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Effects of Sewage Treatment Plant Effluents on Fish:

A Review of Literature

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EFFECTS OF SEWAGE TREATMENT PLANT EFFLUENTS ON FISH: A REVIEW OF LITERATURE

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INTRODUCTION

This literature review was a part of the research project, "Research on the Chesapeake Bay to provide a knowledge base for waste outfall management," conducted by the Chesapeake Research Consortium, Inc.

The primary objective of this review was to prepare a digest of the known effects of sewage treatment plant effluents on estuarine fish drawing from world wide literature. Most studies on the effects of sewage effluents on fish life to date, however, have been devoted to freshwater fish; very little has been done on estuarine and marine fishes. It was considered that many studies which have been done on freshwater fish are essential references for understanding estuarine and marine fish. Thus, in this review not only estuarine fishes but also freshwater and marine fish are included. It is hoped that this review will be useful for management agencies at federal, state, and local government levels which deal with the problem of sewage disposal and the protection of aquatic life. It will also provide the basic knowledge for designing future research projects.

Search of literature was made from July 1973 to June 1974, mainly through Water Pollution Abstracts, Pollution Abstracts, Biological Abstracts, Commerical Fisheries Abstracts, Sport Fishery Abstracts, Selected Water Research Abstracts, Water Research Catalogue, and other available sources. Only that literature relating to domestic sewage was searched. Since domestic sewage contains constituents which directly or indirectly affect fish life, literature relating to known toxic or damaging constituents, and water quality parameters which are known to be affected by domestic sewage effluents damaging to fish life, has been searched. The examples relate to domestic sewage effluents as a whole, as well as residual chlorine and chloramines, detergents, ammonia, hydrogen sulfide, sludge, dissolved oxygen, algae, and bacteria.

I should like to express my deep appreciation to Dr. L. Eugene Cronin, Director of Natural Resources Institute, University of Maryland, for his encouragement during the study. Mr. K. Y. Chang, a graduate student, and Mr. Stephen P. Klett, Biological Aide of the Institute, assisted in the search of literature. Mr. Stuart Bohacek helped edit and Mrs. Irene Baker and Mrs. Noreen Cryan typed the report.

RECOMMENDATIONS

In this review of the literature regarding the effects of sewage treatment plant effluents on fish life, it is found that very few studies have been done on estuarine and marine fish as compared to freshwater fish. It is often felt that it is difficult to establish a satisfactory management policy and methods to protect freshwater fish from sewage polution, even though there is a mountainous accumulation of literature in this field at the present time. Unquestionably, it is almost impossible to have a satisfactory management policy and methods for estuarine and marine fishes simply by referring to the existing knowledge in this field which is so limited. Therefore, the following several studies on the effects of sewage effluents on estuarine and marine fish, which I feel to be urgent and important, are recommended.

1. There is almost complete lack of knowledge of the chemistry of chlorine and its reaction with sewage constituents in estuarine and marine water. The effects of salinity on the formation of various species of toxic combined chlorines, particularly chloramines, need to be learned. Knowledge about their toxic effects on estuarine and marine fishes is extremely limited. There are no published studies relating to the toxicity of free chlorine or chloramines on Chesapeake Bay fish.

2. The dissociation kinetics of ammonia or ammonium salts to produce toxic un-ionized ammonia in freshwater is well known. However, there is apparently no knowledge of the chemistry of ammonia to produce toxic un-ionized ammonia in brackish and marine water. Understanding this chemistry is the only way to determine the toxic form of ammonia in estuarine and marine water so that the toxic effects of ammonia on estuarine and marine fish can be studied. There is virtually no knowledge of the lethal and other biological effects of toxic un-ionized ammonia on the major estuarine and marine fishes, including those species sound in the Chesapeake Bay, and these should be determined.

3. In contrast to soap, toxicity of synthetic detergents may increase with increasing salinity. This suggests that a concentration of synthetic detergents which is sublethal to freshwater fish may be lethal to fish in estuarine and marine water. Although in the past there have been several studies on the effects of synthetic detergents on estuarine and marine fish, no detailed study has been done of the effects of salinity on the toxicity of synthetic detergents on estuarine and marine fish, particularly those species in the Chesapeake Bay, and such research is needed.

The presence of sublethal concentration of detergents in water may damage the gills so as to increase the suscep-

tibility of the fish to other toxic pollutants. Thus, synergistic reaction of the detergents to other common toxicants in estuarine and marine water needs to be studied.

4. Hydrogen sulfide may occur more frequently and in much higher concentration in estuarine and marine water than in freshwater. This chemical may be one of the most toxic chemicals to be found in the water just above the bottom covered with sewage sludges, or in the water column polluted with sewage effluents or other organic materials. Estuarine or marine fish which use the bottom habitat, particularly in their eggs and larval states, will be the ones which will be strongly affected. To date there is almost no knowledge of the effects of this important toxicant on estuarine and marine fishes, and it ahould be obtained.

5. There is extensive literature on the effects of suspended solids, other than sewage sludges, on estuarine and marine fishes. Because sewage sludges have different properties from other suspended solids, it is necessary to determine whether there is also a difference in their effects on fish life.

6. In the Chesapeake Bay there are five species of toxic dinoflagellates which have been known to cause incidents associated with either fish kills or shellfish poisoning elsewhere in the world, We do not know whether they have caused fish kills in the Bay, but at least their presence in the Bay indicates a potential for possible incidents in the future, if the environmental condition becomes favorable. Therefore, for the prediction and prevention of such incidents in the future in the Bay, it is necessary now to study the ecological and nutritional requirements of these dinoflagellates, and particularly how these requirements may be affected by sewage treatment practices.

7. Sewage effluents have complicated constituents. Some of them are toxic to fish life, but others may be beneficial to fish life. Research should be conducted to determine ways to manipulate the method of sewage treatment and discharge so that the sewage nutrients will be fully utilized by the aquatic sustem, including fish, without causing damaging effects. The aquaculture-sewage treatment system which purifies the sewage and, at the same time, produces useful protein seems to be a promising system for the future, and needs to be studied.

SEWAGE EFFLUENTS IN GENERAL

Fresh Water Fish

Damaging factors. Damaging effects of sewage on fish have been generally attributed to the introduction of poisonous compounds, the decrease of the dissolved oxygen as the result of bacterial decomposition and algal bloom, the increase of turbidity of water by suspended materials, the covering of fish food and spawning ground by deposited solid matter, the encouragement of fungus development, the shift in the biological balance in the waters, the increase of the incidence in fish diseases, and the production of tastes and odor in fish flesh. Because the chemical composition of sewage and the environmental condition of receiving streams are extremely complex and vary greatly in time and place, the damaging factors to fish in sewage effluents have been indicated or suspected differently by many authors.

Many investigators have observed that fish can live in undiluted sewage without harmful effects. Fish kills or fish exclusion occurs mainly as a result of the reduction of dissolved oxygen and formation of septic condition by bacteria in decomposition of organic materials. This observation was particularly well documented in the studies of a tributary of the river Colne, England, which is used as a canal to discharge undiluted sewage effluent from the Maple Lodge Works (activated sewage system of 200 to 260 thousand people). In this undiluted sewage canal there was an apparently flourishing population of warm water fish (Allen et al. 1954), and the fish were capable of living for long periods of time (Pentelow et al. 1957, 1958). There was no evidence that the effluent had any adverse effect on a mixed population of coarse fish (Anon, 1955). However, in two and half years ten fish kills were reported in this canal. The fish died when DO concentration of the effluent was below the level of 30 percent of saturation (Pentelow, et al. 1957, 1958). Allan (1953) found that there was a relationship between death of fish and the concentration of oxygen and ammonia in water. In laboratory experiments, however, a much higher concentration of ammonia was required at a given oxygen concentration to cause an equivalent kill of fish, and it was therefore concluded that in the field some other factor or factors were also important. Allan et al. (1958) kept the rainbow trout and other fish in live cages in the undiluted sewage canal and found that the fish mortality was due mainly to low dissolved oxygen. The CO_2 and heavy metals were very low in concentration, but they increased appreciably the sensitivity of the fish to low dissolved oxygen.

Sproul and Ryckman (1963) found in laboratory experiments

that domestic waste organic materials do not appear to be toxic to rainbow trout, whereas the organic extracts from the chemical, meat packing, oil refinery, paint, and soap wastes were toxic to the trout. Whiteworth and Bennett (1970) also found that the fish survived in 100 percent effluent of the Farmington municipal sewage treatment plant. The fish held in cages below the outfall showed only slight effects and there was no difference in species and composition in stations above and below the outfall. Bloodgood (1953), in his discussion of the pollution of surface water by sewage, indicated that the fish are smothered because of dissolved oxygen reduction in streams by bacteria decomposition of organic materials. Gaufin (1955) also indicated that organic wastes stimulate bacterial growth unfavorable for fish. Fish may be excluded from the septic zone. Moore (1932) found that certain species of fish such as bullheads, suckers, chubs, and carp may swim near the sewage outfalls and feed on the fresh organic matter, but farther downstream where septic condition are likely to prevail, fish are not present. They responded negatively by retreating to more tolerable areas. Turner (1960) stated that lethal quality of sewage pollution does not lie in the actual sewage, but in the effect of accumulation of sewage effluent in reducing the amount of dissolved oxygen in water. Minimum DO concentration for a mixed fish fauna was suggested to be 5 mg/l (Ellis, 1937).

In contrast to the above observations, Brinkley (1943) found that in the Ohio River, untreated sewage, when in sufficient concentration, produces a toxic area below the sewer outlet. The region extends downstrean for a variable distance until the sewage is decomposed by bacteria. Longwell and Pentelow (1935) indicated that full strength domestic sewage is toxic to brown trout. Katz and Gaufin (1953) studied the effects of partially treated sewage in Lytle Creek, Ohio. There were no fish in the area immediately below the outfall. The number of fish species, as well as the total population, increased downstream with the degree of purification. In winter and spring, the fish failed to move into the area intolerable during the summer. Thus, factors other than the lack of oxygen made the areas relatively unattractive to fish. The mixed fish fauna could occur, when DO values were often well below 5 mg/l for periods of several hours. Kussat (1969) also found that in Bow River, Canada, where sewage and industrial effluents were received, there were no obvious detrimental effects on the fauna, even when DO level reached as low as 2.7 mg/l.

Anon (1956a) showed that sewage effluents were toxic to fish, but the effluents diluted with four or five times their volume of clean water became non-toxic to fish. In such a mixture the death of fish would be due to lack of oxygen.

In the river Arbia, which received sewage from the City of Sienna, fish mortality frequently occurred. Aeration for six to eight days gave a completely stable effluent which was not toxic to fish (Gori and Cantagalli, 1957, 1959). The further experiments indicated that the toxicity is due to the presence of ammonia (Gori and Cantagalli, 1959). Tanicke and Ludemann (1967) found the effluent of an activated sludge plant was toxic to fish, but removal of free ammonia from the effluent by acidifying the effluent to pH 6.5 detoxicated the effluent. Esvelt et al. (1971) found that secondary effluent is significantly less toxic to the golden shiner, stickle-back, and rainbow trout than primary effluent.

Herbert (1962) held the fish in live cages in aquaria filled with sewage and found the sewage toxcity to fish is due to a complex interaction of pH, temperature, dissolved oxygen concentration, total un-ionized ammonia, free CO_2, and synthetic detergents. Lloyd (1964) indicated that although many sewage effluents are not poisonous, even when undiluted, fish held captive in some streams containing sewage effluent have been killed by poisons even when the DO content was well above the minimum requirement. The laboratory study indicated that ammonia, monohydric phenol, zinc, copper, and free cyanide were present in the effluent. Arora et al. (1970) found that the pH value, free residual chlorine, and total solids were observed to be important parameters for fish mortality in Renusagar, Renukoot.

Porcella et al. (1972) held trout in a live box in Indian Creek Reservoir receiving a tertiary effluent. The fish were killed in 24 hours. The authors suspected the cause to be ammonia whose concentration was 2.5-7.0 mg/l in water and 12-20 mg/l in the effluent. Brown et al. (1970) studied the relationship between the water quality of some polluted surface waters in northern England from where fish were absent and the 48 hour LC_{50} values for cage trout, roach, and dace at the test sites and also for fish in aquaria containing undiluted and diluted water samples. The toxicity of the polluted freshwaters was about 65 percent of that predicted by chemical tests. Ammonia was found to be the principal poison in the waters. Recently sewage effluents after chlorination have been found to become extremely toxic to fish and their residual chlorine is found to be the principal factor causing sewage toxicity (Tsai, 1968, 1973; Basch, 1971; Zillich, 1972; Brungs, 1973).

Modes of affecting fish community. Surber (1953) stated that ordinary sewage pollution reduces the number of species to a few kinds which can adapt to life in low dissolved oxygen. Hynes (1958) indicated that there are two main effects of pollution on fish; either a simple elimination of some species, often accompanied by a corresponding

increase in the remaining species, or a replacement of the normal aquatic community by another which is adapted to the peculiar ecological conditions. Tarzwell and Gaufin (1953) indicated that in a polluted stream game fish may be reduced in number or eliminated, while the coarse species comprise the remaining population. When the coarse fish become abundant, they crowd out the game fish, resulting in a marked decline in sport fishery. The same mode was expressed by Borchardt (1969) who indicated that in hypothetical lakes eutrophication shifts game fish to coarse fish. Larimore and Smith (1963) found that when desirable enrichment changes to undesirable pollution, the effects on a fish population are first a reduction in the number of species, then a reduction in the total weight, and finally a reduction in a number of individuals. Tsai (1968, 1970) found that chlorinated sewage effluents act first as a toxic material which seriously reduces fish species diversity and abundance immediately below the effluent outfalls. It also enriches and deoxygenates the water, causing a species shift without affecting species diversity downstream.

Zonal classification of streams receiving sewage. Streams receiving sewage effluents have been divided into several polluted zones according to water quality and characteristics of biological communities, by many authors and reviewed by Warren (1971). Kolkwitz and Marrson (1908) designed four zones: polysaprobiotic, alpha mesosaprobiotic, beta mesosaprobiotic, and oligosaprobiotic, according to the degree of self-purification of water and their associated animal saprobia. This zonal classification has been commonly used by European workers. In the United States, Richardson (1921, 1928), on the basis of water quality and benthic invertebrates, also divided the sewage polluted stream into four zones; septic, pollutional, subpollutional and cleaner water. Forbes and Richardson (1913) called these four zones septic, polluted, contaminated, and cleaner water. Suter and Moore (1922) also classified streams receiving sewage into four zones but delimited the zones; recent pollution, septic, recovery and cleaner water. These same four zones were later called degradation zone, active decomposition zone, recovery zone, and cleaner water zone by Whipple (1927). Whipple's zonal classification were later adopted by many workers in this country, such as Bartsch (1948), Bartsch and Ingram (1959) and Wurtz (1955). The above zonal classification of streams receiving sewage were based mainly on water quality, bacteria, algae, and benthic invertebrates.

Regarding the zonal classification of stream according to the fish community structure, Campbell (1938) divided the stream into four zones as Whipple (1927) did. They

are recent pollution zone, active decomposition zone, recovery zone, and cleaner water zone. In the zone of recent pollution, the sewage discharged into the water is still fresh and is often relished by fish. Therefore, the fish frequently gather at the vicinity of the outfall. In the zone of active decomposition, bacterial activity decomposes organic materials and reduces DO, and increases CO_2 and sewage fungi. Fish may penetrate the upper and lower limits of this zone. In the zone of recovery, DO gradually increases, and certain fish such as suckers, stone rollers, creek chubs, and shiners are found. In zones of clean water, game fish found conditions favorable for existence. Brinley (1942) characterized five polluted zones in his study of the Ohio River. They are active bacteria decomposition zone, intermediate zone, fertile zone, game fish zone, and biologically poor zone. The first four zones are almost identical to the ones designated by Campbell (1938). In the zone of active bacterial decomposition (DO between 3 mg/l to zero mg/l) immediately below the sewage outfall, only a few fish, mostly coarse fish such as carp and buffalo fish, can penetrate this region. They feed on fresh sewage. In the intermediate zone (DO, 3-5/mgl), carp, buffalo fish, shiners, minnows, catfish, suckers and sunfish are present. In the fertile zone (DO supersaturated during the day time) where plankton become abundant, mixed fish fauna (coarse and game fish) is present. In the game fish zone (DO nearly saturated) various species of bass, perch, walleye, pike, and other game and forage fish are present. In the biologically poor zone, plankton become scarce and fish also become scarce in number.

Fish as an indicator of water pollution. A great number of investigators have published biological measurements or biological indices of water pollution. The benthic macroinvertebrates, because of their low mobility, have been considered to be the most suitable organisms for this purpose (Gaufin and Tarzwell, 1952, 1956; Claassen, 1932; Paine and Gaufin, 1956; Gaufin, 1958, 1964; Burlington, 1962; King and Ball, 1964; Dean and Burlington, 1963; Fremling, 1964; Wilhm, 1967; Wilhm and Dorris, 1966, 1968; Cairns and Dickson, 1971). Other aquatic communities such as sessile protozoa (Sládečková and Sládeček, 1965; Mohr, 1952; Cairns et al. 1968) and algae (Patrick, 1949, 1950; Patrick and Strawbridge, 1963; Palmer, 1969) have also been used. Kaesler and Cairns (1972) and Mackenthun (1966) used algal, benthic and fish communities. Despite the fish community's representing the end product of the food chain and its direct importance to a stream's economical and recreational yield, its use as a biological indicator of stream pollution has received considerably less

attention than other communities. Thomas, et al. (1973) compiled a bibliography of abstracts of 546 references on biological indicators in water pollution.

