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CURRENT FISH DISEASE EPIDEMIC IN THAILAND

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Abstract

Studies on the pathology, etiology, prophylaxis, and therapy of the current fish disease epidemic in Thailand are reviewed. The origins of the disease are still not entirely clear. However, the abnormally low temperature during the cool months of the year may be an important predisposing cause. Fish under stress by environmental changes usually eat less, become weak and more susceptible to pathogens. Important pathogens identified include virus and the bacteria, Aeromonas hydrophila. *Other organisms, such as fungi, protozoa, and ectoparasites are also found to be associated, but are less prominent. The present levels of insecticides and herbicides in natural waters are, however, unlikely to be a predisposing cause of this disease.*

Introduction

During 1982-1985, Thailand faced a series of serious fish disease epidemics, causing wide - spread death of fish in 52 provinces in the country. Loss in 1983 was estimated at one hundred and sixty million baht (approximately seven million U.S. dollars). The epidemic occurred in natural waterways and also in fish ponds. Many species of freshwater fish, especially those which are very popular for local consumption, such as snake-head fish *(Ophicephalus striatus),* eel *(Fluta alba),* and snake-skin gouramy *(Trichogaster pectoralis),* were affected. The dead fish were found to have large ulcers on the head, lips, and cheeks, as well as on the body and tail.

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The first serious fish epidemic took place in the south of Thailand, during early 1982. The second outbreak occurred between late 1982 and early 1983. The Government took immediate action to combat the disaster and identify the cause. The third and fouth outbreaks occurred between late 1983 - early 1984 and between late 1984 - early 1985, respectively. Thus the outbreaks are usually associated with the cooler months in Thailand (i.e., November - February).

This fish epidemic is not just a problem of Thailand but can be considered a regional one. The epidemic first occurred in northern Australia about 8 - 9 years ago[1,2]. When it spread to Java in late 1980, severe losses were experienced by fish farmers[1,2,3]. In 1981 it spread to Malaysia and entered the southern part of Thailand in the following year[2]. The central part of Thailand was badly hit in 1982–1984 as has been mentioned. Now the epidemic has moved to the northern part of Burma as well as Laos[2]. The epidemiology of this disease is similar to the ulcerative dermal necrosis (UDN) which was a problem in northern Europe during 1964 - 1976[4].

This paper aims at summarizing the present knowledge about the pathology, etiology, prophylaxis, and therapy of this fish disease.

Pathological Studies

Several freshwater fish species in both natural waterways and culture ponds have been infected by this disease epidemic. The most serious infected species were carnivorous species, i.e. *Ophicephalus* spp., *Notopterus notopterus,* and *Fluta alba*[5]. Omnivorous species such as *Puntius gonionotus* and *Tilapia nilotica* and the scale-less fish like *Clarias* spp. and *Pangasius* spp. were less susceptible to this disease. Only a small number of these fish showed clinical signs of lesion, which in fact might be caused by other diseases. It was very interesting to note that aquatic invertebrates such as the giant prawn *(Macrobrachium rosenbergii)* which were densely cultivated in the infected areas did not show any clinical symptom. The prawns could withstand the disease even though they were in the same pond as seriously infected fish.

Several pathological studies were carried out on snake-head fish *(Ophicephalus striatus)*. This was because the species is economically important and was seriously infected by this disease. Therefore, the following explanation will deal mostly with studies on this species.

The early symptoms of the disease when the fish are infected are dark coloration and sluggishness; sometimes the fish float, although they are still under the water surface. The fish in the culture ponds tend to eat less or do not eat at all. Small grey erosions develop on the head and fins. Early lesions are usually restricted to these places. Subsequent to this stage, the lesions will eventually develop on other parts of the fish body and become large ulcers within a few days, followed by death (Figs. 1 & 2).

