollutant, causing a reduction in the cod's immune response, is purely speculative. It is probable that an increased proportion of one age class of cod are affected and it is only when they are available for catching that the high disease prevalence rates are observed, thus presenting, over the years, fluctuations in disease levels (van Banning, 1987). There is evidence that the more affected fish occur inshore and these are more

readily caught in trawl nets and by other methods.

B. Skin necrosis in cod

Our investigations have indicated that this disease syndrome in cod is unique, although other forms of skin ulceration have been reported (Jensen & Larsen, 1979, 1982). It is worth noting that in the early 1920s outbreaks of skin diseases in cod caught from the Dogger Bank were significant enough to cause concern within the industry and resulted in a scientific investigation (Johnstone, 1922). At that time there were suggestions that the fish were responding to toxic changes derived from chemicals released from dumped World War I munitions and explosives. That hypothesis was never substantiated as presumably the problem disappeared when those particular year classes which were affected either died naturally or were fished out.

Both these cod diseases will be monitored and when affected fish become available there will be further attempts to identify the causes. However, any suggestions or further information about these diseases and their possible causes would be appreciated.

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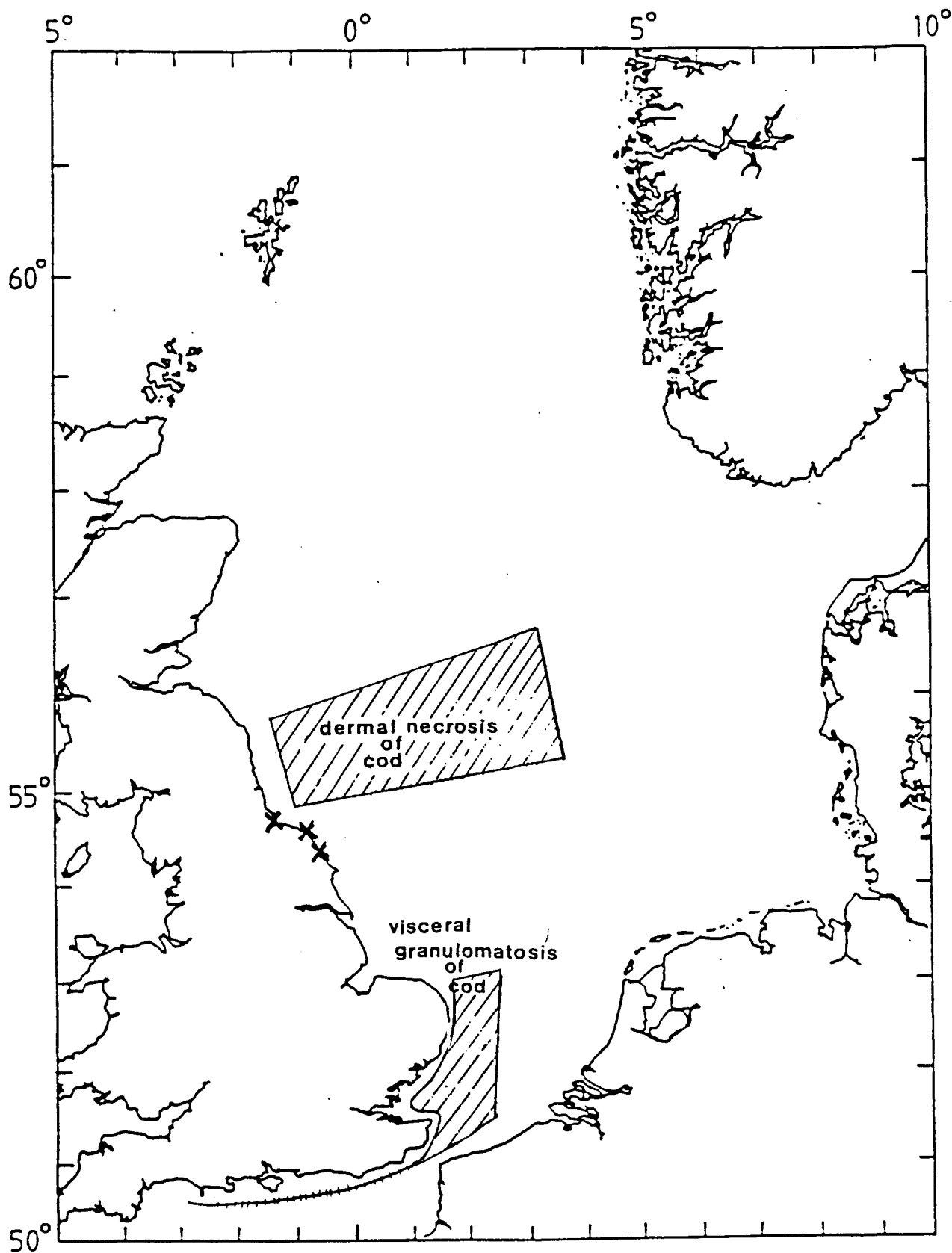


