worth mentioning the remarkable behaviour of rats exposed to warfarin alone on certain East Anglian farms: they seek out marram grass on the sand dunes, since it has a naturally high content of vitamin K.

The potential of calciferol as a rodenticide have recently been studied by Greaves *et al.*[2] They found that in experimental conditions nearly all animals fed on 0·1% of this vitamin were dead in two days. Resistance to warfarin in *R. norvegicus* may be correlated with susceptibility to calciferol, and further studies on mixtures of calciferol and warfarin showed that there was an additive effect between the components.

Field trials made by Rennison[3] confirmed that 0·1% calciferol, alone or combined with warfarin, was an effective poison for warfarin-resistant rats; however, the technique needed to obtain the best results may present difficulties in the hurly-burly of wide-scale usage. Other unpublished field trials have produced rather variable and not easily explained results, though it is generally agreed that the new combined preparation, despite the care needed in its application, always justifies a trial for infestations known to be warfarin-resistant. Fortunately when all else fails we can still fall back on highly lethal compounds like zinc phosphide and fluoracetamide, but they must be applied circumspectly.

[1] Thrower, W. R., *Transactions of the Medical Society of London*, 1971, **87**, 78.
[2] Greaves, J. H., Redfern, R., and King, R. E., *Journal of Hygiene*, 1974, **73**, 341.
[3] Remison, B. D., *Journal of Hygiene*, 1974, **73**, 361.

Fish Farms and Botulism

Botulism is a horrific disease; by the successive involvement of cranial nerves it deprives its victim of sight, speech, and the power to swallow, and unless in a respirator he will die of asphyxia because eventually unable to breathe. Though mercifully very rare it is consequently dreaded, and a nightmare in the canning industry, since inadequate processing of a single batch of some food may cause it in the consumer. It is an intoxication, the toxin being formed under anaerobic conditions in food containing living spores of *Clostridium botulinum*, an organism fairly widely distributed in soil. Meat products and particularly home-canned vegetables are commonly responsible, but fish is another source, notably in countries bordering the northern Pacific Ocean, Canada, Alaska, and Japan. In that region the organism, almost always of type E, exists in the silt on the sea bottom and is swallowed by fish and may be found in their gut contents. The bacterium has no effect on the fish, but it may contaminate fish products—often eaten uncooked, and in forms sounding somewhat unsavoury, in these areas.

Though much less common elsewhere, fish botulism is not entirely confined to the northern Pacific shores. In a communication to a symposium held in Moscow in 1966[1] Cann *et al.* of the Torry Research Station, Aberdeen, reported a study of the distribution of *Cl. botulinum* type E on both sides of the North Sea in herring and white fish and in deposits on the sea bed. Samples of bottom deposit from the neighbourhood of Aberdeen and fish landed from British fishing grounds were uniformly negative, but herrings caught in Norwegian waters and bottom deposits from the Skaggerak and Kattegat gave about 50% of positive isolations.

Fish farming is a new and rapidly expanding industry, and it is natural to inquire what risk it may entail of yielding contaminated fish products. Burns and Williams[2] of the Highland Health Board studied three farms, two of which had concrete "raceways", one taking fresh water from a loch and the other a mixture of fresh water and sea water, and a third was a "mud farm", in which water from a stream passed through a presumably natural channel. Trout of marketable size were netted from each and mud was collected from the bottoms of all the tanks; these were all examined for the presence of *Cl. botulinum*. All specimens from the first two farms were negative, but the organism was found in 1 out of 21 fish from the third farm and in 9 out of 37 samples of mud from it (the numbers of mud samples from the first two were only 5 and 2). All the organisms were of type B. This is perhaps not surprising, since type B is the commonest found in European soils and this farm was supplied with fresh water; type E is found mainly in a marine environment.

There is, then, a risk of contamination in fish raised in natural water channels with mud bottoms; whether these present any advantage—or drawback—except presumably the lesser cost of establishing them these authors do not discuss. *Cl. botulinum* type E was found in German fish farms after the deaths of three consumers of vacuum-packed smoked trout fillets. This type of packaging (if it merits its description) should secure anaerobiosis just as well as canning, and such products should perhaps be viewed with suspicion, unless they have been sterilized. High energy irradiation is presumably a possibility for this, though perhaps not commercially feasible. Another possible safeguard is efficient smoking. Consumers of smoked salmon, the most popular form of raw fish in many countries, may find some reassurance in the work of Nielson and Pederson[3] of Denmark, who studied the effect of smoke constituents on the germination of *Cl. botulinum* spores artificially introduced into slices of the flesh of salmon from the Baltic. They say nothing about essential oils, which might be thought the main bactericidal constituent in smoke, but tested the effects of phenols, which failed to prevent spore germination, and of formaldehyde, which they found in a concentration of 20-40 μg per gram of smoked salmon, and showed this to be effective in short term experiments. What sort of smoke was produced to yield this constituent—incidentally a prohibited food additive—was not revealed. A smoked salmon exposed in the usual way to the atmosphere should almost certainly be safe anyhow.

[1] Cann, D., *et al.*, in *Botulism 1966. Proceedings of the Fifth International Symposium on Food Microbiology*, ed. M. Ingram and T. A. Roberts. London, Chapman and Hall, 1967, p. 62.
[2] Burns, G. F., and Williams, H., *Journal of Hygiene*, 1975, **74**, 1.
[3] Nielson, S. F., and Pedersen, H. O., in *Botulism 1966. Proceedings of the Fifth International Symposium on Food Microbiology*, ed. M. Ingram and T. A. Roberts. London, Chapman and Hall, 1967, p. 66.

Of Moles and Malignancy

The mole that could be malignant is a common problem for general practitioners. It may first be noticed during a medical examination, or the patient's alarm may be triggered off perhaps by a magazine article or by a recent death in the family from cancer.

Since most such lesions prove benign an attitude of optimism is sound, but in an individual patient it may be far from easy for the general practitioner (or the dermatologist) to be certain on clinical grounds of the type of lesion, let alone