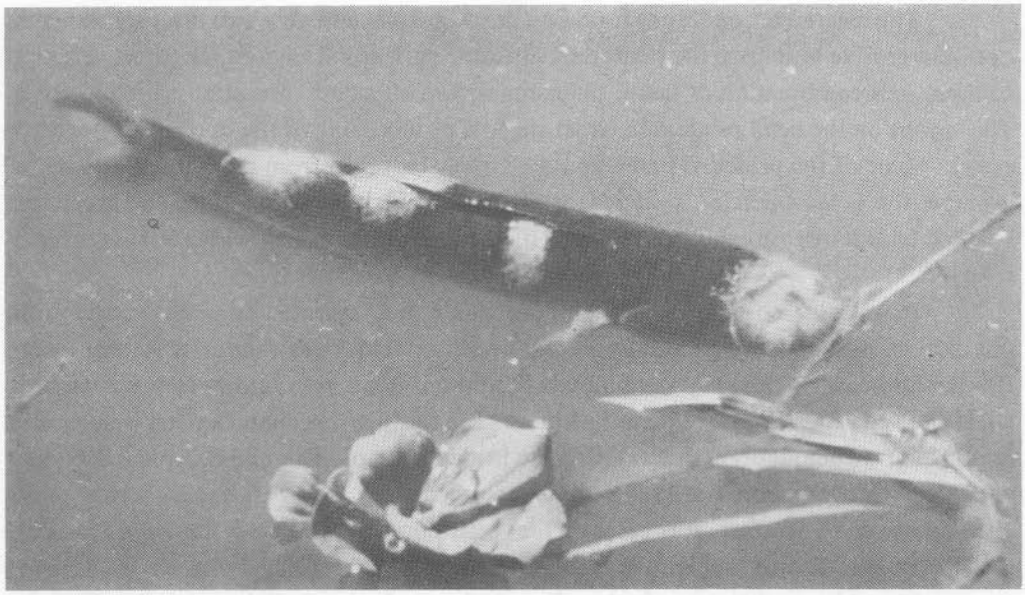

Figure 1. The diseased snake-head fish floats under the water surface. The large ulcers on the fish body are infected by bacteria, protozoa, and fungi.

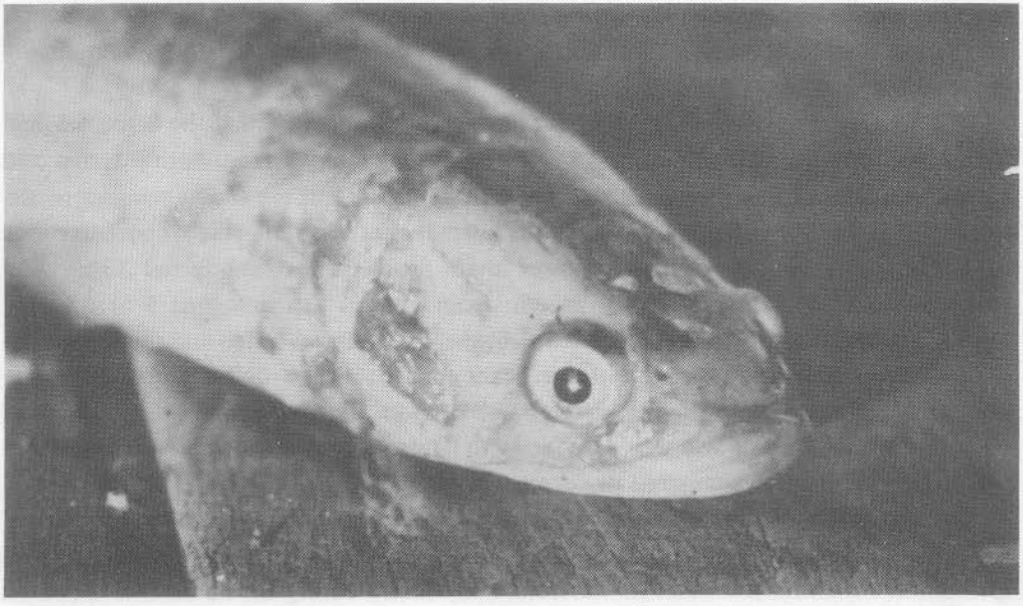

Figure 2. A large ulcer on the head of snake-head fish.

The ulcerative conditions found on dead snake-head fish were variously shown as open-cut erosive lesions to the peritoneal or to the pericardial cavities, or to the vertebral column, or a combination of these. Sometimes the fish's body was almost broken apart. The lesions on the head resulted in either the loss or loosening of the opercular bone(s) or eye(s). Some of the principal bones could be freely loosened from their articulations, for example the jaws, opercles, pectoral, pelvic and caudal elements. The live specimens infected by this disease exhibited the same ulcerative conditions but with a lesser degree of severity[6].