Figure 1. Map showing the distribution of dermal necrosis and visceral granulomatosis in cod following reports and investigations in 1988: X indicates ports where landings of cod with dermal necrosis are recorded.

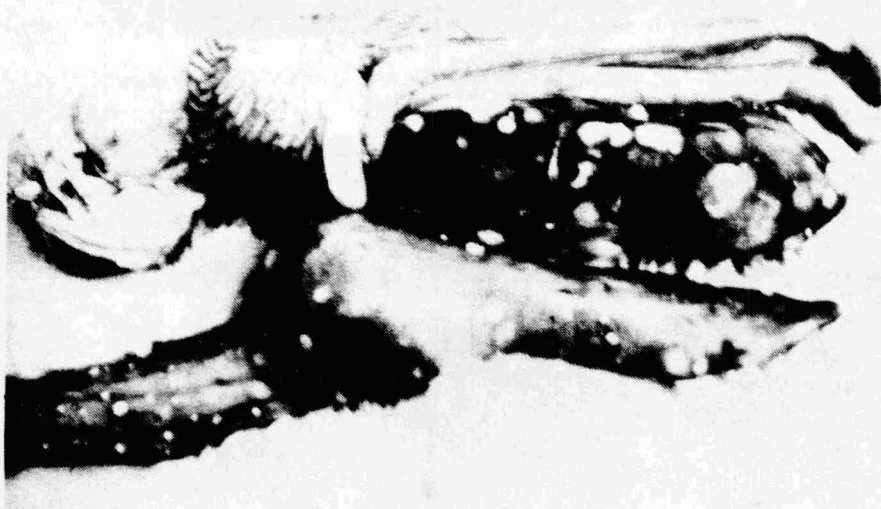


Figure 2. Cod viscera displaying large white nodules on liver and spleen.



Figure 3. Cod viscera displaying multiple small grey nodules on liver and spleen.

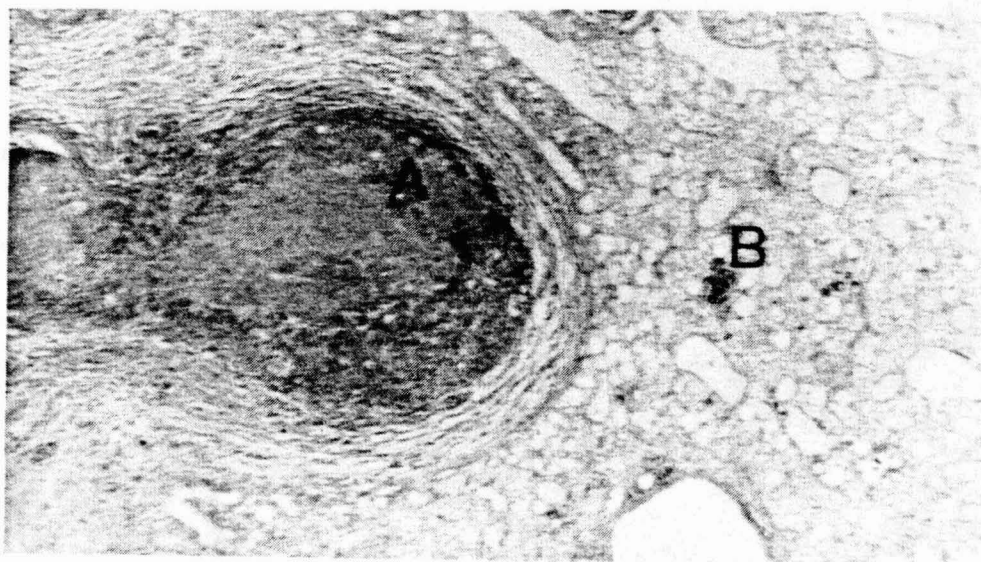


Figure 4. Histological section of cod liver showing a typical granuloma (A) and an "early" lesion (B). HE x 40.