Any stream which contains fish must also maintain a large population of living organisms. Therefore, fish may be used as a good indicator of stream pollution (Claassen, 1932). Jenkins (1965) indicated that the best known indicator of water pollution is fish. Czensny (1949) defined a clean water stream as one in which the natural fish fauna is able to live and breed, the composition of natural flora and the small animals on which fish feed has not been changed or their density diminished, and nothing interferes with the operations involved in fishing. In general, the absence of fish typical of the stream can be taken to indicate pollution. Mackenthun (1966) stated that fish represent the end product of the aquatic phase of the food chain, but because of their mobility, they indicate water quality only at the particular time capture is made. The fish population responds to adverse environmental change in a manner similar to that of the bottom organism community. The use of fish as an indicator of water pollution was also suggested and its importance was discussed by other workers (Lüdemann, 1957; Liepolt, 1952; Shweng, 1957). Doudoroff and Warren (1957) indicated that the absence or extreme scarcity of some fish in a stream below the point of entry of a waste, and not above the point of entry, strongly suggests that the waste is somehow detrimental to these fish. If valuable food and game fish species are among those believed to be adversely affected, pollution is indicated. They concluded, after reviewing the various taxa as indicators for stream pollution, that only fish themselves can be said to indicate reliable environmental conditions generally suitable or unsuitable for their existence.

Fish kills. Fish kills are the most dramatic effect of water pollution. Since 1960 numbers of incidents and number of fish killed caused by pollution in the United States have been summarized annually, first by the U.S. Public Health Service, then by the U.S. federal Water Pollution Control Administration, and now by the U.S. Environmental Protection Agency. The summary of ten years from 1960 to 1970 was reported in "Fish Kills Caused by Pollution in 1970" by the Agency (Anon, 1971). Since 1960 a total of 161 million fish have been reported killed by pollution in 4,548 separate incidents. Municipal sewage pollution was the second largest source of fish kills, next to industrial wastes. In 1970 alone, 6.6 million fish were killed by sewage pollution (Anon, 1971 1972a). In 1971 the number increased to 24.8 million fish, though fish kills from sewage pollution represented only 82 percent

of the total number of incidents of the year. In Ohio alone, 115,234 fish were killed by municipal sewage pollution in 1972 (Anon, 1972b) and DO deficiency was one of the factors causing fish kills (Anon, 1956b).

There are many incidents of fish kills by sewage pollution. For example, in the Anacostia River, near Washington, D. C., approximately 3,180,000 fish were killed on or about September 20, 1962. This appeared to be caused primarily by the discharge of greater than usual volumes of raw sewage (40 million gallons) coupled with the presence of algal bloom. The size of the Anacostia fish kill was probably due to change and the coincidental migration downstream of a school of alewives (Anon, 1962a). In the Kalamazoo River, Michigan, heavy losses of carp due to oxygen depletion occurred in September 1953. The river was polluted with untreated sewage and industrial wastes, particularly from pulp mills. Conditions were aggravated by prolonged hot weather and low stream water. There was no DO in a 40 mile stretch of the river and the concentration of H_2S reached 2.5 mg/l (Anon, 1953). In the Dog River, near Mobile, Alabama, the effluent from an overloaded treatment plant lowered the DO content in a two mile stretch of the river, suffocating 1,023,000 fish (Anon, 1969). The low DO was also reported as the cause of fish kills in Lytle Creek, Ohio, in the fall of 1952 (Tarzwell and Gaufin, 1953). In the Illinois and Rock Rivers, Illinois, Forbes (1912) noted that fish kills occurred due to the flushing out of sludge beds which had accumulated during periods of low water. In the lagoon at Camorin, Brazil, since 1956, there has been a large scale of fish kills during February every year. The death of fish was due to excessive growth of surface plankton and these died when the action of anerobic bacteria resulted in production of H_2S, which was the direct cause of the fish mortality. In Germany, 120 cases of fish kills were reported in 1949. Of them, 103 cases were due to sewage pollution or by trade water (Wagner, 1950).

Difficulties in ascertaining the cause of fish mortality and in distinguishing between mortalities caused by oxygen deficiencies and those caused by toxic compounds was discussed by Burdick (1965). Wood (1960) recommended the use of pathology procedures in conjunction with biological studies and bioassay to determine the cause of fish mortalities.

Fish fauna and fisheries. There are mountainous reports on the effects of sewage effluents on water quality and aquatic life, particularly bacteria, algae and benthic invertebrates, but comparatively fewer on fish. They have been discussed or reviewed by many authors (Donaldson et al. 1934; Pritchard, 1954; Tarzwell, 1955; Dickinson, 1952:

Pentelow, 1956; Anon, 1957 and 1959; Greenburgh, 1951; Hynes, 1960; Klein, 1962; Wilber, 1971; Warren, 1971; Thomas, 1968; Vivier, 1960; Bieleby, 1960; David, 1963). Sinha (1971) made an annotated bibliography which contains 601 informative abstracts of literature on lake and river pollution.

In the United States, effects on fish life in streams and lakes receiving sewage effluents were studied in Lytle Creek, Ohio (Katz and Gaufin, 1953; Katz and Howard, 1954), Ohio River (Brinley, 1942; Anon, 1962b), Branch Creek, Oklahoma (Wade and Craven, 1965), East Gallatin River, Montana (Avery, 1970), Illinois River (Pearse, 1933), Vermillion River and West Branch of the Salt Fork, Illinois (Smith, 1968), Coosa and Blackwarrier Rivers, Alabama (Swingle, 1953), Parkison Hill Creek, Alabama (Rogers, 1960), Lake Erie (Verduin, 1968; Arnold, 1969; Meter and Shepherd, 1967), Patuxent River, Maryland (Tsai, 1968, 1970), Stillwater Creek, Oklahoma (Cross, 1950).

Lake Erie is perhaps the best documented example of a large lake which is deteriorating rapidly because of human activities and pollution. Oxygen depletion is most notable in the deeper central basin. Phytoplankton, oligochaetes, chironomid larvae and sewage fungi and eutrophic species of zooplankton increased, while mayfly and naiads decreased. The population of coregonids, sturgeon and pike, have been reduced, while shad, alewife, smelt, and carp have increased (Arnold, 1969). Meter and Shepherd (1967) indicated that nutrient enrichment from the Detroit River and along the shore and bloom of filamentous algae have changed the fish fauna and fish catch in Lake Erie. Total catches of commercial species in 1966 was 220,000 lbs., well below the average annual production of 852,000 lbs. in the period 1935-1966. The decrease in the catch probably was due in part to the disappearance of certain high-value fishes such as the blue pike and the white fish, and in part to the rising costs of fishery operation.

In the Illinois River, the fish catch has declined since 1909 to a low in 1924. Since that year due to pollution control the fish catch has gradually increased (Pearse, 1933). Fish died when DO was less than 2 mg/l. Chronic pollution from waste disposal plants effluents have prohibited the occurrence of fish at several sites in the Vermillion River and the West Branch of the Salt Fork (Smith, 1968). Pollution ranks fifth in damaging native fish in Illinois, and has exterminated two species and decimated five species of native fish (Smith, 1971). In Alabama, Swingle (1953) reported that the fish population in the stretch below the sewage and industrial effluents was much less than in the unpolluted stretch and the population was unbalanced in the Coosa River and Blackwarrior River. In Parkison Hill Creek, Alabama, fish were absent when sewage

pollution reduced DO to only 1.0 mg/l and ammonia concentration was as high as 5-120 mg/l. Three months after the installation of a sewage treatment plant, fish recovered (Rogers, 1960). In the Ohio River, the species composition varied through the river due to pollution. Due to increased construction of municipal sewage treatment plants and the treatment of trade wastewaters, the fisheries have been improved recently (Anon, 1962b).

In Canada, sewage pollution has not been considered a serious meance to fish in the Atlantic Provinces (Sprague and Ruggles, 1967). In Newfoundland and Labrador, sewage pollution is very common, but the volume of sewage has been insufficient to cause dissolved oxygen depletion, but it is becoming a problem in the area (Taylor, 1965).

Due to sewage pollution fish in the River Hull between Beverley and Driffield, England, have gradually disappeared (Sheppard, 1933). Out of a total 550 miles of flowing water there are more than 100 miles where neither plants nor animal can live. In the last century, salmon, eel, and lamprey fisheries were important industries in the Trent River, but, by 1925 they had disappeared. During the last ten years the rainbow trout have begun to reestablish themselves. The coarse fish were also slow recovering. This was due to improvements in the treatment of sewage and trade waste water (Spicer, 1937). In the River Tees, the concentration of sewage was probably not sufficient to be directly harmful to trout. However, the Skerne near Croft was too badly polluted to support fish life and on occasions contained no DO (Butcher et al. 1937).

In Germany, over 100 sewage outlets are situated in the Main River, particularly concentrated at Aschaffenburg, Stockstadt, Offenbach, Frankfurt, and Höchst. The most heavily polluted stretch of the Main River was between Kelsterbach and Tlörsheim. The reduced development of fish food diminished the yield of fish and fishing was hindered by heavy growth of sewage fungi (Lowartz, 1933). In the River Fulda, the fish population above the town of Fulda was typical of a barbel river. Below the point of sewage discharge from Fulda, fish life was destroyed and has reappeared only gradually (Müller, 1949). For at least 15 km below the discharge the river was unsuitable for fish (Schmitz, 1949). Similar pollution, but on a much smaller scale, was contributed by the town of Hersfield (Müller, 1949). In the Rhine River the continued pollution has changed a salmon river to an eel river and in which the eel population is decreasing (Schweng, 1957). In many streams in Germany, a trout population has been replaced by bleak and pike.

In Spain, in recent years there has been a marked decline

in the numbers of salmon and trout in the river Bidasoa. In some zones urban and industrial discharges caused considerable turbidity, preventing salmon from passing to the upper reach (Sart, 1969).

Growth and reproduction. In Lytle Creek, Ohio, receiving partially treated sewage, the growth rate of creek chub (0-year class) in the lower portion of the recovery zone was faster than in clean water and other polluted zones (Katz and Howard, 1954). In the Bow River, receiving domestic and industrial wastes, coefficients of the condition of Catostomus commersoni and of C. catostomus taken at the downstream stations increased (Kussat, 1969). In Lake Constance (Bodensees) which was enriched with organic materials, Nümann (1964) found that, the white fish (Coregonus wartmanni)grew faster, but at any given size the gonads and the eggs were smaller. Therefore, the existing fishery practice removed the immature and prevented inadequate spawning. Also, mortality of the embryo was greater (50 percent) than before (20 percent). In addition, eutrophication caused the migration of perch from shore into the pelagial, where they probably ate more whitefish eggs and fry than before.

Haines (1973) found that carp were able to take advantage of the high productivity in enriched ponds and grew at a greater rate than in low fertility ponds. Smallmouth bass, on the other hand, were inhibited by conditions in high fertility ponds and grew at a slower rate than they did in low fertility ponds. The depression of bass growth rate may have resulted from the increased diurnal dissolved oxygen flux (2 mg/l in night), decreased water clarity, or increased ammonia concentration. Chew (1972) found the non-reproductive populations of adult largemouth bass in a number of lakes where there were eutrophic and overcrowded with bream and forage species. Reproductive failure was due to a refusal of the adult population to spawn. Reproductive inhibition was attributed to the excretion and build up of a hormone-like repressive factor by overcrowded bream and forage species.

Fish movement. It has been observed repeatedly that certain species of fish will swim to sewer outlets and feed there. However, the septic zone below the domestic sewage effluents may become a barrier to migratory fish either up or downstream (Moore, 1932). During the time of low water flow in the Boberröhrsdorf, Boberullersdorf and Mauer inpounding reservoirs on the River Bober below Hirschberg, the fish crowded to the points at which the tributaries discharge (Bachmann, 1937). Tsai (1970) indicated that as sewage pollution increased in the Little Patuxent River, Maryland, the upstream migration of white catfish, white perch, white sucker was apparently blocked by chlorinated

sewage effluents.

Fish flavor and odor. Sewage and industrial wastes have often been known to cause objectionable taste and odors in fish (Fisher, 1939; Ricker, 1946) and thus, these polluted areas are unsuitable for commercial fishing (Anon, 1962b). Carp in sewage oxidation ponds also acquire an off-flavor (Aschner et al. 1967). For fisheries, the fish must not be exposed to substances which impart an unpleasant taste to fish (Collette, 1967). Powers (1962) stresses the need for evaluating pollution in terms of its direct effects such as odor, taint, flavor, and toxicity to fish, as a necessary preliminary measurement to meaningful control.

The bad taste and odor was reported to fish flesh in the waters polluted by sewage such as in the Boherröhrsdorf, Boberullersdorf, and Mauer inpounding reservoirs in the River Bober (Bachman, 1939), in the Ohio River (Anon, 1962b), and in the Mississippi River below St. Louis (Robinson, 1970; Baldwin et al. 1970). In the Mississippi River below St. Louis, flesh tastes and odor of carp, flathead catfish, and freshwater drum were adversely affected by pollution of their environment from a municipal-industrial complex. Freshwater drum was more subjected to flavor difference apparently related to water site than were carp or flathead catfish. Since differences varied through seasons, water temperature might play a role in the deposition of components of such unpleasant flavor in the flesh of the fish. On the other hand, load and type of pollution associated with different times of year might have influence on the flavor of the fish flesh.

Shumway and Palensky (1973) exposed rainbow trout for 48 hours to various concentrations of primary and secondary treatment wastes from the Corvalis Municipal Sewage Treatment Plant in Oregon. The threshold concentration value for flavor impairment of fish ranges from 11 to 13 percent by volume for secondary effluent and from 20 to 26 percent by volume for secondary chlorinated effluent. Addition of chlorine to the effluent appears to reduce the flavor caused by the effluent.

Fish disease and anomaly. In polluted water there is an increased incidence of bacterial disease and encysted trematodes in fish (Hubbs, 1933). Brinley and Katzin (1944) observed that fish collected downstream from a sewer outfall were sickly, abnormal or parasitized. Klinke (1970) found that furunculous lesions and necrosis of the intestine caused by Aeromonas salmonicida was the major factor of death of graylings, brown trout, and char which passed through areas heavily polluted by organic materials in rivers in the region of the northern Alps, between Lake Constance and Salzburg. Bullock and Snieszko (1970) suggested that fin and tail rot of fish is associated with poor

sanitary conditions in aquaria and with water pollution in nature. Bullock (1968) indicated that bacterial invasion occurs via a lesion resulting from the presence of another disease, nutritional imbalance, injury, or other predisposing factors.

In the polluted zones of the Bow River, Canada, receiving domestic and industrial effluents, there were decreases in the incidence of and the number of certain internal parasites (Octospinifer macilentus, Neoechinorhynchus cristatus, Rhabdochona fortunatowi) in Catostomus commersoni and C. catostomus, indicating that intermediate hosts were unable to survive in the more heavily polluted reach of the river (Kussat, 1969).

In the grossly polluted reach of the Schuylkill and Delaware Rivers in or near Philadelphia, many brown bullhead catfish were affects with papillomas (Lucke and Schlumberger, 1941). This neoplasm usually occurs as a solitary or multiple, large, red, fleshy mass upon the lips or dental plates. The tumor is comprised of epithelial cells, often in papillary arrangement, supported by a delicate vascularized connective tissue stoma. The larger growths frequently increase adjacent normal tissues and force their way into vessels where they are found as emboli. Mawdesley-Thomas (1972) indicated that bottom feeding fish living in polluted waters have a high incidence of squamous cell carcinoma of the lip, snout and mouth, and the possible link between these two factors cannot always be highly dismissed. The high incidence of knothead disease in carp increased in domestic waste discharge (Thompson, 1928). Some osteological abnormalities of carp are thought to be connected with pollution (O'Donnell, 1945).

Contamination of human pathogens. Freshwater fish do not have a permanent coliformor Streptoccocus flora in their intestinal tract. The composition of the internal flora of the fish is related in varying degree to the level of contamination of water and food in the environment (Geldreich, 1967; Glantz and Krantz, 1965; Margolis, 1935). If fecal coliforms are present in the fish intestinal tract, they are a reflection of pollution from some warm-blooded animal sources, particularly sewage pollution (Geldreich, 1967; Geldreich and Clarke, 1966).

Coliform and Streptococcal groups of bacteria have been isolated from the intestinal tract of various species of freshwater fish caught in relatively clean to moderately polluted waters (Amyot, 1901; Johnson, 1904; Venkataraman and Sreenivasan, 1953; Evelyn and McDermott, 1961). Leiguarda et al. (1950) examined the intestinal bacterial flora of 97 fish from the river Plate. Coliform bacteria were present in 92 fish, Streptococcus faecalis in 52 fish, and Salmonella in 19 fish. Of the Salmonella, S.

typhimurium was most abundant. In general, distribution of the various types corresponded to the distribution in river water, and it appeared that fish were not important factors in the transmission of pathogenic bacteria. Geldreich and Clarke (1966) also examined the intestinal contents of 78 fish from a moderately polluted section of the Little Miami River. The fecal coliform densities were lowest in bluegill sunfish (less than 20 per gram) and highest in catfish (1,090,000 per gram) and levels of fecal Streptococci for the two species were 200 and 240,000 per gram respectively. It appears that these differences are mainly due to habitats and feeding habits of the species.

Mackenzie and Campbell (1963) found that in Tilapia stocked in sewage lagoons, Proteus was isolated from the skin, Paracolon organisms from the gills, flesh, and intestine, but no Salmonella and Shigella were isolated. Because no significant pathogenic organisms were found and the authors concluded that the fish would be safe for human consumption when properly cooked. The fish can also cause erysipelas on the hand of persons working with fish (Brunner, 1949; Steiniger, 1954).