It was also observed that slightly or moderately infected fish can recover from this disease. A study during the 1984 outbreak revealed that 50% of the infected snake-head fish could recover when they were moved to another pond with improved water quality. Ulcers healed after a variable period of time. The healing was characterized by excessive numbers of melanophores which caused the healed ulcer places to be conspicuously dark in color. Recovery from the 1985 outbreak seemed to be much greater than that of the 1983 outbreak. Therefore, it is believed that resistance to this disease has been gradually built up in fish through subsequent generations.

The histopathology of the disease in snake-head fish was described by Tesaprateep *et al.*[7]. and Tesaprateep *et al*,[8]. Necrosis of the gill lamella was a common feature in the diseased fish. Partial lesion and necrosis of the liver, spleen, and kidney were observed in severely infected fish. Nevertheless, such pathological features were not observed in the slightly infected fish. This finding agrees with the report of Limsuwan and Chinabut[9]. The pathobiology of the disease was possibly linked to bacterial toxemia. The lesion started from the skin, spread to the underlying musculature, and finally involved the systemic organs[8].

During the outbreak in 1984, Tangtrongpiros *et al.*[10] studied the hematological change in snake-head fish. Examination of 283 blood samples from both infected and normal snake-head fish revealed that only the hemoglobin in the blood samples of the infected fish was significantly lower than the normal ones. Serum protein concentrations of the infected fish were slightly lower than those of the uninfected fish. Gel electrophoresis revealed that serum protein from normal fish indicated 5 or 6 major bands. However, in the severely infected fish, the 4^{th} band was faded and the 5^{th} and/or the 6^{th} band was clearly stained. No significant changes in electrophoretic patterns were observed in the sera from slightly infected fish or from those which recovered from the infection[11]. The infected fish were also found to have significantly lower levels of vitamin A and C in the liver as compared to normal fish[12].

Disease agents which were observed in association with fish indicating signs of ulcerative pathology are shown in Table 1. Among these disease agents, *Aeromonas hydrophila,* the hemolytic bacteria, was commonly found during the outbreaks. Poonsuk *et al.*[13] reported that *A. hydrophila* could be isolated from necrotic skin, gills, liver, and

blood of the diseased snake-head fish during the 1983 outbreak at the rates of 96.5%, 96.5%, 87.5%, and 100%, respectively. However, this organism could be isolated at a lower percentage from diseased fish during the 1984 outbreak (73.1%). It was further reported that fish at the early stage of infection (small grey erosion at the head) are not associated with *A. hydrophila*.

As regards virus, Wattanavijarn *et al.*,[14] observed virus-like particles in the necrotic muscle fibers and endothelium of capillaries. Oval, round, and kidney shaped pheomorphic forms with nucleocapsid-like structures surrounded by an envelope with budding processes were observed. It was later reported by Fryer[15] that one of the virus-like particles is IPN virus, similar to the Ab serotype.

Unlike bacteria, virus was detected in all diseased fish but not in fish lacking symptoms of disease. Much attention has been recently given to virus, because it may have some association with the primary cause of this fish disease epidemic. Figs. 3, 4, and 5 show viruses which have been detected in various organs of the diseased fish.

Table 1. Disease agents observed in association with fish showing signs of ulcerative pathology.

Group	Scientific Name	References
Bacteria	*Aeromonas hydrophila*	13, 16, 17, 9
	Aeromonas sobria	18
	Aeromonas punctata	17
	Edwardsiella tarda	17
	Flavabacterium	17
	Pseudomonas	17, 13
	Vibrio parahaemolyticus	17, 16
	Alcaligenes faecalis	16
	Streptococus	17
Fungi	*Achlya*	19, 20
	Saprolegnia	9
Protozoa	*Epistylis*	9
	Costia	9
	Trichodina	9, 21
	Trypanosoma	22
Trematode	*Dactylogylus*	21
Virus	(Virus-like particles)	14
	IPN virus similar to Ab serotype	15