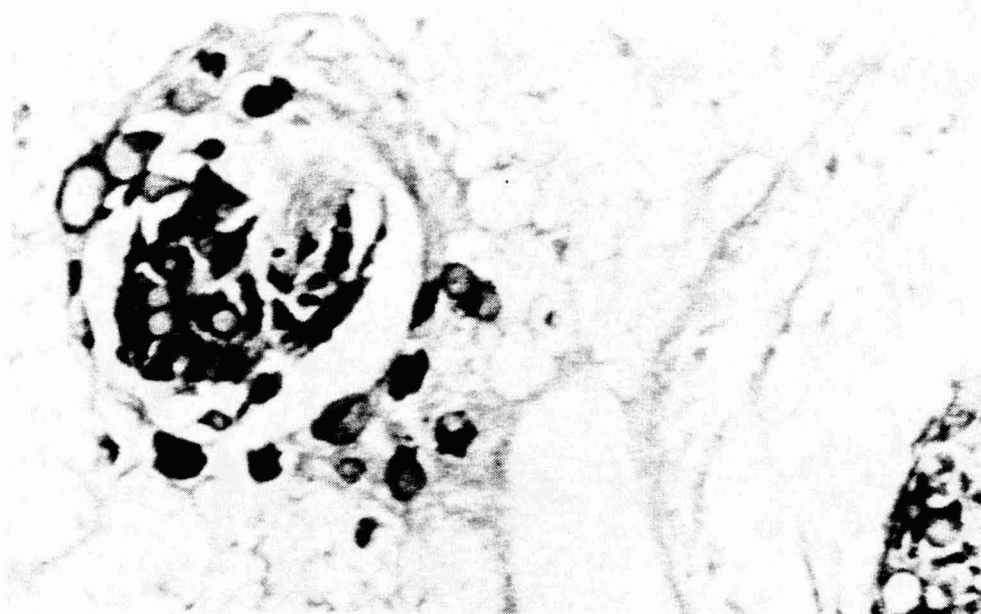


Figure 5. Histological section of cod liver showing agyrophilic substance in an "early" granuloma (Fig. 4 (B)). Grocott-gomori's methenamine silver method x 500.

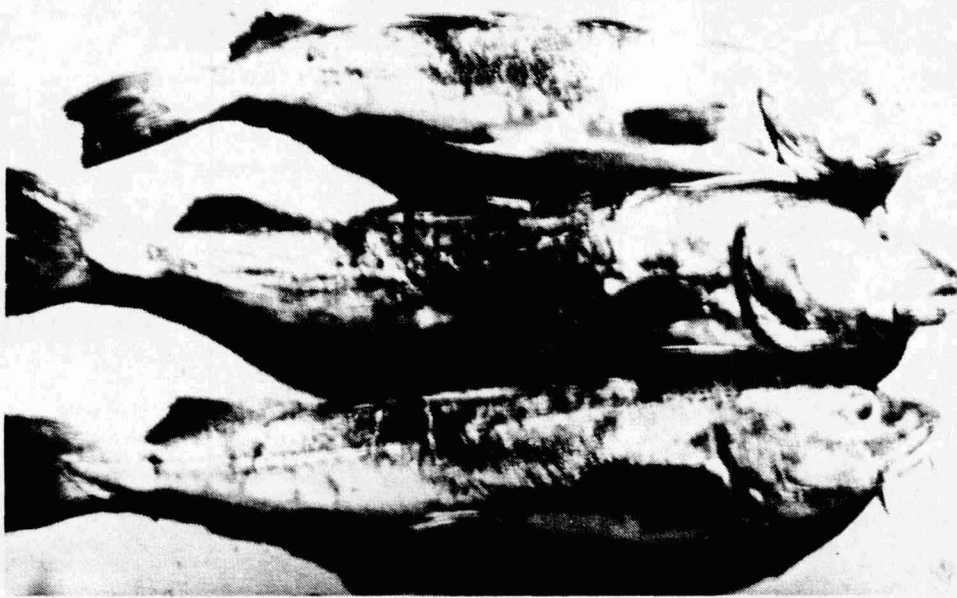


Figure 6. Large cod exhibiting dermal necrosis.