Steiniger (1954) infected the carp with typhoid bacteria and showed that 24-48 hours after infection bacteria were present in all organs and persisted for eight to fourteen days. They did not disappear until after four to six weeks. The fish remained healthy. Temperatures attained by cooking are not, in general, high enough to destroy the bacteria, so that transmitting to humans is possible. Brunner (1949) found that when fish ate food infected by Gärtner bacteria or were kept in infected water, bacteria could be detected within a few days in the gut and later in the flesh and other organs. After the initial infection several weeks were required for the disappearance of the bacteria. Even after seven to nine weeks in the fish the bacteria did not lose their pathogenecity to man. Danger of infection of fish is greatest where pollution of water by industrial waste waters weakens the resistance of fish to bacteria, rather than where fish are grown in sewage works effluent. Geldreich and Clarke (1966) indicated that the fecal coliform and Streptococcus faecalis can probably survive and multiply in the fish intestinal tract, when fish and water temperature are 20 C or higher, but only when the organisms are retained in the gut for periods beyond 24 hours. Fish may also be carriers of pollution from warm-blooded animals for periods up to approximately seven days and in this manner could transfer potential pathogens to clean water areas.

Beneficial effects. Organic wastes serve as nutrients which stimulate growth and reproduction of aquatic life (Gaufin, 1955). Introduction of small quantities of sewage or moderate amounts of organic materials which do not seriously affect dissolved oxygen levels, may serve as nutrient materials and increase fish production (Claassen, 1932; Tarzwell and Gaufin, 1953), but too

much organic material may destroy the freshwater organisms including fish through oxygen depletion (Claassen 1932). Surber (1953) and Bloodgood (1955) also indicated that if the decrease in oxygen is not enough to harm fish, the fish should appear in greater number or size, because organic materials increase fertility which stimulates the production of large amounts of fish food.

In Lake Erie, as Hubbs (1933) indicated, sustained fish production was due to fertilization of the lake by the wastes of the adjacent human population. Metcalf (1942) found that in Lake Erie, sport fishing was stimulated near the Lakewood, Ohio, sewage disposal plant outfall. The biota in the effluent supported populations of microcrustacea which in turn attracted minnows and white bass. In Stillwater Creek, Oklahoma, an increase in the catch of fish was observed after raw sewage was diverted into the stream (Cross, 1950) and increased sewage load increased fertility which stimulated the large amounts of fish food (Wade and Craven, 1965). In the Illinois River, increased yields of fishes were observed after the sanitary sewage of Chicago was diverted into the river (Hubbs, 1933). In the East Branch, Illinois, a fish population apparently benefited from enrichment of stream water by the domestic sewage of Rantoul, from which untreated sewage ran through an open ditch where it was well digested before entering the river (Thompson and Hunt, 1930). This beneficial effect of sewage on stream fish was also found in Drumma Creek and Piney Branch in Illinois (Larimore and Smith, 1963). In the Green-Duwamish River, Washington, receiving the effluent from the Renton Waste Water Treatment Plant, the overall effect was not detrimental to fish and desirable organisms and the treatment plant may have contributed to the increase in population of some species of fish (Matsuda et al. 1968).

Downstream from the direct adverse effects of domestic effluents there are generally favorable conditions for the growth of fish food organisms and fish (Ricker, 1946). In the Ohio River, downstream from the outfalls, the decomposition products of domestic sewage increase the growth of plankton, and, in turn, fish production (Brinley, 1943). When the sewage has received proper secondary treatment, the toxic or degradation zone does not exist and the entire stream will be benefited by the available plant food introduced. In the Black River, Alabama, 100 miles below the point of sewage and industrial discharges, sewage has a fertilizing effect. The weight of fish per acre is much greater than in similar unpolluted rivers (Swingle, 1953). Tebo (1959) compared the fish population in to polluted arms receiving sewage and industrial effluents, and a non-polluted arm of a reservoir. The populations of both coarse and game fish were higher in the polluted arms than in the non-polluted arm.

Fish fauna and fisheries. Coastal and estuarine regions constitute an important commercial and sport fishing grounds and the habitats of many species. About 73 percent of the value ($970,909,000) of 40 species of edible fish harvested in the United States in 1939 derived from the species that may be subject in greater or less extent to the effects of coastal pollution (Fisher, 1939). An interruption of the shallow coastal margin region, which is an important path for nomadic and coastal pelagic fishes, with detrimental effects from discharged sewage, may seriously damage these limited areas of high productivity, and may interfere or even block the normal movements of sensitive species. (Limbaugh, 1957). In estuaries, free passage of fish is essential for maintaining fish stock (Collette, 1967). Estuaries must be kept free from localized pollution which is likely to change the composition of the bed, or blanket it with silt in fish spawning ground. Sewage discharges in estuaries reduce the DO content, eliminate fishing and passage of migratory fish (Buckley, 1974). The effects of pollution of estuaries on aquatic fauna may be much more complex than that of polluting streams (Newell, 1958). The importance of studying tidal waves in estuaries, to determine whether pelagic larvae may be carried, and to assess the effect on fisheries of the effluents borne by these currents, was stressed.

Liebmann (1956) indicated that the notion that sewage entering the sea is quickly mixed and diluted is false. Persistent layering occurs and sewage "bars" form along the coast, cutting off bays and fjords, and causing alteration in fauna and flora as well as damage to fish. Pike and Gameson (1969) stated that although discharge of sewage into the sea will have ecological effects, these will not be serious from an economic or nuisance view point, if outfalls are sited away from fisheries and shellfish beds in regions where a high degree of dilution can be rapidly made. The same idea was expressed by Pentelow (1961): that although discharge of sewage at sea can have short-term harmful effects, these can be avoided if proper care is taken, and in such cases discharge of sewage may have a long term beneficial effect on fisheries as a result of its nutritive effect. In the North Sea, it was found that the effects of sewage and trade waters are mostly local and restricted in coastal areas.

Weisbecker et al. (1970) indicated that in the San Francisco Bay, the trend of the king salmon fishery over the past 90-year period in the Sacramento-San Joaquin Delta and Suisun Bay has been downward. Pollution has been involved in the salmon decline. It has resulted in an extensive loss of habitat, destruction or forage organisms and in the outright killing of the fish. The absence of striped bass in many areas of the bay, such as

south San Francisco Bay, is rather clear evidence of pollution. Perlman (1970) summarized the findings of various bodies commissioned to study the water quality of the bay. Some specific effects of pollution are high algal population, high concentration of dissolved solids limiting fish spawning, fish kills, and the prohibition of commerical harvest of shellfish. In site bioassay, Norris et al. (1973) suspended three-spined sticklebacks in cages at the North Point effluent field. The observed survivals ranged from 0 to 100 percent after 96 hours. Survivals were apparently unrelated to distance from the point of effluent discharge. Allen and O'Brien (1967) conducted laboratory tests on the survival of juvenile king salmon in effluent from a raw sewage lagoon at Arcata, California and in sea water mixed with hatching water, and with lagoon effluent. It was found that the lagoon effluent was not toxic to the salmon. At the same time the mixture of sea water and lagoon effluent had a therapeutic effect on the fish which suffered from unidentified internal parasites. Using the bioassay of the San Francisco Bay wastes, Kaiser Engineers Consortium (1969) found that there was a linear relationship between the concentration of the waste toxicity and the reduction in benthic animal species diversity. Armstrong et al. (1971) defined a toxicity parameter which is used for a measure of gross toxicity of the San Francisco Bay wastes, not requiring identification of the specific toxicants. They developed a relation between their parameter and a diversity index of benthic animals, and established a design criterion for the San Francisco Bay providing that the relative toxicity of a wastewater discharge must be diluted to 25:1 ratio. Brown and Beck (1972) also made a similar recommendation that 0.04 toxic unit calculated from the stickleback bioassay be the maximum permissible toxicity concentration in San Francisco Bay Delta water. Norris et al. (1973) made a bioassay of San Francisco Bay sewage effluents by using shiner perch, walleye perch, ling cod, skulpin, rock bass, and three-spined stickleback as test animals. The results showed that to assure 90 percent survival in 96 hours the dilution ratio is 20-50:1. No significant toxic effect on any organism tested after 96 hours of exposure in 100:1 could be demonstrated. In a solution of 10:1 dilution for seven days, the stickleback expressed stress by a change in the number of white blood cells and thrombocytes. The initial responses seemed to be a reaction of protection against large numbers of bacteria. The haemotological responses seems to be a very sensitive index. They recommended that the sewage effluent dilution ratios be 1,000:1 for intertidal community, 500:1 for the benthic community, and 200:1 for the pelagic community.

For the Santa Monica Bay, California, Hume et al. (1962) summarized the results of studies carried out to the end of 1959 on the effects of effluent and sludge discharged from

the Hyperion Sewage Works of Los Angeles. Bioassay had shown no toxic effects to fish in concentrations which occur in the Bay except in the immediate vicinity of the outfall. The effluent concentration at which 50 percent of the test fish survived during a 24 hours to 96 hours test period varied from 30 to 91 percent. The safe concentration is 3.0 to 9.1 percent, fairly similar to the dilution ratio recommended from San Francisco Bay by Armstrong et al. (1971) and Brown and Beck (1972). In the vicinity of the outfall there was no evidence of a build-up of sludge deposits. The number of and species of fish in the areas did not change greatly (Hume and Carber, 1967). Carlisle (1969, 1972) made a trawl study in the Bay from 1958 to 1963. He also indicated that the fluctuations in abundance could not be related to the polluting discharges, and anglers' catches showed only samll fluctuations over the study years. There was no evidence of the effect of waste discharge on the catch per angler day. The attraction or repulsion to sludge and effluents caused changes in fish fauna, but did not show Santa Monica Bay to be an area untenable to fish life as was once thought (Carlisle, 1972). Ludwig and Storrs (1970) also indicated that fluctuation in fish abundance in Santa Monica Bay were not due to the result of waste discharge. However, some fish species tended to avoid the areas of high effluent concentration, while other species were attracted to such areas. Thus the species diversity in the discharge area may be lower than in an effluent-free area. In the vicinity of the Los Angeles sewer outfall at White Point, near San Pedro, California, Grigg and Kiwala (1970) found that there was no observable damage to fish life. However, many species of fish were either rare or absent around the vicinity of outfall. The number of species at each station was negatively correlated with the amount of fine grain organic laden sediment. Since these benthic organisms made up the diet of many resident fishes, the number of resident fishes in turn would be expected to decline.

Stevens (1966) evaluated the benefits of water pollution control to recreational angling in Yaquina Bay, California. It was found that the direct benefit of pollution prevention could be measured by the economic success of the fisheries. Turner et al. (1966) studied the ocean bed in the vicinity of the outfall from the sewage works of Orange County Sanitation District, California, near the mouth of Santa Ana River, in January and February, 1965. It was found that the variety of species and numbers were smaller on the artificial reefs. However, the fish were healthy and many of them, such as kelp bass, sand bass, and brown rockfish, were relatively large.

In the estuary of the Green Duwamish River, receiving the Renton activated sludge of Seattle, Washington, the cycle of fish abundance, indicated by trawl studies, corresponded

to changes in the DO concentration near the bottom. It appeared that there was an avoidance reaction by the fish to low DO (Gibbs and Isaac, 1968).

In Galveston Bay, Texas, Bechtel and Copeland (1970) made a correlation of fish species diversity with percent wastewater. They gave the species diversity index values from 0.02 in these areas receiving up to 88 percent effluents by volume to 2.2 in less polluted areas of the bay.

Beeston (1971) compared the fish fauna in three brackish water ponds receiving sewage and thee control ponds. Fundulus heteroclitus was the dominant species in the experimental ponds, whereas spot was the most abundant in the control ponds. Low diversity at the experimental ponds was attributed to low DO. Hyle (1971) also indicated that in the same ponds commercial species were present in control ponds and coarse fish were found in the experimental ponds polluted with sewage.

In the Mayaguez Bay on the west coast of Puerto Rico, receiving crude sewage from the city of Mayaguez and waste water from tuna fish canneries, Valle et al. (1970) made and ecological survey to determine the effects of pollution on the distribution of fish in the area. The bay was found to be a fairly good balance between the nutrient input and fish produced, although a few patches of red tide appeared in the eastern part of the bay.

Cox and Gordon (1970) made a statewide survey of estuarine pollution in Hawaii. A few fish kills occurred in areas such as Pearl Harbor, Honolulu Harbor, and Hilo Harbor. However, none were found to be connected with sewage. The obvious effects of the pollution of Hawaii estuaries were still limited.

For the Delaware River estuary, Raney (1952) stated that the lower Delaware River was an outstanding example of the destruction of a striped bass habitat by industrial and domestic pollution. Chittenden (1971) studied the striped bass in the lower Delaware River between 1960 and 1967. The results indicate that the decline in this species was due to sewer pollution of the tidal freshwater area which had destroyed its potential as a spawning and nursery area. Daily minimum DO was at or near zero mg/l consistently in the summer and during much of the spring and fall. This destruction of the spawning and nursery grounds may explain the present low status of Delaware River striped bass stocks. Talbot (1966) also stated that pollution is one of the factors adversely affecting the striped bass population. In the Indian River Bay and Rehoboth Bay, Delaware, Derickson and Price (1973) found that changes in fish fauna in the bays were apparently due, at least in part, to increased pollution, primarily by sewage. This increased pollution appears to have had a greater effect on the species which utilize these bays as nursery and feeding ground than on the five most abundant resident species, Fundulus majalis, F. heteroclitus, Menidia menidia, Pseudo-pleuronectus americanus,

Anchoa mitchilli. A reduction in the number of potential predators (Pomatomus saltatrix, Paralichthys dentatus, and Monroe saxatilis) and high tolerance to pollution account for large populations. The population of P. americanus has increased in the bays over the last few years concurrent with severe reduction in P. dentatus.

In contrast to the above findings in the Delaware Bay, Mansueti (1961) indicated that increasing sewage fertilization in an estuary might be indirectly responsible for increasing striped bass population in the Chesapeake Bay. However, he noted that if fertilization is uncontrolled and unpredicted, it could have deleterious as well as beneficial effects on striped bass. Wiley (1971) conducted a survey of Baltimore Harbor, Maryland, which is polluted with industrial and domestic sewage. There were large populations of fishes particularly white perch. However, there was a notable absence of bottom fishes, probably due to heavily polluted sediments and low concentrations of dissolved oxygen in bottom water during warm months. Anon (1968) estimated the average annual loss of fishery resources in the Chesapeake Bay area due to the destruction of fish and shellfish habitats to be $2,950,718. The total affected area is 462,521 acres.

Pollution and fisheries in the River Thames, England, was reviewed by Wheeler (1969, 1970). Commerical fisheries (lamprey, shad, salmon, smelt, flounder, and eel) of considerable economic value existed in its tidal reach until the middle of the 19th century. Increasing pollution from industrial and domestic effluents led to their complete destruction and, in the 20th century, to the virtual absence of a local fish fauna except eel. It was normal not be be able to detect DO in its water around 1960. Hydrogen sulfide produced in anarobic water made the river unpleasant for workers. Recent measures to control pollution have resulted in a partial chemical restoration of the river. Now fish are present in considerable number again. Forty-one species were taken in the period from September 1967 to October 1968. Migratory fish have penetrated through the worst polluted reach into upstream clean water. Wheeler stated that to have cleaner water is an improvement, but to have fish living in the river is a major advance. Organic pollution in the lower Thames River may have benefited the fisheries in the southern North Sea. Carruthers (1954) stated that the fish catch per unit area in the sourthern North Sea was about double that in the rest of the North Sea in the English Channel and in the Kattegat Skagerak region. This high catch of fish may be due to the rich supply of nutrients from the populations in London. On the basis of response of aquatic organism in New York Harbor and Thames estuary, Torpey (1967) distinguished five zones of pollution, according to oxygen requirement.

In the river Humber, England, which is a large estuary formed by the confluence of the rivers Ouse and Trent, a survey

was made in summer, 1949 (Anon, 1951). It was concluded that the decline in the salmon fisheries in the river Ouse was caused by the increased pollution in the river.

Hardy (1938) explained the reason for the extermination of salmon in some rivers in England. There are two races of salmon, one which ascends the rivers in the spring and the other in the summer and autumn. Salmon ascend the Severn River in the spring when the river is in flood and dilution of the polluting matter is greatest. In the Thames and Mersey rivers, the salmon ascend in the summer and autumn when the rivers are low. This race of the autumn migration is the one easily exterminated by pollution.

In the lower river Viskan, Sweden, which was influenced by domestic and trade waste water, the principal fish were eels and roach; salmon were also present, but there were no areas suitable for spawning (Grimas, 1967). Turbidity ranged from 116-650 zp. Dominant bottom organisms were those living in the sediment, reducing their availability for predation by fish and consequently also reducing the yield of fish. Bond (1967) discussed pollution in the sound separating Denmark and Sweden which was severely polluted by sewage. Of various fish examined, herring was the only one in which the yield appeared to be reduced. No effects on the yields on plaice, cod and flounder were reported. The author concluded that discharge of sewage from the increasing population might still be permitted, if adequate treatment were provided and advantage were taken of hydrographic conditions to avoid spread of pollution.

Freeman (1969) examined the DO concentration in the Usk estuary, which supported a large population of migratory fish. Fifty percent DO reduction in the polluted reaches of the estuary was found. It was concluded that the estuary was in a threshold state. In the brackish water of the upper Lane Cover River, Sydney, Australia, numerous eel and sea mullet died during a drought in 1951. The fish appeared to die from lack of DO and probably partially due to the oxidation of the hydrogen sulfide. (Costin, 1954).

Sprague and Ruggles (1967) indicated that most fishing communities in the Atlantic provinces of Canada are located in protected harbors into which they discharge their domestic sewage. This sewage pollution, perhaps more than any other, is causing concern to the fishing industry, because the industry has a high bacterial standard for sanitary control.

Braarud (1955) reported that the deepwaters of the Bonne-Fjord were void of DO, where hydrogen sulfide occurred up to 75-50 meters in November 1950. Direct and indirect effects of the sewage discharge into the fjord seem to be responsible for the high rate of oxygen consumption. The 50 year old commercial trawling was stopped. The feeding ground of the fish populations was also greatly reduced.