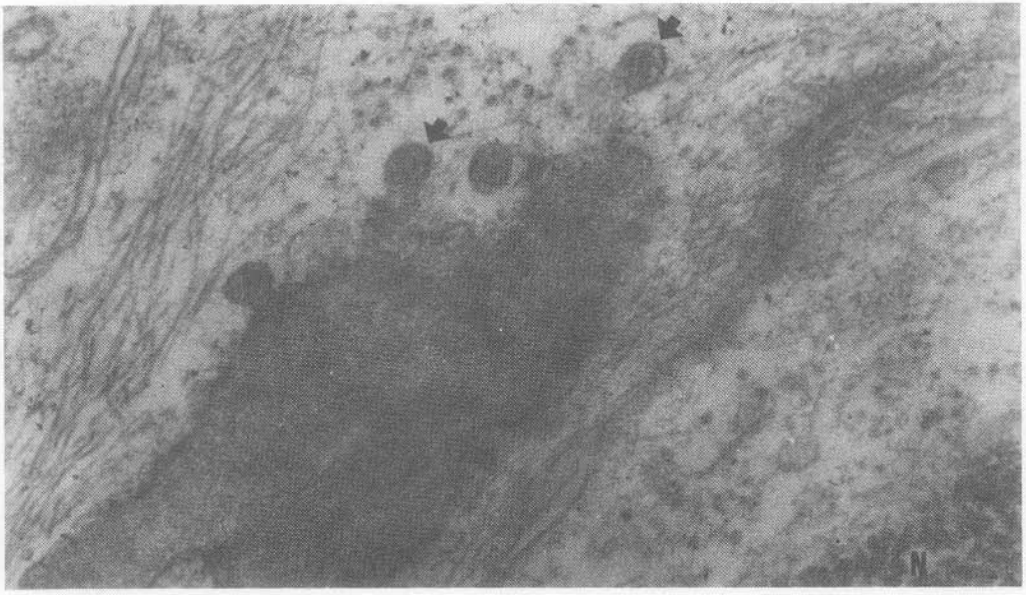

Figure 3. Viruses in the spleen cell cytoplasm of a diseased snake-head fish *(Ophicephalus striatus)*. Note cell organelles are destroyed. N = nucleus. Magnification 103,000 X Photography by Dr. Wattana Wattanavijarn, Faculty of Veterianary Science, Chulalongkorn University.

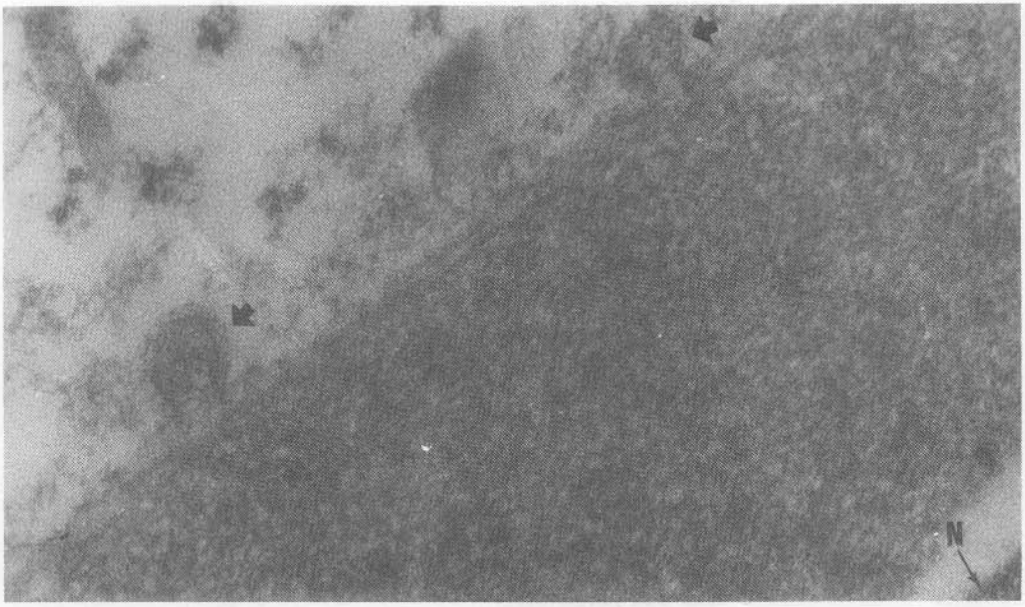

Figure 4. Viruses with the outer projections (arrows) budding from the red blood cell of the sick snake-head fish. N = nucleus. Magnification 112,000X. Photography by Dr. Wattana Wattanavijarn, Faculty of Veterinary Science, Chulalongkorn University.

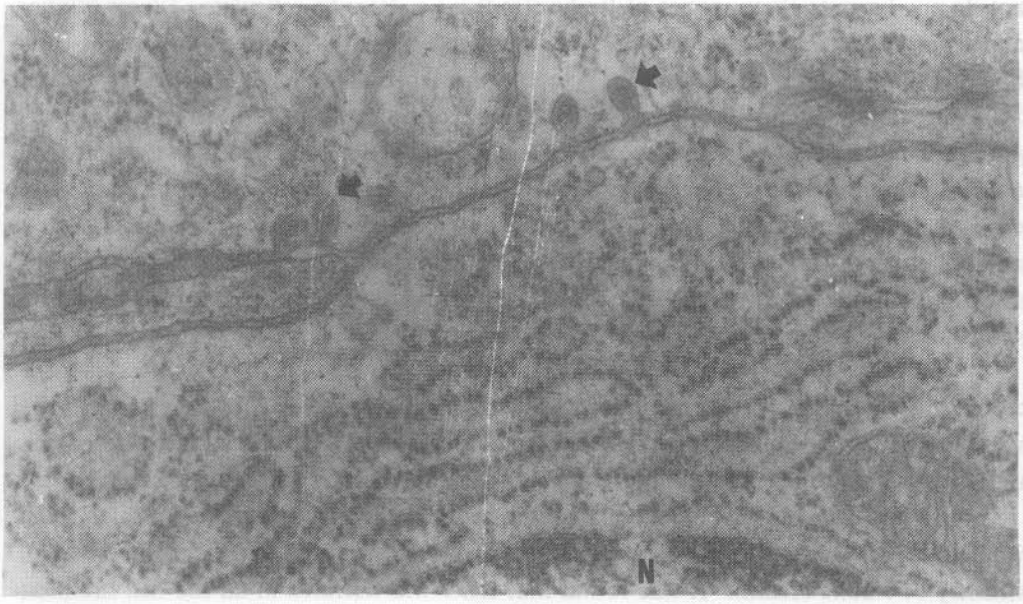

Figure 5. Budding of virions from a cell of the snake-head fish. N = nucleus. Magnification 53,800X. Photography by Dr. Wattana Wattanavijarn, Faculty of Veterinary Science, Chulalongkorn University.

Studies on Predisposing Causes

It is generally accepted that stress is a very important factor in outbreaks of infectious diseases of fish.[23,24] There are many causes of stress, for instance temperature changes, changes in pH, high content of dissolved organic substances in the water, overcrowding, oxygen depletion, presence of toxic substances, and nutritional deficiency. Besides stress, the deteriorating genetic condition of the fish themselves could be another factor which impairs the resistance to the disease. Such conditions can occur if the fish used for propagation and culture purposes have never been genetically improved.

During the outbreak in early 1983, paraquat was believed to be the main predisposing cause of the disease epidemic. According to the announcement of the Department of Fisheries (Thai Fisheries Gazette, Vol. 36, No. 3, 1983), paraquat, a herbicide, was detected in natural waters at a concentration of 0.030-0.051 ppm. It was believed that the herbicide at this level would be harmful to fish and could cause wounds on the body of the fish, making them more susceptible to bacterial infection. Later a series of experiments was conducted to confirm this announcement. The results, however, revealed that paraquat was unlikely to be the primary cause as believed. Menasveta *et al.*[25] found that paraquat at a level 50 times more than those reported by the Department did

not do any harm to three species of freshwater fish i.e. *Poecilia reticulata, Tilapia nilotica,* and *Ophicephalus striatus* (14 days exposure); acute toxicity of paraquat in terms of 96 hours median tolerance limit (TL_m) to the three previously mentioned species were >2.65 ppm, > 17.6 ppm, and 10 ppm, respectively. Exposure of snake-head fish to 1.0 ppm paraquat in combination with *Aeromonas hydrophila* at 10^6 cells/ml for seven days did not result in infection[26]. Singhaseni and Tesprateep[27] reported that *Puntius gonionotus* exposed to 2.0 ppm for 12 days did not show any marked histological changes in various organs; however, at 4.0 ppm swelling of the gill filaments was observed. Kanchanopas[28] also reported that snake-head fish exposed to paraquat at 3.0 ppm for 14 weeks did not show any significant histological changes.