In the Oslofjord, a virtually enclosed bay, Foyn (1959) showed that there was a marked density stratification, particularly during the summer, and interchange of water with the

open sea is very limited. Discharge of sewage has brought about an anaerobic condition in the bottom and destroyed the prawn fishery, and causing eutrophication with blooms of dinoflagellates. Only the surface water offers a suitable condition for fish. Because of the risk of algal toxicity, gathering of shellfish has been stopped.

In the Kulti estuary, near Calcutta, India, sewage pollution has almost eliminated the fish, owing to deoxygenation (David, 1959). Fish can survive in the Kulti only for periods of about four hours in each tidal cycle near the outfall. The catfish (Pangasius pangasius) has, however, remained unaffected by pollution and is attracted at the sewage at the outfall. Its survival is largely attributed to storage of air in the buccal cavity and subcutaneous respiration, and the species is proving successful for culture in ponds.

Fish reproduction and growth. In the Delaware River estuary, a heavy pollution load from municipalities, agriculture, industries, and shipping has made the estuary and its relative water unsuitable as nursery and spawning grounds for many commercial species (DeSylva et al. 1962). The unsuitability of the area could affect the fish populations through increased mortality of larvae or avoidance of these waters by adult breeding populations. Striped bass eggs and larvae are absent in the estuary near Philadelphia due to the pollution (Murawski, 1969).

In the Baltimore Harbor, Maryland, only the fish eggs and larvae of pelagic species such as anchovy, white perch, naked goby, silver side, and killifish were found. The epibenthic species, such as hogsucker and Atlantic croaker were absent. The bottom did not appear suitable for the support of critical early stages of epi-benthic communities (Dovel, 1971). In the upper Patuxent River estuary, Maryland, the spawning grounds for estuary species such as white perch, white catfish, shad and herring fish have been gradually destroyed by sewage pollution (Tsai, 1971). The freshwater portion of the river has lost its value as a spawning grounds of these fishes and present stocks of the commercial estuary species are maintained by completion of their life cycle in the estuary alone.

In the Santa Monica Bay, California, Young (1964) found that spotted turbot from the polluted Long Beach area were of significantly lighter weight than those caught in clean water. Also, halibut were abnormally listless, dull-colored and soft to the touch. Those from the outside of the harbor during the same period were vigorous, brightly spotted and firm fleshed.

In the Mersey estuary, England, heavily polluted with sewage, Hardy (1938) found many young fish living but not spawning there. In the water of the Oslofjord, also polluted with sewage, DO was absent and hydrogen sulfide was present up to 75-50 meters. Hatching of cod eggs seems to be imparied but the growth rate of cod has been found to be good. Davis (1972) indicated that there is no direct effect

on reproduction of aquatic organisms of pollutional enrichment of waters. However, it can have far-reaching indirect effects.

Fish behavior. It has been reported that attraction and avoidance of fish to the nutrient enriched area in the vicinity of a sewage outfall depend very much on the species of fish. In the Santa Monica Bay, California, Carlisle (1969, 1972) found that in the vicinity of marine outfall of the Hyperion Works of Los Angeles, speckled sanddab and yellow sculpin showed an attraction to the nutrient-enriched area near the sludge outfall, while plain-fin midshipman and slender sole showed no special effects of the sludge or effluent. In the vicinity of the marine outfall from the Orange County Sanitation Sewage Works, most fish were concentrated at the terminus where they formed loose schools or aggregations between the bottom and 20 feet felow the surface, apparently feeding on the effluent itself (Turner et al. 1966).

Fish migration through the estuary may be interfered with or even blocked by pollution. Salmon movement in a polluted estuary was studied by Elson et al. (1970). Pentelow (1955) indicated that sewage causes severe deoxygenation in some estuaries such as the Thames to promote a condition which blocks the upstream and downstream migration to sea trout and salmon, so that these runs become extinct. This interference or blockage of salmon migration by sewage pollution in estuaries was also reported in Gray Harbor (Townsend et al. 1938) and Du Wamish Waterway within the city limits of Seattle, Washington (Smith et al. 1972).

Fish diseases and deformation. In Santa Monica Bay, Russell and Kotin (1957) found ten out of 353 white croakers taken about two miles from the outfall of the sewage treatment plant contained papilloms. No similar lesions were noted in 1,116 white croakers from non-polluted waters 50 miles away. All lesions were characterized by marked diffuse squamous epithelial hyperplasma. Young (1964) did bottom trawling in and around Long Beach Harbor and revealed a large number of fishes which were underweight, or were suffering from tumors, lesions, or the abnormality. In some cases only young fish appeared to be affected and it was suggested that these fishes died before becoming full-grown. Evidence indicates that the injuries were caused by toxic substances in sewage and waste waters discharged into the sea in this area. Spotfin crocker suffered from exophthalmia and large skin ulcerations. Dover sole had cancerous lesions about the mouth or body, while crocker, tongue soles, cusk eels and Pacific sanddabs had lip papilloma. Lesions similar to those found on the white sea bass were induced experimentally on killifish by exposing them for 12 days to three to seven percent of polluted water taken from the effluents. Chamberlain (1971) indicated that head papilloma and epidermal and body lesions were found in six species of fish in polluted south California coastal waters.

In the New York Bright, Mahoney et al. (1973) found 22 species of fishes being infected with fin rot disease. During the height of the epizootic in August 1967, incidence averaged eight percent in bluefish, four percent in winter flounder. Disease incidence started in spring, reaching a peak in July to September and decreasing in the fall. The infected fish had necrosis at the fins, skin hemorrhages, and skin ulcer. Some fish had abnormal necrotic kidneys. Bacteria belonging to Aeromonads, Pseudomonads, and Vibrio were isolated and implicated. The cause of epizootic was due to a dense bacteria population and environmental stress resulting from gross domestic and industrial pollution. Pollution might have promoted widespread and abundant growth of the disease-causing bacteria. Wood (1968) described conditions which promoted epizootic caused by Aeromonads and pseudomonads among salmonids in hatcheries. Organic enrichment of the habitat permitted abundant growth of the bacteria in the water. Accompanying this, the fish suffered some environmental stress which increased their susceptibility to infection by the bacteria. Nigrelli et al. (1965) stated that epizootics may be a contributing cause to the high natural mortality in the sand sole in Hecate Strait, British Columbia.

In the Baltimore Harbor, Maryland, Wiley (1971) found that white perch showed signs of stress in the form of infected lateralis system and deterioration of the tissues on the fish, probably due to pathogenic organisms. In clean water of the neighboring Chester River, none of these systems were noted on the fish.

In Europe, cauliflower disease, a papillomatous growth on the skin of eels, appeared to have spread as an epidemic during 1944 to 1956 from Baltic waters to northwewtern Europe (Deys, 1969). The disease reached such a proportion in 1946 that Danish fishmen complained it was disastrous because the eels could not be sold. Since 1956 the disease has spread to Holland and Belgium. The causative factors include infectious agents (virus), temperature, salinity, and oxygen tension. This papilloma was also found in oyster, flounder, croackers, bullhead, flathead sole, and Atlantic eels. All these fishes live at the bottom, at least for a certain period of their life history.

Wunder (1971) found many deformed cod fish near the mouth of the Elbe River which was found to be low in salinity and to contain wastewater effluents. Deformities included the gill covers and verebral column.

Contamination of human pathogens. The bacterial flora of marine fish depends solely upon the fish's recent intake and the degree of contamination in the food and water (Gibson, 1934a and b). Thus, human pathogens may occur in marine fish. Guelin (1952) found that fish caught in polluted coastal waters of the Mediterranean Sea, where coli-phages are present, contained many bacteria, including E. Coli,

Clostridium perfringens, and others of the intestinal group. On the other hand, the open sea where no coli-phage occurs, usually did not contain lactose-fermenting bacteria, sulphite reducing bacteria, or E. Coli. In Iwayama Bay, Palaw Islands in the Pacific Ocean, where sewage pollution was severe, Hardy and Hardy (1972) found that crabs, oyster and coral fish were contaminated with a high content of E. Coli; 0.2 to 1.7 million coliform per 100 ml in crabs, 10,000 times the concentration of the surrounding water. The oyster concentrated this bacteria even more, about 20,000 times the concentration in the surrounding water. Pike and Gameson (1969) reviewed the medical, aesthetic, and ecological aspects of sewage discharge to coastal water. Fish or shellfish taken from a polluted marine environment may be contaminated with sewage bacteria, and possible potential pathogens, and these organisms can be present in the products which reach the consumers.

Janssen and Myers (1968) collected white perch from the Chesapeake Bay drainages and studied the antibody to human pathogens. The fish taken from the streams in heavily polluted areas of the western shore of Potomac River, Patuxent River, Back River, Magothy River, Severn River, Patapsco River, and Middle River, showed the presence of specific antibodies to several bacteria pathogenic to humans. The fish taken from the streams in sparsely polluted areas (Chester River, Choptank River, Nanticoke River, Wicomico River) were free of such antibodies. The detection in fish of antibodies to the bacteria that cause human pseudotuberculosis, paratyphoid fever, bacillary dysentery, and a variety of chronic infections is especially ominous. They suggested that the possibility of fish becoming active vectors of human disease as a result of their infection with pathogenic bacteria in contaminated water deserves much more attention. It has been reported that the pathogenic bacteria associated with human and mammals are short-lived contaminants of the surface and guts of the fish, they derived from human fecal pollution of the environment, and they are unable actively to infect the fish. However, there is evidence that at least some of these bacteria can establish active infection in fish and persist for several weeks or longer.

Jeddeloh (1938) posed various theories regarding the origin of Haff disease among dwellers on the Haff island - that the illness probably resulted from eating fish which were contaminated by some poisonous substances. The greatest number of cases had occurred on the east side of the Haff where waters were considerably polluted by sewage from Komgasberg and waster waters from two cellulose factories. The similarity between Haff disease and known infectious diseases would suggest that the illness is caused by microorganisms in the fish or in the waters of the Haff.

Pollution indicator. Several authors have suggested using fish as an indicator for estuary and marine pollution. Mills (1952) suggested using fish as an index of tidal water

pollution. Sindermann (1972) suggested that warning signs of inshore water degradation include mass mortality of fish, changes in species composition, and increasing occurrence of abnormalities and diseases.

Bechtel and Copeland (1970) found that species diversity indices were a useful indicator of environmental and pollution stresses in Galveston Bay, Texas.These areas receiving the greatest amounts of effluents, domestic and industrial, and toxic materials have the lowest index. Beside fish species diversity, another result of deteriorated conditions, which would be evident only after a period of time, would be the absence of large fish in the area as they come unable to meet maintenance requirements due to stress.

In the San Francisco Bay, Pearson et al. (1971) also found that there was a striking degree of conformity in the general pattern of species diversity indices for fish, benthic animals, zooplankton, and microplankton populations. The apparent relationship between diversity indices of the several communities at different trophic levels and the diversity of fish population gives preliminary evidence of relationships that exist between trophic levels.

DeSylva and Scotton (1972) indicated that the larval stages of Stomiatoid fish were encountered in Biscayne Bay, Florida, instead of their typical offshore habitat. This indicates that wastewater emptied into the offshore waters was being carried into the bay.

Wheeler (1970) indicated, as many pollution control officers have discovered, that the presence of a percentage of dissolved oxygen in the water is a nebulous concept - being invisible, odorless and tasteless, but a live fish taken out of the same water is a measure of the health of a river, which everyone can understand and appreciate.

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RESIDUAL CHLORINE

Fresh Water Fishes

Effects on receiving streams and fish life. Chlorine has been commonly used for sewage treatment plants, mainly for effluent disinfection. It also serves to reduce biological oxygen demand, control odor, remove grease and for filter pooling control, filter fry control, sludge bulking control, diffuser plate cleaning and foaming control. These uses of chlorine in sewage treatment were well reviewed by Chamberlin (1948). At the present time chlorination is the basic and legal sanitary process in treating sewage. In most plants in Maryland, Virginia, and Pennsylvania, 0.5 to 2.0 mg/l residual chlorine is maintained in effluents (Tsai, 1973). The chemistry of chlorine in water to produce hypochlorous acid (HOCl) and its reaction with impurities such as ammonia to produce chloramines have been reviewed by several authors (Moore, 1951; Isomura, 1967; Draley, 1972; Zillich, 1972). Smith (1971) made an annotated bibliography of chlorine in 488 publications during the years between 1824 and 1971 in which the medical, toxicological, industrial, hygienic and environmental aspects are listed. Of them, only a single paper is found to be related to fish.

In the past there were several reports indicating that chlorination of sewage effluents is advantageous for streams and aquatic life due to the considerable retardation of biological action which produces moderation in the rate of oxygen depletion in waterways (Baity and Bell, 1929; Baity et al. 1933; Enslow, 1932). It retards bacterial decomposition until the polluted material has been carried downstream to a point where, by increased dilution, the oxygen requirement can be met (Baity and Bell, 1929). It improves streams by increasing DO content, decreasing BOD, cleaning the growth of slimy and filamentous organisms on banks and the beds, reducing turbidity, odor, and bacterial content (Baity et al. 1933). Enslow (1932) indicated that at Austin, Minnesota, superchlorination of packing plant wastes had improved the stream condition so that fish were in the river below the plant for the first time in years. Chamberlin (1948) indicated that chlorination, sufficient to produce a chlorine residual in the stream, will retard the normal decomposition process and will tend to prevent anaerobic conditions during normally critical periods of rapid oxygen depletion. Almquist (1937) concluded that investigations had failed to show that chlorine is responsible for the death of fish by chlorination in water works.

In contrast to those early reports, Enslow (1932) pointed out that chlorination of many organic compounds, closely allied to compounds present in sewage effluents, results in the production of end-products entirely different in nature from the organic materials, and, in many cases, non-assimilable by organisms. Further than that, these products act toxically on organisms. Allen et al. (1946) found that sewage plant effluents containing gas liquor chlorinated with quantities much smaller than those required to give residual chlorine detectable by orthotolidine test were highly toxic to stream fish. They suspected that thiocyanate was the composed product responsible for the toxicity of chlorinated sewage. Allen et al. (1948) indicated that the toxicity of chlorinated sewage effluents was caused by formation of cyanogen chloride from the reaction between chlorine and cyanate in the effluents. Visintin and Errera (1958) studied the occurrence of eels in the cistern water of the Island of Pantelleria, Italy, and showed that eels have no effect on the hygienic quality of the water. The chlorination strongly affected eels and free chlorine was lethal for them within a few hours. Roy (1961) found that chlorine present in pulp liquor was lethal to fish. The presence of colloidal suspended matter in the effluents accelerated the toxic effects of chlorine. The Manufacturing Chemist Association (1972) indicated that chlorination of waste waters containing certain organic compounds can result in the formation of stable reaction products, which may or may not contain chlorine and that these products can have adverse effects on aquatic organisms.

Hubault (1955; 1957) held roach in running water obtained from the river Meurthe and found that one of the toxic compounds in the water was chlorine. Tsai (1968; 1970) studied the effects of the chlorinated sewage treatment plant effluents on fish in the Patuxent River, Maryland. He found that chlorinated sewage effluents act first as toxic materials which seriously reduce fish species diversity and abundance immediately below the effluent outfalls. Further downstream, where toxicity of chlorinated effluents decrease to such a degree that it is no longer harmful to fish life, then a species shift will result, probably due to organic enrichment and deoxygenation. Wuerthele (1970a, b, c, d) held fathead minnows in live boxes in the Grand River and the St. Joseph River above and below the outfalls of the Lansing and Hillsdale sewage treatment plants in Michigan, at a distance of one and a half and four and a half miles. None of the fish held in cages below the outfall survived and fish mortality in cages extended to a point of four miles. The last cage at four and one half miles had 25 percent mortality. In continuous-flow bioassay using the same stream water and the effluent for 24 hours, the author found that no fish survived in wastewater with concentrations stronger than 25 percent and fish in concentration as low as 8.35 percent were unable to maintain themselves in the water column and probably would not have survived at this concentration during a longer bioassay

or under river conditions.

Basch et al. (1971) also held rainbow trout and fathead minnows for 96 hours in live boxes in the receiving streams above and below four different Michigan municipal wastewater treatment plants. Fish held below those outfalls were subjected to both chlorinated and non-chlorinated exposures during effluent discharges. They found that residual chlorine was the principal toxic constituent of the chlorinated effluents which were toxic to rainbow trout at distances of up to 0.8 miles and to fathead minnow to 0.6 miles. The 96 hr. TLm of total residual chlorine for the rainbow trout was 0.023 mg/l. Zillich (1972) made on-site continuous-flow bioassay at two Michigan wastewater treatment plants using chlorinated and dechlorinated sewage effluents. He showed that combined residual chlorine is the principal toxicant in these effluents. They were toxic to fathead minnow after they were diluted to two to four percent. The 96 hr.TLm for the fathead minnow was between 0.05 and 0.16 mg/l, and 0.04 to 0.05 mg/l were considered as threshold concentrations. Tsai (1973) made comparative studies of water quality and fish species diversity in stream locations immediately above and below the outfalls of 149 secondary sewage treatment plants in Virginia, Maryland, and Pennsylvania. He stated that presence of chlorine in effluents and in streams below the sewage outfalls was often detected by its odor and, in particular, through the physical and biological appearance of the stream bottom. The streams which received heavily chlorinated sewage often had clean bottoms, without living organisms in the area immediately below the outfall. These bottoms differed greatly from those in natural water upstream where algae and other vegetation were present. It also differed from the bottoms of the streams receiving unchlorinated sewage, where sewage fungi usually grew in abundance. No fish were found in water with total chlorine of 0.37 mg/l and the species diversity index went to zero in 0.25 mg/l. The species diversity index reduced 50 percent at 0.1 mg/l total chlorine and 25 percent at 0.025 mg/l.