Since paraquat is not accumulated in fish muscles, the toxicological effects of the chemical by means of accumulation is less likely[29]. It was also reported that paraquat did not combine with plasma protein or with tissue homogenates. Paraquat is absorbed less and once it is absorbed, the elimination rate is very fast [29].

With regard to insecticides, 127 samples of water, fish, and fish food from the problem areas were collected and analysed by the Department of Medical Science, Ministry of Public Health. It was found that DDT was detected in all the fish samples; however, the levels were still within the acceptable limit [30]. DDT was detected in one out of 24 of the water samples [30]. Both carbamate and organophosphorus were not detected in either the fish or the water samples[30]. Heavy metals in the water samples were analysed by a Chulalongkorn University research team [31]. The concentrations of these metals were somewhat comparable to those reported six years ago by Menasveta [32].

Temperature change is considered to play an important role as one of the predisposing causes. It is interesting to note that the disease breakout usually coincided with the cool months, i.e. November through January. The peak of the infection rate was usually at the end of December and early January, the coolest period of the year.

In the past 3 years, the temperature during the cool season was abnormally low compared with the preceeding years. This might be due to the influence from the El Nino phenomenon (Southern Oscillation).

It is generally accepted that a change in temperature can cause stress in fish. A four to five degree celsius drop in the water temperature could considerably lower the fish metabolic rate. The fish will then eat less and become weak. In addition, it has been reported that a decrease in temperature can increase the population growth rate of *Aeromonas hydrophila*[33]. This might be attributed to the higher infection rate of this disease agent at a low temperature, as verified by Mahamontri *et al.*[34]

Snieszko[35] stated that "it is well known, from epidemiology, that an infectious agent causes a disease of the host if environmental conditions are right. The influence of each subset is variable - disease breaks out only if there is a sufficient relationship between them (Figure 6)".

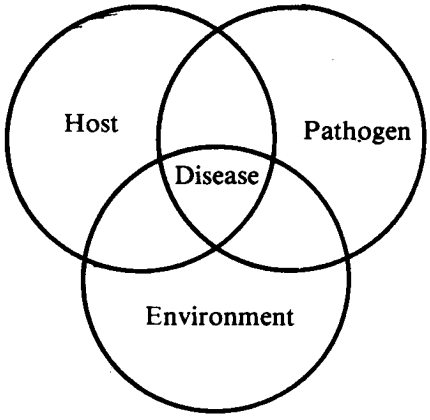

Figure 6. An overt infectious disease occurs when a susceptible host is exposed to a virulent pathogen under proper environmental conditions. After Snieszko[35].

In the case of the fish disease epidemic in Thailand, low temperature might be an important physical factor in the environment subset. Chemical factors such as changes in pH and alkalinity might also be contributing factors, but to a lesser degree. As for the pathogen subset, *Aeromonas hydrophila* and/or virus are probably the most important.

Studies on Prophylaxis and Therapy

During the second outbreak in early 1983, several measures for prophylaxis and therapy were suggested to fish farmers. According to the second announcement made by the Department of Fisheries (Thai Fisheries Gazette, Vol. 36, No. 3, 1983), fish farmers were recommended not to change the water in their culture ponds. If a water change was really necessary, it was suggested that incoming water should be filtered through a charcoal bed in order to reduce the amount of pesticides. Potassium permanganate was recommended to be used as a prophylatic chemical at the rate of 5 kg per 1 rai (1 rai = $1/_6$ ha) of a 1 m deep pond; this is approximately 3 mg/l. As for therapy, administration of chloramphenicol, kanamycin, neomycin, and bactrim were suggested to be used at the dose of 2-3 grams/1 kg of food for a duration of 5-7 days.

Some of these measures caused reactions from some groups of people. With respect to the use of potassium permanganate ($KMnO_4$), the Chemical Society of Thailand argued that the amount of $KMnO_4$ recommended by the Department was not sufficient to disinfect *Aeromonas hydrophila* in water, and if the concentration of this chemical was increased to the proper level, it would be too costly and the fish would be killed before the chemical could inactivate the bacteria. This argument is reasonable

because in water MnO_4^- quickly reacts with organic matter and other reduced substances before it can react with bacteria. Therefore, if dissolved organic matter in water is high, the toxicity of $KMnO_4$ to bacteria will be reduced. Most of the snake-head fish culture ponds have a high organic content due to the food-residue.

Tachushong and Saitanu[36] reported that $KMnO_4$ at 5 mg/l could not inactivate *Aeromonas hydrophila* in fish pond water. However, in clean water, the result was the opposite.

Antimicrobial susceptibility testing of *A. hydrophila* isolated from the 1983 breakout showed that this kind of bacteria was highly sensitive to neomycin, chloramphenicol, erythromycin, kanamycin, and tetracycline.[37] Saitanu and Poonsuk[38] reported that curing the diseased fish by a combination of three antibiotics was more effective than a combination of two or a single antibiotic.

In connection with the use of antibiotics, a group of medical doctors warned that some antibiotics such as bactrim should not be used in an uncontrolled environment. They were afraid that some human contagious disease could build up resistance to such antibiotics.

During the third outbreak in 1984, lime and salt were suggested as the prophylactic chemicals. This method resulted in some beneficial results. The lime increased the water alkalinity and stabilized the pH condition. Salt could also control some external parasites and temporarily inhibit the activity of bacteria.

The increase of disease resistance in fish by improving the nutritional requirements should receive more attention. The cultured fish should be fortified with vitamin C and other important vitamins well ahead of time (i.e. before the cool season). It was reported by some aquaculturists that snake-head fish fortified with vitamin C had less chance to be infected by this disease.

During the recent disease breakouts, a quarantine system has never been adopted although it has been suggested by some academicians. This enables the disease to spread quickly so that it is uncontrollable.

Concluding Remarks

During the past three years when fish disease outbreaks have occurred in Thailand, experts have been divided in their opinions on the underlying causes of the epidemic. Some believed that pesticides and herbicides were the main cause of the epidemic, whereas others contended that herbicides and insecticides had little to do with the problem. Until now, a number of research findings have come up with different explanations.

The cause of the fish epidemic could be divided into two parts, i.e., predisposing causes and the cause of death. The predisposing causes appear to be a combination of

abnormally low temperature, and changes in the acidity of the water brought about by heavy rain. These environmental changes cause stress in the fish as they try to adapt themselves to changing water conditions. This environmental stress may make the fish eat less and eventually become so weak that they are easily susceptible to virus and bacteria infections. Such infections may be caused by a virus or by the much publicized cause of death, the bacteria *Aeromonas hydrophila.* Other organisms such as fungi, protozoa, and ectoparasites may play a less prominent role in causing infection. The present levels of insecticides and herbicides in natural water probably had little to do with this fish disease epidemic.

The above explanations are some conclusions that can be extracted from the three years study. Nevertheless, there is still a need for further verification on the causes and other aspects of this fish disease. It should be noted that this fish epidemic has a tendency to recur every year during the cool months, especially in the higher latitudes of Thailand and possibly in Laos, Burma, and the southern part of China.

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บทคัดย่อ

ได้ทำการศึกษา พยาธิวิทยา สาเหตุแห่งการเกิดโรค การป้องกันและการรักษาโรคระบาดปลาที่เกิดขึ้นในประเทศไทยในระยะสามปีที่ผ่านมา ในขณะนี้รายังไม่อาจสรุปต้นเหตุแห่งการเกิดโรคได้อย่างแน่ชัด อย่างไรก็ดี อุณหภูมิน้ำที่ต่ำอย่างผิดปกติในช่วงฤดูหนาวอาจเป็นสาเหตุเบื้องต้นที่สำคัญ เชื้อโรคที่ตรวจพบและน่าจะเป็นสาเหตุของโรคระบาดได้แก่ ไวรัสและแบคทีเรีย โดยเฉพาะอย่างยิ่ง *Aeromonas hydrophila* เชื้อโรคชนิดอื่น เช่น เชื้อรา โปรโตซัว และพาราสิตภายนอก อาจมีส่วนร่วมทำให้เกิดโรคแต่ไม่เด่นชัด ระดับของยาฆ่าแมลงและยาฆ่าวัชชพืชที่ตรวจพบในแหล่งน้ำไม่น่าจะเป็นสาเหตุเบื้องต้นที่ทำให้เกิดโรคระบาดปลา