Besides the fish mortality studies mentioned above, Shumway (1971) found that chlorinated secondary domestic wastewater treatment plant effluents and unchlorinated effluents caused off-flavor in fish, but chlorination apparently reduced slightly the inducement of off-flavor.

Toxicity. Addition of chlorine to water containing sewage produces several species of chlorine compounds, such as hypochlorous acid (HOCl), hypochlorite ion (OCl^-), monochloramine (NH_2Cl), dichloramine ($NHCl_2$) and trichloramine (NCl_3). Merkens (1958) indicated that the toxicity of chlorine depends, not on the amount of chlorine added, but on the concentration of residual chlorine remaining in the solution, and this in turn depends on the relative proportions of free chlorine (HOCl and OCl^-) and chloramines of which it is composed. He also indicated that toxicities of chlorine and chloramines to fish are of the same order, with free chlorine

being the most toxic. Rosenberger (1971) also found that free chlorine is the most toxic form of chlorine and that dichloramine appears to be more toxic than monochloramine to coho salmon. However, Holland et al. (1960) found that toxicity to silver salmon is higher for monochloramine than chlorine, and dichloramine is higher than monochloramine.

On the other hand, Doudoroff and Katz (1950) suspected that there is no very great difference in the toxicity of free chlorine and chloramines to fish. Brungs (1973) concluded from literature that the lethal effects of free chlorine to aquatic life are more rapid and occur at lower concentrations than those of chloramines, and indicated that environmental variables do not appear to affect residual chlorine toxicity significantly. Hubbs (1930) attributed the toxicity of solutions of free chlorine and hypochlorites, as well as ozone and hydrogen peroxide, to the presence and action of nascent oxygen.

Perhaps because of the importance of chlorination in disinfection practice in water works and simplicity in mode of toxic action on bacteria, toxicity of different species of chlorine compounds to bacteria and virus have been fairly well understood. Hypochlorous acid (HOCl) is more effective in bacteriocidal action than chloramines (Brazis et al. 1959; Chambers, 1971; Heicken, 1956; Yagovi, 1959; Moore, 1951). The monochloramine is somewhat better than dichloramine (Moore, 1951; Yagovoi, 1959). The theory has been propounded that the disinfecting action of chlorine compounds is due entirely to small quantities of HOCl and OCl^- formed from them by dissociation and that the compounds themselves have no disinfection action. However, for anthrax spores, Heicken (1956) indicated that NH_2Cl is more effective than HOCl. Kitchatova (1952) showed that hypochlorite is more efficient in bacteriocidal action at low concentration of NH_3. Popp (1968) found that a primary decrease in counts for Salmonella, colon bacilli, and enteroviruses was caused by HOCl with a slower secondary decrease being affected by the chloramines present. Morris (1971) concluded that over the past quarter century, numerous studies on inactivation of human entric virus by aquaeous chlorine has demonstrated quite clearly that of all the common forms of aqueous chlorine, only undissociated hypochlorous acid (HOCl) can be regarded as an effective antiviral agent. Fetner (1962) studied the chromosome breakage in Vicia faba by chlorine compounds and also found that HOCl is more effective than NH_2Cl.

Modes of chlorine intoxication. Death of fish exposed to chlorine and its related compounds has been generally attributed to damage to the epithelial cells of gills, resulting in ultimate suffocation of the fish (Doudoroff, 1957; Mann, 1950; Penzes, 1971; Pike, 1971; Dandy, 1967, 1972). Rosenberger (1971) indicated that larger trout died faster than small fish in chlorine solution, because a large fish has less gill surface area per unit body than a small fish. In contrast, Forbes (1971) incubated the

white sucker for 30 to 60 minutes in a lethal concentration of chlorine (1 mg/l) and found that the gills were not the primary site of chlorine toxicity. On the basis of symptoms before the death of the fish, he hypothesized that chlorine enters through gills and somehow, either directly or indirectly, affects the nervous system.

Eaton et al. (1973) found that chloramines may cause marked metahemoglobinemia and hemolysis in hemodialyzed patients. They exposed the fathead minnow to water containing 1.5 ppm chloramines for about 40 minutes and found also that metahemoglobin was 32 percent (average) in the test fish, whereas the control fish had less than three percent. They concluded that chloramines may cause marked metahemoglobinemia in fish. Hiatt et al. (1953) found that a chlorine concentration of 10 mg/l produced violent irritation on fish, whereas 1.0 mg/l produced only slight irritation. They concluded that nearly all chemical irritants attack the common chemical senses. Skin irritants and lachrymators have been found to be the most effective. Lachrymators have positive helogens, as do chloramines, which irreversibly inhibit all enzymes whose activity depends on the sulfhydryl (-SH) group.

The effects of chlorine and chloramines to cellular level have been studied extensively in bacteria and virus. Green and Stumpf (1946) attributed the effects of chlorine compounds on a cellular level, particularly in connection with their bacteriocidal action, to the inhibition of glucose oxidation at the proper point and of bacterial growth. Fair et al. (1948) and Hess et al. (1953) indicated that the bacteriocidal action of chlorine compounds is based on their rate of diffusion through cell walls due to their small molecular size and electrical neutrality. Knox et al. (1948), on the basis of the fact that trace quantities of chlorine (0.2 to 2 mg/l) have been found to be bacteriocidal, concluded that these bacteriocidal quantities of chlorine are far below the amount causing cell lysis on nonspecific protein denaturation and that inactivation is most readily explained in terms of their effects on enzyme systems. They conclusively demonstrated for a variety of bacteria the precise parallelism between the effect of chlorine on bacterial growth and its effect on the rate at which glucose was oxidized. It may be concluded that chlorine is an effective SH reagent, capable of interrupting essential metabolic systems, possibly at several loci, the most important being the glucose oxidative system. Chlorine may react nonspecifically and irreversibly with any of more than twenty different enzymes associated with glucose oxidation. It was shown to be an exceptionally good inhibiter of several sulfhydryl enzymes, of which one is triose phosphate dehydrogenase, an enzyme essential to the glucose oxidative cycle. It is possible that multiple effects of chlorine occur in the metabolism of the cell. Moore (1951) concluded from literature that

oxidation of the living cell is not involved in disinfection by chlorine compounds, but that chlorine compounds enter the cell to react with certain essential enzymes and thus kill by stopping the metabolic processes of the cell. Naumann (1958) and Viglierchio and Croll (1969) attributed the mortality to the oxidizing power of chlorine-releasing compounds which can oxidize cellular enzymes of bacteria and the constituents of virus. Poduska and Hershey (1972) also concluded from literature that the bacteriocidal action of aquaeous chlorine occurs in two steps: diffusion of chlorine through the cell walls, and oxidation reaction with intercellular enzymes, terminating cell metabolic function. Chlorine attacks both the sulfhydral and amino group of the protein chain. Kulikouskii (1969) found that low concentrations of chloramines caused the aggregation of nucleoid fibrillae without disturbing the cytoplasmic membrane and cellular structure of E. coli. Subbacteriocidal doses caused a spread of the nuclear component which in turn caused changes in the cytoplasm with formation of electron-dense osmophilic lumps. Bacteriocidal doses caused alterations in all cellular structure.

Gard (1960) concluded from literature that destruction of the virus particles was due to diffusion of HCHO molecules through the proteins with subsequent inactivation of the nucleic acid. Fetner (1962) and Ingels (1958) found that monochloramine, like organic chloramines and nitrogen-mustards, can cause chromosome breaks at concentrations which show little evidence of tissue damage.

Hayatsu (1971) found that sodium hypochlorite is a potential carcinogen. Pangborn et al. (1970) indicated that chlorine primarily imparts an odor, rather than a taste, to drinking water. The total flavor-by-mouth impression of the solution was differentially modified. Undoubtedly, high concentrations of chlorine would modify the sensory properties of the resultant water both qualitatively and quantitatively.

Lethal effects. Toxicity of chlorine and chloramines to fish has been summarized by McKee and Wolf (1963), Draley (1972) and Brungs (1973). On the basis of all evidence available, cold water salmonid fish are much more sensitive than warm water species (Brungs, 1973).

Taylor and James (1928) reported that 0.3 mg/l of free chlorine killed rainbow trout in two hours and that 0.25 mg/l proved fatal to fingerling trout in four to five hours, but had no effect on goldfish in 42 hours. Conventry et al. (1935) indicated that some delicate species of fish are sensitive to residual chlorine or chloramines as low as 0.05 mg/l. Average 0.01 mg/l with maximum 0.06 mg/l chlorine are fatal to trout fry in 48 hours, while large carp and bullhead died in 0.76 mg/l (average) with a maximum concentration of 1.2 mg/l. The trout fry were killed instantly at 0.3 mg/l. The Water Pollution Research Board (1947) found that free chlorine concentrations above 0.3 mg/l were very toxic to trout and concentrations between 0.2 and 0.3 mg/l

were somewhat toxic. Westfall (1946) indicated that trout were killed at 0.06 mg/l, and warm water fish were killed at 0.4 mg/l. Merkens (1958) showed that 0.08 mg/l residual chlorine, mostly in the form of monochloramine, kill about one-half of a batch of rainbow trout in seven days, when the dissolved oxygen concentration was nearly 100 percent of the air saturation value. It appears that a safe concentration may be very low. An estimated chlorine concentration for median survival period of one year old trout is 0.004 mg/l. The Department of Scientific and Industrial Research (1958) found that residual chlorine, in concentrations as low as 0.1 mg/l, is markedly toxic to trout, particularly if the dissolved oxygen of water is low. Since fish liberated ammonia in the water, part of the chlorine was present as chloramines. Pyle (1960) found that rainbow trout are killed in two hours at 0.3 mg/l and that small mouth bass median mortality is 15 hours at 0.5 mg/l. Pike (1971) found that hatchery-reared yearling brown trout were killed by exposure to free residual chlorine for one hour at levels as low as 0.04 mg/l. At 0.01 mg/l free chlorine in water 50 percent of the test fish died in 43.5 hours and at 0.02 mg/l it was 10.5 hours. Dandy (1972) indicated that the mortality of brook trout is 67 percent after four days at 0.01 mg/l. The median survival time of the brook trout is 9 hours at 0.35 mg/l, 18 hours at 0.08 mg/l and 48 hours at 0.04 mg/l.

McKee and Wolf (1963) found, from the literature, that there is a wide discrepancy in the tabulations of the effects of chlorine concentrations on fishes. The authors attributed this to the fact that, pH, temperature, DO, and the synergism and antagonisms on the other pollutants markedly affect the toxicity of chlorine. Brungs (1973) indicated that most of the early reports deal with studies in which no measurements were made of residual chlorine which declined at rates dependent on the chlorine demand of the experimental water. Probably for this reason, seemingly high levels of added chlorine were nontoxic to fish. For example Panikkar (1960) observed that some black bullhead, green sunfish and other aquatic species were alive at 2.0 to 3.0 mg/l of chlorine in static bioassay. Ellis (1937), using static bioassay, observed no mortality of goldfish after 48 hours at 1.0 mg/l. Hagen (1940) found that sterilizing hatchery water, with 2 to 4 mg/l liquid chlorine added to spring ponds caused destruction of less than 30 percent of the fish. Therefore, Brungs (1973) suggested using continuous-flow bioassay procedures for tests of residual chlorine toxicity.

Besides the fish mortality studies, Arthur and Eaton (1971) found that the most marked sublethal effect of chlorine was the reduction in the number of eggs produced by the fathead minnows. The lowest measured total chlorine in the long term tests of the fathead minnow having no significant effect was 0.0165 mg/l.

Pike (1971) observed essentially the same responses after

the transfer of affected brown trout to clean water. He indicated that the cause of fish mortality due to chlorine, may not be detectable at the time the fish kill occurred. It is possible for fish kills to appear up to four days after exposure to low concentrations of chlorine for a limited time, and at the time of the observed fish kill, the presence of chlorine is undetectable. Dandy (1967) found that brook trout exposed to chlorine solutions and then transferred to freshwater will not recover. He indicated that irreversible gill damage is done by the time of transfer to freshwater, and a fatal ionic imbalance will continue. Tompkins and Tsai (1973) studied the relationship between survival time and exposure time of blacknose dace subjected to chlorine solution. They found that the lethal exposure time is much shorter than survival time of the fish in chlorine solution and their difference becomes wider with increasing chlorine concentrations.

Brungs (1973) recommended, after reviewing the literature, that in an area receiving wastes treated continuously with chlorine, total residual chlorine should not exceed 0.01 mg/l for the protection of more resistant aquatic organisms, or not exceed 0.002 mg/l for the protection of most aquatic life. Pike (1971) indicated that chlorinated sewage could be disastrous to trout fisheries. The trout are intolerant of chlorine at levels near the limit of detection by ordinary means. This means that it is not safe to discharge chlorinated water into a river at any concentration. Dandy (1972) suggested that since depressed general activity of fish by pollutants must be considered as unhealthy, concentrations of 0.005 mg/l chlorine should be avoided in water used for brook trout. Tsai (1973) indicated that if all species of fish are to be protected in areas immediately below sewage outfalls, the standard would be no detectable total residual chlorine in water. Cherkiaskii(?) recommended the standard of chlorine for freshwater organisms in USSR is zero mg/l.

Fish behavior. Dandy (1967, 1972) showed that exposure of brook trout to chlorine evoked changes in activity, ventilation, the coughing reflex and, at lethal levels, a heavy secretion of mucus. Locomotory activity increased initially and was subsequently depressed. Both responses were seen at 0.35 and 0.08 ppm, but at the sublethal level of 0.005 mg/l, the initial increase in activity was not seen. Forbes (1971) found that white sucker in one mg/l chlorine solution appeared nervous, more active, prone to darting and occasionally swam upside down on their side. Their rapid operculating became irregular near death, and they occasionally gulped air at the surface. Some test fish displayed small points of hemorrage in the caudal and anal region.

Tsai (1970) found the chlorinated sewage effluents became an ecological barrier, blocking upstream migration of semi-anadromous white perch and white catfish, and potamodromous white sucker and northern redhorse in the Patuxent River,

Maryland. Dandy (1972) suspected that if brook trout were released in a trough containing chlorinated and freshwater zones, the prediction is that levels of 0.35 to 0.08 ppm would stimulate activity so that the fish would rapidly move by chance into freshwater where the response would subside. In concentrations of 0.04 mg/l, activity would be depressed and chances of movement to the freshwater zone would be lessened. The fish would be effectively trapped in the lethal solution. Zillich (1972) asked why, if chlorine is toxic to fish at a few hundredths of a milligram per liter, there are no fish kills below treatment plant outfalls. The answer is that fish are known to avoid toxic materials at concentrations well below those required to cause toxic symptoms. Sprague and Drury (1969) studied the avoidance response of rainbow trout and Atlantic salmon to chlorine. The fishes can avoid the concentration of 0.01 mg/l which is lethal in 12 days, and of 1.0 mg/l which is rapidly lethal. However the fish preferred 0.1 mg/l. It is uncertain that preference of rainbow trout for intermediate lethal chlorine levels resulted from a depressant effect on activity. The author observed that at the boundary with freshwater the fish stopped short and turned back into chlorine. The momentary entrance into clean water apparently triggered an unpleasant sensation. Fava and Tsai (1973) found through experiments that the blacknose dace cannot discriminate and avoid sewage treatment plant effluents which are unchlorinated, or chlorinated to the level equivalent to chlorine demands of the effluents. The fish can discriminate and avoid chlorinated sewage effluents containing total residual chlorine as low as 0.01 mg/l.

Dechlorination of sewage effluents. Detoxication of chlorinated sewage effluents by dechlorination methods is essential for aquatic life. Krock and Mason (1971) indicated that dechlorination with sodium bisulfite eliminates chlorine-related toxicity in municipal waste effluents. Pyle (1960) used sodium thiosulphate and indicated that this chemical is non-toxic to fish and that it effectively neutralizes chlorine without producing any toxic neutralization products, and so is suitable for use in hatcheries. Brungs (1973) cited from Arthur's progress report that dechlorination of laboratory water with sulfur dioxide greatly reduced the acute and chronic toxicity to fish and invertebrate species. Hoppe (1950) recommended dechlorination of aquarium water by Aquola tablets. Anon (1957) outlined the methods of dechlorination of water, including filteration through activated carbon, addition of sodium thiosulphate and aeration.

Estuarine and Marine Fishes

In contrast to those made on freshwater fishes, very few studies have been done on the effects of chlorine or residual chlorine in sewage effluents on estuarine and marine fishes. There are no reports at all for chloramines.

Field studies. Bartsch et al. (1967) held O. keta in live boxes in the Snohomish River in the vicinity of Everett, Washinton, which received pulp and paper mill wastes. They found that times lethal to the test fish, where temperature and salinity were not levels expected to be lethal, free chlorine concentration was up to 50 mg/l, pH below 6.5, sulfide concentration 0.5 mg/l, or dissolved oxygen content approached zero. Krock and Mason (1971) studied the toxicity of municipal wastewater entering San Francisco Bay and surrounding areas and found a significant increase in toxicity to fish after chlorination of the effluent. Norris et al. (1973) held three-spined stickleback in live cages in the effluent field at various distance from the point of discharge in San Francisco Bay. Test results fluctuated widely, with observed survivals ranging from zero to 100 percent after 96 hours. Survival was apparently unrelated to distance from the point of effluent discharge, and the only conclusion that could be drawn was that some factor (boating), other than effluent, affected survival of the test fish. The authors did not mention chlorination. Beauchamp (1969) indicated that growth of plaice and sole in the discharge from power stations using a chlorine dosage of 0.5 mg/l for control of fouling condenser tubes can be successfully maintained during winter. The author did not mention the concentration of residual chlorine in water where the fish grew. Johannes (1972) indicates that unexplained coral mortality in the vicinity of sewage outfalls in Kaneohe Bay, Hawaii, might be due to residual chlorine. The effluent from the Kaneohe sewage treatment plant contained about 1.1 mg/l of residual chlorine, 45 times greaters than the lowest level found to be lethal to plaice larvae. Coral was also destroyed by being engulfed by a blanket of algae. Fish population in the area became strikingly less abundant.

Laboratory studies. Holland et al. (1960) indicated that chinock salmon started to die in sea water containing 0.25 mg/l of chlorine. The maximum non-lethal (in 23 days) concentration of residual chlorine for pink salmon and coho salmon in sea water was 0.05 mg/l. The authors stated that no chloramines were formed in sea water containing 0.05 to 0.5 mg/l chlorine and 3 mg/l ammonia. Perhaps the authors used orthotodiline and startch-iodide methods which might not detect chloramines in sea water. Alderson (1972) found that 48 and 96 hours TLm of free chlorine for the plaice larvae were 0.032 mg/l and 0.026 mg/l respectively. Survived larvae after 96 hours exposure gradually reduced feeding rates by 50 percent at 0.03 mg/l. Eggs were not affected by 0.75 and 0.04 mg/l chlorine solutions for eight days indicating that egg membrane gives considerable protection, allowing normal development over long periods even in concentrations of chlorine which would be rapidly lethal to hatched larvae. 72 hour and 192 hours TLm for the eggs was 0.7 and 0/12 mg/l respectively.

Muchmore and Epel (1973) found that unchlorinated domestic sewage is a relatively mild inhibitor of external marine fertilization in three species of marine invertebrates. Chlorinated sewage is a very potent fertilization inhibitor, active in concentrations as low as 0.05 mg/l available chlorine. Primary effects of both chlorinated and unchlorinated sewage are on sperm. Use of chlorine disinfection in sewage outfalls could contribute to reproductive failure in external fertilization of marine invertebrates in the vicinity of the initial diluting water of such outfalls.

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AMMONIA

Fresh Water Fish

Ammonia in water. In natural water ammonia is mainly a product of natural biological processes from aerobic or anerobic decomposition of nitrogenous organic matter (Klein, 1959). Water of unpolluted flowing streams generally contains extremely small amounts of ammonia nitrogen. Their amounts were found to be 0.0 mg/l to 0.9 mg/l in U.S. (Ellis, 1o37), 0.0 mg/l to 0.17 mg/l in England (Klein, 1959), 0.125 mg/l in Germany (Reichenbach-Klinke, 1967), and 0.05 mg/l to 0.4 mg/l in Pennsylvania, Maryland and Virginia (Tsai, 1973).

Water polluted with sewage contains very high amounts of ammonia as the result of insufficient decomposition of sewage protein (Schäperclaus, 1952). Concentrations as high as 11.7 mg/l by Ellis (1937), 25 mg/l by Tsai (1973) and 50 mg/l by Reichenbach-Klinke (1967) have been recorded. In sewage effluents concentration was recorded as high as 92 mg/l (Lloyd and Jordan, 1964). Klein (1959) indicated that if ammonia is present in appreciable amounts in stream water, this provides strong presumptive evidence of the presence of sewage pollution.

The chemistry of ammonia in water to produce ammonium hydroxide (NH_4OH), ammonium ion (NH_4+) and hydroxyl ion (OH^-) was reviewed by McKee and Wolf (1963). The ammonium hydroxide is commonly regarded as un-ionized or undissociated ammonia and the ammonium ion is ionized or dissociated ammonia. All common ammonium salts are also readily soluble in water, yielding both ionized and un-ionized ammonia. The concentration of un-ionized ammonia can be calculated from the formula shown by Herbert (1962) and Ball (1967) or from a table provided by Trussell (1972) showing the percentage of un-ionized ammonia in aqueous ammonia solutions over a range of temperature and pH values that are common in temperate freshwater environments.

In the field study, Ellis (1937) indicated that the good fish fauna showed a preference to water containing less than 2 mg/l ammonia nitrogen. Spicer (1952) found that in the Trent River where the ammonical nitrogen exceeded 5 mg/l, few, if any, coarse fish were likely to be found and that even when the values were over 1 mg/l the river was not very attractive to fish. In contrast, Tsai (1973) found that in the sections of the rivers immediately below the outfalls of sewage lagoons, where ammonical nitrogen concentrations were as high as 10 mg/l with no or very little residual chlorine fish species diversity and

abundance appeared not to be affected.

Modes of ammonia intoxication. Burrows (1964) observed that exposure of chinook salmon fry to sublethal level of un-ionized ammonia as low as 0.006 mg/l for six weeks, produced considerable hyperplasia (extensive proliferation and consolidation of the gill epithelium). The surface area of the gills was reduced and thereby the ability of the fish to liberate CO_2 and to absorb oxygen was reduced, increasing bacterial infection. Elis (1968) indicated that ammonia caused marked deterioration in organs in direct contact with the ammonia, such as skin, gills, and intestine, and a lesser degree of damage to other organs. The presence of ammonia in water resulted in increased production of mucus from gills and skin. Gill damage by ammonia was also indicated by Málăcea (1966) and Reichenbach-Klinke (1967). On the contrary, Herbert and Shurben (1964) found that the gills of some fish which died in ammonium chloride solution were normal histologically.

On the other hand, Jones (1967) suggested that ammonia acted on fish as a true internal poison entering the body by way of the gills. He suspected that fish were affected by high concentrations of ammonia similar to the way people were affected by spasms of the glottis and such swelling and congestion of the mucus membranes of the larynx and trachea that death occurred from asphyxia. Entry of ammonia via gill into fish was also indicated by Nehring (1963) and McCay and Vars (1930).

The mechanism of ammonia toxication appears to depend mainly on the permeability of the gills (Wurhmann and Woker, 1948). This relationship between ammonia toxicity and gill permeability was demonstrated in a carp suffering from ulcer disease. The sick carp was more susceptible to ammonia than the normal fish because of high permeability of the gills to water. This susceptibility might be reduced following treatment with supratin causing reduction in epithelial permeability to either water or un-ionized ammonia. Maetz and Romeu (1964) demonstrated, by adding ammonium ions to the water, that the sodium influx and net uptake of goldfish was inhibited, while intraperitoneal injection of ammonia produced the opposite effect. This fact seemed to support the hypotheses of a passive diffusion of ammonium ions out of the gill cells. This flux would then be stopped or even reversed as the concentration of ammonium ions in the external medium equalled or exceeded those in the cells.

Because normal blood ammonia values of fish are 0.1-0.4 mg/l and the excretion of ammonia by the fish is by passive diffusion via gills (Smith, 1929), blood ammonia level always exceeds that in the environment in which the fish live and the source of the blood ammonia must have been endogenous (Fromm, 1970). As water ammonia level increases, the ammonia excretion decreases. Intoxication of ammonia occurs when the rate of metabolic production and release of ammonia into the blood exceeds the combined rates of excre-

tion and detoxication in fish. Ammonia kills fish by preventing excretion of ammonia through the gill surface and not by interfering with gill respiratory exchange or by inhibiting oxygen transport by haemoglobin (Fromm, 1970).

Phillips et al. (1949) found that increased ammonia in water decreased the oxygen level of the blood in trout. In other words, the ability of haemoglobin to combine with oxygen decrease and hence, the fish might be suffocated (Brockway, 1950). This oxygen reduction in trout blood was noticed in water containing ammonia as low as 0.3 mg/l. With the reduction of DO in the blood, Reichenbach-Klinke (1967) found the number of erythrocytes was also reduced, while Kortings (1969) found that, with the reduction in haemoglobin level, there was a sudden rise in blood pressure, increase in lactate level and occurrence of acidosis in several species of fish. Wuhrmann and Woker (1953) found that ammonia affected the mechanism of oxygen uptake, oxygen transport, and oxygen demand. Penaz (1967) found that sublethal concentration of ammonia (2-10 mg/l as NH_3) caused irreversible damage to heart and muscle. Irreversible blood damage occurred in trout fry in water containing ammonia of 0.27 mg/l as un-ionized ammonia (Korting, 1969).

Lloyd and Orr (1969) demonstrated that a sublethal concentration of ammonia resulted in the increase of urine excretion of rainbow trout. It was suggested that this diuresis was caused by an increase in the permeability of the fish to water. The concentration of un-ionized ammonia of 0.046 mg/l as nitrogen would have zero effect on urine production; that was about 12 percent of the threshold value of 0.39mg/l.

The nervous system is also suggested as one of the main tactic organs in ammonia intoxication in fish (Malácea, 1966; Fromm, 1970). Wilson et al. (1968) found that ammonoletic species (fish) were more tolerant to the intraperitoneally administered ammonium acetate than either the ureotelic or uricotelic species. This may be due to the fact that fish have a high brain glutamine synthetase activity which detoxifies more ammonia at the cerebral level (Wu, 1963) along with rapid excretion rate of the blood ammonia (Goldstein et al. 1964).

Environmental factors affecting ammonia toxicity. Several environmental factors are known to greatly affect the toxicity of ammonia to fish, such as pH value, temperature, carbon dioxide, dissolved oxygen and hardness (Lloyd, 1961a; Food and Agriculture Organization, 1970; McKee and Wolf, 1963; Jones, 1967). Allan (1955) found in a laboratory experiment that a much higher concentration of ammonia was required at a given oxygen concentration to cause an equivalent kill of fish in a stream, and it is therefore concluded that in the field some other factor(s) was also important.

1. pH value. Cell membrane is relatively impermeable to ionized ammonia, because of its charge and low lipid

solubility, unlike un-ionized ammonia which passes tisuue barriers freely, because of its lipid solubility and lack of charge (Milne et al. 1958; Stabenaur et al. 1959; Fromm, 1970). Since ammonium ions do not cross cellular barriers in an appreciable rate and un-ionized ammonia does so freely, and equilibrium occurs which is dependent on the pH on both sides of the barrier. Therefore, one would expect that the pH value of an ammonia solution would have a great effect on the toxicity of the solution (Burrow, 1964; Reichenbach-Klinke, 1967; Fromm, 1970).

Lawrence et al. (1957) found that there was a direct relationship between an increase in blood pH and the diffusion of ammonia from blood into tissue. A relationship like that between alternation of blood pH and ammonia concentration in the tissue is well demonstrated between the blood and cerebrospinal fluid (Stabenaur et al. 1959). During alkalosis brain and muscle ammonia concentration increases, while acidosis decreases. Alkalosis enhances ammonia toxicity. The same phenomenon between the blood pH and brain ammonia concentration was also demonstrated by Warren and Nathan (1958).

At higher pH value a greater proportion of the ammonia in water solution is present in the toxic un-ionized form (Chipman, 1934; Grindley, 1946; Wuhrmann and Woker, 1948; Tabata, 1964; Food and Agricultural Organization, 1970). Doudoroff and Katz (1950) indicated from literature that high concentration of ammonium ions (NH_4^+) in water at low pH value may not be toxic, but if the pH is raised, toxicity will probably increase. Downing and Merkens (1955) found that ten times as much ammonium chloride had to be added to water at pH 7 to achieve a killing rate comparable to that at pH 8. In an aqueous solution at pH 7 only one percent of the total ammonia is un-ionized, whereas at pH 8, 10 percent of the total ammonia is in the un-ionized form (Warren, 1962). Jones (1967) indicated that the ammonia threshold to fish was about 12 mg/l at pH 8.6 and more than 100 mg/l at pH 7. In carp-breeding ponds fed by water containing sewage, Schaperclaus (1952) found that when a high pH value coincided with high concentrations of ammonia, fish mortality occurred. The high pH value of water was caused by mass development of phytoplankton. Hemens (1966) also found that algal bloom in summer in a sewage lagoon increased pH value and killed stocked mosquito fish. However, in a sewage lagoon low pH value occurs in winter when the ammonia concentration is high, and high pH value in summer coincides with low concentrations of ammonia and thus keeps the un-ionized ammonia lower than the toxic threshold value to the mosquito fish.

Ammonium salts appear to be relatively non-toxic to organisms. When administered or absorbed at a slow rate, vast quantities of ammonia may be tolerated by most organisms, because of their high detoxication mechanism (Warren, 1962). However, many authors have demonstrated that

regardless of the kind of ammonium salts their toxicity to fish is always a function of the concentration of un-ionized ammonia, which in turn is a function of pH values (Chipman, 1934; Wuhrmann and Woker, 1948; Wuhrmann et al. 1947; Southgate, 1952). Because of pH the alkaline ammonium salts are much more toxic to fish than the acid salts (Chipman, 1934). Ammonium hydroxide which is alkaline in solution, is much more toxic to fish than the neutral salts such as ammonium carbonate and ammonium acetate. The acidic salts such as ammonium chloride and ammonium sulfate are least toxic (McCay and Vars, 1930; Chipman, 1934; McKee and Wolf, 1963). The toxicity of ammonium sulfide is controlled by the resulting combinations of H_2S and NH_4OH in the water and also depends on pH value.

II. Carbon dioxide. Carbon dioxide lowers the pH value of water and is a major factor reducing the pH value of sewage. It is one of the important factors affecting the toxicity of ammonia. Alabaster and Herbert (1954) showed that 30 mg/l of ammonia nitrogen in tap water (pH 7.9) killed rainbow trout in 15 minutes, but if 30 mg/l of CO_2 was added, it lowered the pH value, and therefore the concentration of un-ionized ammonia became less and the trout were not affected until after 12 hours. The concentrations of CO_2 which reduced the toxicity of ammonia to fish were between 15 mg/l and 60 mg/l. This reduction of the toxicity of ammonia in high CO_2 solution was also demonstrated by Tabata (1964). However, increases in CO_2 increases sensitivity of fish to lack of oxygen (Herbert, 1955a). CO_2 is excreted by the fish through gills, the pH value at the gill surface will be lower than in the bulk of the solution, thereby reducing the proportion of un-ionized ammonia (Lloyd and Herbert, 1960; Anon, 1960a, 1961a).

III. Temperature. Temperature affects the dissociation constant of ammonia in aqueous solution. The dissociation constant increases with increasing temperature (Trussell, 1972) and is an important factor determining ammonia toxicity (Burrow, 1964; Reichenbach-Klinke, 1967). Increase of temperature increases toxicity of ammonia to fish (Wuhrmann and Woker, 1948, 1953; Herbert, 1962; Ball, 1967; Wilson, et al. 1968). Jones, (1967) found that the threshold value of ammonia to fish can be up to two and a half times higher at lower temperatures. On the contrary, Woker (1949) exposed Squalus cephalus to ammonia solution in temperatures ranging from 10 C to 25 C and found no effects of temperature on the toxicity of ammonia on this species. Lloyd and Orr (1969) demonstrated that changes in temperature did not change the urine production of trout in a constant sublethal concentration of ammonia. The authors suggested that reduced ability of salmoid fish to excrete at low temperature may have increased the susceptibility of the fish to this poison. This is of practical importance, since ammonia in the river polluted by sewage effluents may be higher

in the winter than in the summer.

IV. Dissolved oxygen. The survival period of trout in a given concentration of un-ionized ammonia increases considerably with the increase of DO concentration (Anon, 1954; Downing and Merkens, 1955). In other words, toxicity of ammonia to fish increased markedly at low tension of DO (Anon, 1955; Herbert, 1956; Merkens and Downing, 1957). This effect of DO on ammonia toxicity to fish is greater in lower concentrations of un-ionized ammonia. Jones (1967) and Lloyd (1961a) found that the threshold value of ammonia to fish was much lower in water deficient in oxygen.

The mechanism of increasing toxicity of ammonia in water of low DO tension has been explained by a few investigators. Reichenbach-Klinke (1967) attributed this to the infiltration rate of ammonia, which was higher in tissue lowering oxygen than those rich in oxygen. Lloyd and Herbert (1960) attributed it to the water pumping rate over the gill surface, which was more rapid in water deficient in DO, so that the concentration of ammonia in water contacting with gill surface would be increased. Anon (1961a) found that when the oxygen tension in water was reduced, the concentration of excreted CO_2 was also reduced, and the pH value of the water at the gill surface was raised, causing an increased toxicity of an ammonia solution. Thus, Lloyd (1961a) indicated that the effect of a given reduction in oxygen content of the threshold concentration for un-ionized ammonia depended upon the concentration of free CO_2 in solution.

V. Hardness. Toxicity of ammonia varies with the hardness, and is considerably greater in alkaline tap water than in distilled water (Well, 1915; Shelford, 1917, 1918; Grindley, 1946; Doudoroff and Katz, 1950; LeClere, 1960; LeClere and Devlaminck, 1955), and greater in hard water than in soft water (Anon, 1960b; Herbert and Shurben, 1964). This fact is particularly well demonstrated in various ammonium salts for various species of fish by these authors in Table 3-6. In other words, the un-ionized ammonia is less toxic to fish in a relatively unbuffered water than in a well-buffered water at the same pH value (Anon, 1960b). The effect of hardness on ammonia toxicity to fish differs with that found in heavy metals, which are more toxic to fish in saft water or distilled water than in hardwater.

Lethal effects. Data on the lethal effects of ammonia and ammonium salts on fish have been greatly accumulated in the literature (Table 1-6). As Doudoroff and Katz (1950) indicated, the results of the toxicity lists presented in the literature in many cases failed to distinguish clearly between undissociated ammonia or ammonium hydroxide (as NH_3) and the ammonium radical (reported as ammonia). This is somewhat confusing. The Federal Water Pollution Control Administration (1968) also expressed the opinion

TABLE 1. LETHAL EFFECTS OF AMMONIA ON FRESHWATER FISHES

Concentration mg/l	Fish	Test condition	Survival time	References
2.0	Fish	--	Lethal	Coburn (1950)
2.5	Goldfish	--	1-4 days	Rudolfs et al. (1953)
2.9	*Cichla ocellaris*	--	13 hours	Bastos (1954)
3.1 and 3.4	Bluegill sunfish	Softwater, 30 and 20 C	96-hr. TLm	Anon (1960b)
5.0	Rainbow trout	--	Lethal	Meinck et al. (1956)
5-7	Minnow	Distilled water, 20 C	6 hours	LeClere (1960)
6-7	Minnow	Hard water, 20 C	6 hours	LeClere (1960)
7-8	*Lepomis humilis*	Tap water	1 hour	Shelford (1917)
8	Rainbow trout	13 C, pH 7.4	24-hr. TLm	Fromm (1970)
15-20	Carp	--	Lethal	Elis (1968)
23.7 and 24.4	Bluegill sunfish	Hardwater, 30 and 20 C	96-hr. TLm	Anon (1960b)
35	Trout	--	50% mortality	Idler (1969)
35-36	Rainbow trout	--	48-hr. TLm	Lloyd (1961b)
37.5	Channel catfish	--	LC_{50}	Knepp and Arkin (1973)

TABLE 1. LETHAL EFFECTS OF AMMONIA ON FRESHWATER FISHES (continued)

Concentration mg/l	Fish	Test condition	Survival time	References
40	Goldfish	20-23 C, pH 7.4	10% mortality in 24 hours	Fromm (1970)
74.7	Rainbow trout	--	6 min. 43 sec.	Corti and Weber (1949)
75.7	Trout	--	4 minutes	Corti (1951)
120(N)	Gambusia affinis	pH 7.75	60-min. TLm	Hemens (1966)
120(N)	Gambusia affinis	pH 6.55	No mortality in 16 hours and 40 minutes	Hemens (1966)

TABLE 2. LETHAL EFFECTS OF AMMONIUM HYDROXIDE ON FRESHWATER FISHES

Concentration mg/l	Fish	Test condition	Survival time	References
2-2.5	Goldfish	Hardwater, 17-21 C	24 hours	Ellis (1937)
4.3 as NH_3	Minnow	--	No effect in 1 hour	Schaut (1939)
4-5	Goldfish	--	Lethal	Jones (1967)
5.0 as NH_3	Silver Salmon	Hardwater, 14.2 C, pH 8.0	Critical for 72 hours	Holland et al. (1960)
5.0 as NH_3	Silver Salmon	14.2 C, pH 8.0, DO 9.0, total alkalinity 85 m/l as $CaCo_3$	72 hours	Holland et al. (1960)
8.0 as NH_3	Silver Salmon	14.2 C, pH 7.15, DO 9.0	1.97 hour	Holland et al. (1960)
6.25	Brook trout	--	24 hours	Belding (1927)
10	Sucker, shiner, carp	--	Lethal	Clark and Adams (1913)
10	Creek chub	15-21 C	No effect	Gillette et al. (1952)
13	Sucker, shiner	--	24 hours	Belding (1927)
15	Bluegill sunfish	Tap water, 20 C	48-hr. TLm	Turnbull et al. (1954)

TABLE 2. LETHAL EFFECTS OF AMMONIUM HYDROXIDE ON FRESHWATER FISHES (continued)

Concentration mg/l	Fish	Test condition	Survival time	References
18.5	Bluegill sunfish	Tap water, oxygenated at 20 C	48-hr. TLm	Turnbull et al. (1954)
17.1	Minnow	--	1 hour	Schaut (1939)
24.8 as NH_3	Brown bullhead	--	Survived by 8 hours	McCay and Vars (1930)
30	Perch	--	Lethal	Jones (1967)
30	Sucker, shiner	--	24 hours	Belding (1927)
30	Creek Chub	15-21 C	Lethal	Gillette et al. (1952)
37	Mosquito fish	--	96-hr. TLm	Jones (1967)
37	Mosquito fish	20-20 C, pH 3.2-8.8 turbidity 121 mg/l	24-hr. and 96-hr. TLm	Wallen et al. (1957)
45	Mosquito fish	20-26 C, pH 8.2-8.8 turbidity 121 mg/l	24-hr. TLm	Wallen et al. (1957)
49.4 as NH_3	Brown bullhead	--	32 minutes	McCay and Vars (1930)
124 as NH_3	Brown bullhead	--	5 minutes	McCay and Vars (1930)

TABLE 3. LETHAL EFFECTS OF AMMONIUM CHLORIDE ON FRESHWATER FISHES

Concentration mg/l	Fish	Test condition	Survival time	References
6.0-7.7	Bluegill sunfish	20 C, CO_2 3-9 mg/l	96-hr. TLm	Cairns and Schier (1958)
26.4	Rainbow trout	17 C, pH 7.8, hardness 320 mg/l as $CaCo_3$, CO_2 5 mg/l	48-hr. TLm	Herbert and Vandyke (1964)
20-28	Rainbow trout	pH 8.0, CO_2 5 mg/l	24 hours	Lloyd and Herbert (1960)
24.6	Rainbow trout	17.5 C, pH 7.8, DO 45% saturation, hardness 320 mg/l as $CaCO_3$	48-hr. TLm	Herbert and Shurben (1964)
30 as N	Rainbow trout	17.5 C, pH 7.9, DO 4.4 mg/l	15 minutes	Alabaster and Herbert (1954)
30 as N	Rainbow trout	CO_2 30 mg/l, pH 7.4, DO 4.4 mg/l	12 hours	Alabaster and Herbert (1954)
31.1-36.5 as N	Rainbow trout	10-14 C, pH 7.7-7.8, hardness 320 mg/l as $CaCO_3$	24-hr. TLm	Herbert and Shurben (1963)
38	Rainbow trout	--	7.5 hours	Selitrennikova and Sachurina (1953)
160	Rainbow trout	--	30 hours	Southgate (1948)

TABLE 3. LETHAL EFFECTS OF AMMONIUM CHLORIDE ON FRESHWATER FISHES (continued)

Concentration mg/l	Fish	Test condition	Survival time	References
193	Rainbow trout	17.7 C, pH 6.9, hardness 44 mg/l as $CaCO_3$	48-hr. TLm	Herbert and Shurben (1964)
157	Rainbow trout	Tap water, 18 C, pH 7.7	16 hours 40 minutes	Grindley (1946)
180	Shiner, carp	--	No effect	Clark and Adams (1913)
268	Goldfish	Hardwater, 17-21 C	6 days	Ellis (1937)
300	Salmo trutta	--	6 hours	Scheuring and Leopoldselder (1934)
314	Rainbow trout	Tap water, 18 C, pH 7.6	Overturned time 8 hours, 43 minutes	Grindley (1946)
314	Rainbow trout	Dist. water, 18 C, pH 6.3	Overturned time 3 days	Grindley (1946)
400-500	Minnow	Hardwater, 18 C	6 hours	LeClere and Devlaminck (1955)
431	Rainbow trout	Tap water, 18 C, pH 7.7	Overturned, 10 hours, 13 minutes	Grindley (1946)

TABLE 3. LETHAL EFFECTS OF AMMONIUM CHLORIDE ON FRESHWATER FISHES (continued)

Concentration mg/l	Fish	Test condition	Survival time	References
468	Brown bullhead	--	Survived at the end of 9 hours	McCay and Vars (1930)
490	Mosquito fish	17-21 C, pH 7.4-8.1, turbidity 180 mg/l	96-hr. TLm	Wallen et al. (1957)
500	Carp	--	6 hours	Kraus (1936)
500	Rainbow trout	--	2 hours	Kraus (1936)
500	Rainbow trout	pH 7.6-7.9	2 hours	Thumann (1950)
510	Mosquito fish	17-21 C, pH 7.4-8.1, turbidity 180 mg/l	48-hr. TLm	Wallen et al. (1957)
535	Bluegill sunfish	Tap water	4 hours	Wells (1915)
535	Bluegill sunfish	Dist. water	18 days	Wells (1915)
700-800	Orange spotted sunfish	--	1 hour	Shelford (1917)
865	Mosquito fish	17-21 C, pH 7.4-8.1, turbidity 180 mg/l	24-hr. TLm	Wallen et al. (1957)
1,570	Rainbow trout	Dist. water, 18 C, pH 5.6	Overturned time 17 hours	Grindley (1946)

TABLE 3. LETHAL EFFECTS OF AMMONIUM CHLORIDE ON FRESHWATER FISHES (continued)

Concentration mg/l	Fish	Test condition	Survival time	References
1,712	Minnow	Dist. water	6-18 hours	Powers (1917)
3,140	Rainbow trout	Dist. water, 18 C, pH 6.0	Overturned time 12 hours 5 minutes	Grindley (1946)
4,000-5,000	Minnow	Dist. water, 16 C	6 hours	LeClere and Devlaminck (1955)
5,350	Goldfish	Dist. Water	12 hours	Chipman (1934)
6,280	Rainbow trout	Dist. water, 18 C, pH 5.7	Overturned time 4 hours 8 minutes	Grindley (1946)
9,420	Rainbow trout	Dist. water, 18 C, pH 5.2	Overturned time 4 hours 42 minutes	Grindley (1946)

TABLE 4. LETHAL EFFECTS OF AMMONIUM CARBONATE ON FRESHWATER FISHES

Concentration mg/l	Fish	Test condition	Survival time	References
5.5-7.0	Fish	--	Lethal	Moore (1932)
10	Goldfish	Hardwater, 17-21 C, pH 7.7	No effect	Ellis (1937)
29.8 as NH_3	Brown bullhead	--	Survived by end of 24 hours	McCay and Vars (1930)
35	Goldfish	--	Threshold	Rudolfs et al. (1953)
100	Goldfish	Hardwater, 17-21 C, pH 7.8	4-10 hours	Ellis (1937)
149 as NH_3	Brown bullhead	--	39 minutes	McCay and Vars (1930)
185	Mosquito fish	17-22 C, pH 7.9-8.5, turbidity 185 mg/l	24-hr. TLm	Wallen et al. (1957)
155-197	Shiner and carp	--	A few minutes to a few hours	Clark and Adams (1913)
240	Goldfish	Dist. Water	3.58 hours	Chipman (1934)
600-800	Orange spotted sunfish	--	1 hour	Shelford (1917)
1,000	Goldfish	17-21 C, hardwater, pH 8.1	1-2 hours	Ellis (1937)

TABLE 5. · LETHAL EFFECTS OF AMMONIUM SULFATE ON FRESHWATER FISHES

Concentration mg/l	Fish	Test condition	Survival time	References
66	Bluegill sunfish	Tap water	3 hours 30 minutes	Wells (1915)
66	Bluegill sunfish	Dist. water	17 days	Wells (1915)
148 as NH_3	Brown bullhead	--	Survived at end of 9 hours	McCay and Vars (1930)
160	Minnow	--	Threshold	Málácea (1966)
200	Carp	--	Minimum lethal concentration	Málácea (1966)
264	Goldfish	Hardwater, 17-21 C	6 days or less	Ellis (1937)
296 as NH_3	Brown bullhead	--	Average 215 minutes	McCay and Vars (1930)
300	Gudgen		Minimum lethal concentration	Málácea (1966)
388	Rainbow trout	Dist. water, pH 6.5	Overturned time 74 days	Grindley (1946)
420-500	Orange spotted sunfish	Tap water	One hour	Shelford (1917)

TABLE 5. LETHAL EFFECTS OF AMMONIUM SULFATE ON FRESHWATER FISHES (continued)

Concentration mg/l	Fish	Test condition	Survival time	References
600	Phoxinus phoxinus	20 C	3 days	Malácea (1968)
1,290	Mosquito fish	20-21 C, pH 6.3-7.4, turbidity 240 mg/l	96-hr. TLm	Wallen et al. (1957)
1,400	Mosquito fish	20-21 C, pH 6.3-7.4, turbidity 240 mg/l	48-hr. TLm	Wallen et al. (1957)
1,530	Mosquito fish	20-21 C, pH 6.3-7.4, turbidity 240 mg/l	24 hr. TLm	Wallen et al. (1957)
2,000	Rainbow trout	pH 6.5	3 hours 20 minutes	Thumann (1950)
3,840	Rainbow trout	Tap water, pH 7.1	Overturned time, 29 minutes	Grindley (1946)
3,880	Rainbow trout	Dist. water, pH 5.4	Overturned time, 9 hours 7 7 minutes	Grindley (1946)
7,760	Rainbow trout	Dist. water, pH 5.5	Overturned time, 4 hours 27 minutes	Grindley (1946)
11,640	Rainbow trout	Dist. water, pH 5.4	Overturned time, 5 hours 18 minutes	Grindley (1946)

TABLE 6. LETHAL EFFECTS OF AMMONIUM ACETATE, AMMONIUM NITRATE AND AMMONIUM SULFIDE ON FRESHWATER FISHES

Chemicals	Concentration mg/l	Fish	Test condition	Survival time	References
NH_4COOCH_3	149 as NH_3	Brown bullhead	--	Average 7 hours 25 minutes	McCay and Vars (1930)
NH_4COOCH_3	238	Mosquito fish	23-25 C, pH 7.6-8.4, turbidity 1,400 mg/l	24-,48-, and 96-hr. TLm	Wallen et al. (1957)
NH_4COOCH_3	298 as NH_3	Brown bullhead	--	Average 4 hours 8 minutes	McCay and Vars (1930)
NH_4COOCH_3	560-1,000	Mosquito fish	--	8 hours	Wallen et al. (1957)
NH_4NO_3	800	Bluegill sunfish	Tap water	3.9 hours	Wells (1915)
NH_4NO_3	800	Goldfish	Dist. water	16 days	Wells (1915)
NH_4NO_3	4,545	Goldfish	Dist. water	3 days 8 hours	Powers (1917)
$(NH_4)_2S$	10	Goldfish	17-21 C, hardwater, pH 7.7	No effect	Ellis (1937)
$(NH_4)_2S$	240	Mosquito fish	20-21 C, pH 2.7-6.7, turbidity 220 mg/l	24-,48-, and 96-hr. TLm	Wallen et al. (1957)
$(NH_4)_2S$	248	Mosquito fish	21 C	24-,48-, and 96-hr. TLm	Wallen et al. (1957)

TABLE 6. LETHAL EFFECTS OF AMMONIUM ACETATE, AMMONIUM NITRATE AND AMMONIUM SULFIDE ON FRESHWATER FISHES (continued)

Chemicals	Concentration mg/l	Fish	Test condition	Survial time	References
$(NH_4)_2S$	100	Goldfish	17-21 C, hardwater, pH 7.8	72 hours	Ellis (1937)
$(NH_4)_2S$	1,000	Goldfish	17-21 C, hardwater pH 7.9	15 minutes to 1 hour 30 minutes	Ellis (1937)

that because of inadequate reporting and unsatisfactory experimental control much of the work was not usable for establishing ammonia standards for aquatic life. As mentioned in the previous chapter, only un-ionized ammonia is a toxic component of ammonia solution and many environmental variables greatly affect its formation. The ammonia toxicity level designed by concentration of ammonia or ammonium salts, such as carbonate, chloride, sulfate, and acetate as shown in Table 3-6, are useless and often misleading.

Several publications list pH value and temperature in their experiments. The ammonium concentrations in those publications can be converted to the concentration of un-ionized ammonia by using the table provided by Trussell (1972) and compared with survival time of fish (Table 7 and 8). The values of 24-hr. TLm to 96-hr. TLm of un-ionized ammonia in rainbow trout fall between the concentrations of 0.05 mg/l to 0.79 mg/l. In concentrations higher than 0.79 mg/l the fish usually died in a few minutes or a few hours. There is a great discrepancy in concentrations for the same lethal effect. This may be due to the difference in test condition by different authors. Godlfish and mosquito fish are much more tolerant than the rainbow trout.

Regarding the difference in lethal effects of ammonia in various species of fish, the rainbow trout is more sensitive than coarse fish (Merkens and Downing, 1957; Wuhrmann, 1952; Wuhrmann and Woker, 1948; Ball, 1967). Fromm (1970) showed that under comparable conditions, goldfish was more resistant than trout to ammonia. Goldfish did not appear to be bothered at all by ammonia as high as 25 mg/l, some eight times greater than the concentration which affected the trout. Ball (1967) indicated that for a period of one to two days exposure, the rainbow trout was much more susceptible to ammonia, in terms of TLm value than the coarse fish. The rate of mortality of the trout fell rapidly, while that of the coarse fish changed little until at least two days had elapsed. The ultimate values were similar in all species. Male Gambusia affinis are filled more rapidly than females at the same concentration of un-ionized ammonia (Hemens, 1966). The eggs of brown trout are more resistant than the fish fry and older fish reacted more rapidly to the poison than did immature fish (Penaz, 1967)

Brown et al. (1969) found that there was no difference in the median survival time of trout immersed in constant concentrations of ammonia, or in concentrations fluctuaring with $\pm$ 50 percent of the 48-hr. TLm at equal intervals of time, as long as the periodicity of the fluctuations did not exceed the resistance time (that is the period of exposure after which irreversible changes occur). Herbert and Shurben (1963) forced the rainbow trout to swim for one to two days in water with current velocity of 37.7 cm/sec (85 percent of maximum sustained velocity). The 24-hr. TLm

TABLE 7. LETHAL EFFECTS OF UN-IONIZED AMMONIA ON RAINBOW TROUT (concentrations of un-ionized ammonia are converted by using the table provided by Trussell [1972], except those with stars which indicate the concentrations of un-ionized ammonia expressed by authors)

Un-ionized ammonia mg/l as NH_3	Chemicals used		Test condition	Survival time	References
	Chemicals	Conc. mg/l			
0.05	NH_4Cl	26.4 as N	17 C, pH 7.8, DO saturated, hardness 320 mg/l as $CaCO_3$	48-hr. TLm	Herbert and Vandyke (1964)
0.05	NH_4Cl	193 as N	17.7 C, pH 6.9, DO 95-100% saturated, hardness 44 mg/l as $CaCO_3$	48-hr. TLm	Herbert and Vandyke (1964)
0.05	NH_4Cl	24.6 as N	17.5 C, pH 7.8, DO 45% saturated, hardness 320 mg/l as $CaCO_3$	48-hr. TLm	Herbert and Vandyke (1964)
0.05	NH_3	8	13 C, pH 7.4	24-hr. TLm	Fromm (1970)
0.23	NH_4Cl	30 as N	Tap water, 17.5 C, pH 7.4, CO_2 30 mg/l, DO 4.4 mg/l	12 hours	Alabaster and Herbert (1954)
0.46	NH_4Cl	31.1-36.5 as N	10-14 C, pH 7.7-7.8, hardness 320 mg/l as $CaCO_3$	24-hr. TLm	Herbert and Shurben (1963)
0.35-0.4	NH_4Cl	--	9.4-14.6 C, pH 7.75-8.30, hardness 2290 mg/l $CaCO_3$	96-hr. TLm	Ball (1967)

TABLE 7. LETHAL EFFECTS OF UN-IONIZED AMMONIA ON RAINBOW TROUT (concentrations of un-ionized ammonia are converted by using the table provided by Trussell [1972], except those with stars which indicate the concentrations of un-ionized ammonia espressed by authors) (continued)

Un-ionized ammonia mg/l as NH_3	Chemicals used		Test Condition	Survival time	References
	Chemicals	Conc. mg/l			
0.42-0.84*	NH_4Cl	--	pH 7.0-8.2	8 hours 20 minutes (median)	Lloyd and Herbert (1960)
0.6*	--	--	--	100-200 minutes	Anon (1961b)
0.6	NH_4OH	--	--	24-hr. TLm	Fromm (1970)
0.79	NH_4Cl	20-28 as N	pH 8.0, CO_2 5 mg/l	24 hours	Lloyd and Herbert (1960)
1.08 as N	NH_4Cl	30 as N	Tap water, 17.5 C, pH 7.9, DO 4.4 mg/l	15 minutes	Alabaster and Herbert (1954)
2.62	NH_4Cl	157	Tap water, 18 C, pH 7.7	16 hours 40 minutes (over-turned time)	Grindley (1946)
4.18	NH_4Cl	314	Tap water, 18 C, pH 7.6	8 hours 43 minutes (over-turned time)	Grindley (1946)

TABLE 7. LETHAL EFFECTS OF UN-IONIZED AMMONIA ON RAINBOW TROUT (concentrations of un-ionized ammonia are converted by using the table provided by Trussell [1972], except those with stars which indicate the concentrations of un-ionized ammonia expressed by authors) (continued)

Un-ionized ammonia mg/l as NH_3	Chemicals used		Test condition	Survival time	References
	Chemicals	Conc. mg/l			
7.20	NH_4Cl	431	Tap water, 18 C, pH 7.7	10 hours 13 minutes (over-turned time)	Grindley (1946)
8.35	NH_4Cl	500	pH 7.6-7.9	2 hours	Thumann (1950)
8.48	NH_4Cl	8.48	Tap water, pH 6.9, 18 C	4 hours 33 minutes (over-turned time)	Grindley (1946)

TABLE 8. LETHAL EFFECTS OF UN-IONIZED AMMONIA ON GOLDFISH AND MOSQUITO FISH. Concentrations of un-ionized ammonia are converted by using the table provided by Trussell (1972)

Un-ionized ammonia mg/l as NH_3	Chemicals used: Chemicals	Chemicals used: Conc. mg/l	Test condition	Survival time	References
			Goldfish		
0.18	$(NH_4)_2CO_3$	10	Hardwater, 17-21 C, pH 7.7	No effect	Ellis (1937)
0.45	NH_3	40	20-23 C, pH 7.4	10% mortality in 24 hours	Fromm (1970)
2.26	$(NH_4)_2CO_3$	100	Hardwater, 17-21 C, pH 7.8	4-10 hours	Ellis (1937)
38.1	$(NH_4)_2CO_3$	1000	Hardwater, 17-21 C, pH 8.1	1-2 hours	Ellis (1937)
			Mosquito fish		
0.20	NH_3	120 as N	pH 6.55	No mortality in 15 hours	Hemens (1966)
1.1	--	--	--	16 hours	Hemens (1966)
3.13	NH_3	120 as N	pH 7.75	60 minutes TLm	Hemens (1966
4.13	$(NH_4)_2SO_4$	1200	20-21 C, pH 6.3-7.4, initial turbidity 240 mg/l	96-hr. TLm	Wallen et al. (1957)

TABLE 8. LETHAL EFFECTS OF UN-IONIZED AMMONIA ON GOLDFISH AND MOSQUITO FISH.

Concentrations of un-ionized ammonia are converted by using the table provided by Trussell (1972) (continued)

Un-ionized ammonia mg/l as NH_3	Chemicals used		Test condition	Survival time	References
	Chemicals	Conc. mg/l			
4.48	$(NH_4)_2SO_4$	1400	20-21 C, pH 6.3-7.4, initial turbidity 240 mg/l	48-hr. TLm	Wallen et al. (1957)
4.89	$(NH_4)_2SO_4$	1530	20-21 C, pH 6.3-7.4, initial turbidity 240 mg/l	24-hr. TLm	Wallen et al. (1957)
4.98	NH_4OH	37	20-26 C, pH 8.2-8.8, initial turbidity 121 mg/l	48-hr. and 96-hr. TLm	Wallen et al. (1957)
6.05	NH_4OH	45	17-21 C, pH 7.4-8.1, initial turbidity 180 mg/l	24-hr. TLm	Wallen et al. (1957)
11.07	NH_4Cl	490	17-21 C, pH 7.4-8.1, initial turbidity 180 mg/l	96-hr. TLm	Wallen et al. (1957)
11.53	NH_4Cl	510	17-21 C, pH 7.4-8.1 initial turbidity 180 mg/l	48-hr. TLm	Wallen et al. (1957)

TABLE 8. LETHAL EFFECTS OF UN-IONIZED AMMONIA ON GOLDFISH AND MOSQUITO FISH.

Concentrations of un-ionized ammonia are converted by using the table provided by Trussell (1972) (continued)

Un-ionized ammonia mg/l as NH_3	Chemicals used		Test condition	Survival time	References
	Chemicals	Conc. mg/l			
19.55	NH_4Cl	865	17-21 C, pH 7.4-8.1, initial turbidity 180 mg/l	24-hr. TLm	Wallen et al. (1957)

value of the trout in this flowing water was only 81 percent of that in still water which was 31-36.5 mg/l at pH 7.7-7.8 in hardwater. The data supports the conclusion that metabolic rate and susceptibility to poison are related.

Grindley (1946) found that only a small proportion of the rainbow trout overturned in ammonium chloride and ammonium sulphate recovered on being transferred to freshwater. Málácea (1966) also found that after losing equilibrium the fish would not recover on being transferred to clean water. Wuhrmann and Woker (1948) also indicated that fish poisoning by ammonia was usually irreversible. In contrst, Vamos (1963) found that carp suffering from ammonia poisoning could recover after being transferred to freshwater. The same result was also indicated by Fromm (1970) for the rainbow trout.

Fish may become more resistant to ammonia through acclimation in sublethal ammonia solutions. Málácea (1968) exposed minnow and bitterlings to sublethal levels of ammonium sulfate for several days and found they became more resistant to lethal concentrations. Lloyd and Orr (1969) found that acclimation of rainbow trout to ammonia might occur. The permeability was reduced and the fish could more readily withstand ammonia solution. Vamos (1963) found that the carp which had survived exposure to ammonia solution were more resistant when they were given a similar exposure after a recovery period of 12 hours.

Fish behavior. Shelford (1917) showed that fish were unable to recognize even a highly toxic solution of ammonia. Lepomis humilis reacted positively to a strong concentration and negatively to a weak concentration of ammonium chloride and ammonium sulphate. The author claimed that fish failed to recognize the wastes and avoid the danger zone, with the result they stayed in it and died there. The same result was obtained by Jones (1947) who indicated that Pyogsteus pungitus were not repelled but were even apparently attracted by lethal concentrations of ammonia. Jones (1948) found that Gasterosteus oculeatus showed immediately negative reaction to 0.05 N solution (pH 10.6) of ammonia, but with a 0.01 N solution the reaction was slow and some fish were overcome by exposure to the highly toxic solution. With 0.001-0.0001 N solution a definite positive reaction occurred. Fava and Tsai (1972) found that Rhinichthys atratulus could not avoid ammonium chloride solutions as high as 270 mg/l at pH 6.9-7.1. Doudoroff and Katz (1950) concluded from the literature that fishes were not repelled or were even apparently attracted by lethal concentrations of ammonia. The fact suggests how danger to fish from toxic pollutants can be aggravated in the vicinity of sewer outfalls, where mixing of wastes with available diluting water is incomplete.

In a strongly lethal ammonia solution fish often become

hyperexcited. Shelfore (1917) found that L. humilis in an ammonia solution lethal in an hour or less, the fish were often much stimulated, the head often floating lower than the rest of the body. Erratic movements followed after a time and the fish usually turned over in convulsion and remained comparatively still with peculiar twitching of the tail and fins until death. Belding (1929) indicated that brook trout in NH_4OH solution became wild, jumping and thrashing at the surface. Jones (1948) observed that Gasterostus oculeatus died suddenly in ammonia solution with widely opened mouths suggesting asphyxia. McCay and Vars (1930) observed that when bullhead were exposed to lethal concentrations of ammonium salts, activity and respiration were normal during the preliminary period. Suddenly the fish swam madly. It might even leap from the solution. The fish lost equilibrium, became stiffened, rigid, with its mouth open. Very similar behavior was found by Holland et al. (1960) who exposed silver and chinook salmon in lethal concentration of NH_4OH. The fish became restless, swimming in the upper half of the aquarium. Extremely violent activity was followed by quietness, feeble swimming, loss of equilibrium and the fish died with opened jaws and gills. Fromm (1970) also observed that rainbow trout in a solution containing more than 3 mg/l NH_3 (24-hr. TLm, 8 mg/l) became hyperexcitable. Any disturbance of the tank or movements resulted in disoriented escape attempts which sent the fish crashing into the side of the tank. However, goldfish were not so excitable as the trout in the ammonia solution. In 40 mg/l ammonia solution in which 10 percent of goldfish died in 24 hours, the onset of death was characterized by gradual cessation of swimming movement, and during that time the fish slowly settled to the bottom of the aquarium. After one or two hours nearly all of the dying fish rested laterally in the form of a "U" and opercular movements dropped considerably.

Fish growth. Broadway (1950) indicated that growth rate of salmonid fish was reduced due to an increase in ammonia concentrations in culture water by fish excretion. Burrows (1964) found by experiment that the prolonged but intermittent exposure to un-ionized ammonia (sublethal level) resulted in reduction in growth rate, physical stamina, and decreased resistance to bacterial gill disease in chinook salmon. The salmon fingerlings could tolerate levels of ammonium hydroxide as great as 0.7 mg/l for one hour per day, but when the exposure period exceeded 12 hours per day at levels of 0.1 mg/l or above, the growth rate was reduced. This inhibition of growth rate by ammonia is also found in carp. Kawamato et al. (1957) indicated that the fish in populations of large density grew less well than fish in populations of small density. The authors indicated that such inhibition of fish growth came mainly from the accumulation of large quantities of excretions such as ammonia. In experiments Kawamoto (1961) found that

ammonium chloride solution inhibited the growth of carp. The carp survived in 1.2 mg/l ammonium chloride solution but body weight decreased.

Toxicity of ammonia combined with other toxicants. Ammonia commonly associates with other toxic pollutants in domestic sewage and industrial wastes. Herbert (1962) indicated that the solution containing un-ionized ammonia and phenol were more toxic than solutions of un-ionized ammonia alone. Reichenbach-Klinke (1967) also found the increase in the effect of ammonia on fish in water containing phenol or other waste poisons. In the mixture of ammonia and heavy metals, Herbert and Vandyke (1964) found that there was different affinity between ammonia and zinc, and ammonia and copper. In the solution containing ammonium and zinc salts, less than one percent of the total zinc and even small percentages of the total ammonia would have been present as zinc-ammonium ion ($Zn(NH_3)_n^{2+}$), whereas affinity of Cu^{2+} and NH_4^+ was greater. About 75 percent of added copper but only a small number of added ammonium ions (about 0.3 percent) would have been in the form of cuprammonium ions ($Cu(NH_3)_n^{2+}$). The toxicity of the copper-ammonia mixture if related to the toxicity of individual poisons and thus, cuprammonium ions are equal or very nearly equal in toxicity to copper ions. Also cuprammonia complex dissociates easily to copper and ammonia when in intimate contact with the gills. Herbert and Schurben (1964) indicated that ammonia and zinc were not synthetic, but exerted toxic effects independently in such a way that the percentage mortality in a given mixture was directly related to the percentage mortality which would have been produced from separate exposure to the individual concentration of each toxicant.

The toxicity of the mixture of ammonia and other toxicants to fish could be predicted from the toxicity of the individual poisons and the toxic threshold concentration of the mixture being that at which the sum of the individual ratios of concentrations in solution to threshold concentration equals unity (Herbert and Vandyke, 1964). Total toxicity of a sewage effluent can be predicted fairly well from the sum of the toxicities of the individual poisons which include ammonia, phenol, heavy metals, and cyanide (Lloyd and Jordan, 1963), but less so for sewage effluents mixing with industrial wastes (Lloyd and Jordan, 1964).

Threshold concentration. The threshold lethal concentrations of un-ionized ammonia reported by various investigators are shown in Table 9. The concentrations range from 0.1 mg/l for fish (Málaćea, 1968) to 2.1 mg/l for carp (Nehring, 1963). Wuhrmann et al. (1947) suggested that 0.1 mg/l is the maximum concentration of un-ionized ammonia which is tolerated by fish in fish culture. The Food and Agriculture Organization (1970) recommended from literature that 0.025 mg/l un-ionized ammonia may be the

maximal concentration which can be tolerated by fish for a long period.

Besides the un-ionized ammonia, the other forms of ammonia in water solution are also used for determining the toxicity of ammonia solutions to fish and their threshold concentrations. Ellis (1937) considered that 1.5 mg/l as NH_3 is the concentration unharmful to most varieties of fish. However, it was found that there was no experimental evidence to support the validity of this concentration (Doudoroff and Katz, 1950). Wuhrmann et al. (1947) showed that at pH 7.5 to 8.0 about 10 to 15 mg/l as ammonium radical would be required to produce a concentration of non-ionized ammonia that would be harmful to most sensitive fish. Lloyd (1961b) indicated that the threshold concentration of total ammonia for trout is 17.0 mg/l as nitrogen. The State of Illinois sets 1.5 mg/l as nitrogen as a water quality standard to prevent toxicity to fish (Bauer and Snoeyink, 1973). The Water Pollution Control Administration (1968) recommends that permissible concentrations of ammonia for fresh water should be determined by the flow-through bioassy with the pH of the test solution maintained at 8.5, DO concentration between 4 and 5 mg/l and temperature near the upper allowable levels. However, a recommendation on ammonia standard was not made.

Estuarine and Marine Fish

In contrast to freshwater fish, very few studies have been done on estuarine and marine fishes. Also the fish tested in brackish or sea water are salmoid fish and not the resident species of brackish or sea water. Anon (1963) indicated that the predicted toxicity of ammonia to rainbow trout in freshwater was found to be much higher than the observed values when applied to sea water. This fact is also demonstrated by Anon (1965) and Herbert and Shurben (1965) who found that the resistance of rainbow trout to ammonium chloride increased with increasing salinity up to maximum of about 30 percent sea water at which concentration of the 24-hr. TLm value was 2.3 times greater than in freshwater. The effect was less marked at high salinites, although resistance was still greater in 100 percent sea water than in freshwater. Hazel et al. (1971) showed with concentration of toxic un-ionized ammonia in ammonium chloride solution was influenced by pH, temperature, and salinities. Less un-ionized ammonia was formed in sea water at the same pH and temperature than freshwater. The authors also found that striped bass and three spined stickleback showed increased resistance to un-ionized ammonia in one-third sea water as opposed to fresh and sea water. The stickleback was about twice as resistant to ammonia as striped bass in brackish water at 15 C and six times as resistant in sea water. The 96.-hr. TLm values of striped bass in freshwater, brackish water and sea were respectively 2.8, 2.8, and 2.0 mg/l

TABLE 9. THRESHOLD CONCENTRATIONS OF UN-IONIZED AMMONIA PROPOSED FOR VARIOUS FRESHWATER SPECIES OF FISH

Concentration mg/l	Fish	References
0.1	Phoxinum phoxinus, Rhodeus sericeus	Malacea (1968)
0.29-0.41	Perch, roach, rudd, bream, trout	Ball (1967)
0.3-0.4	Rainbow trout fry	Wuhrmann and Woker (1948)
0.39 as N	Rainbow trout	Lloyd and Orr (1969)
0.5	Fish	Vamos (1963)
0.6	Rainbow trout	Herbert (1962)
0.6	Phoxinus phosinus	Woker and Wuhrmann (1950)
0.6	Minnows	Wuhrmann and Woker (1950)
0.6	Perca flavescens	Allan (1955)
0.6	Perca flavescens	Woker and Wuhrmann (1950)
0.7	Trout	Nehring (1963)
0.8	Brown trout	Woker and Wuhrmann (1950)
0.84	Rainbow trout	Lloyd and Herbert (1960)
0.9	Perca flaviatilus	Nehring (1963)
1.1	Mosquito fish	Hemens (1966)

TABLE 9. THRESHOLD CONCENTRATIONS OF UN-IONIZED AMMONIA PROPOSED FOR VARIOUS FRESHWATER SPECIES OF FISH (continued)

Concentration mg/l	Fish	References
1.2	Rutilus rutilus	Nehring (1963)
1.0-1.2	Squalus cephalus	Wuhrmann and Woker (1948)
2.1	Carp	Nehring (1963)

at 15° C and 1.9, 2.1, and 1.9 mg/l at 23 C. For the stickleback the 96-hr. TLm values in freshwater, brackish water and sea water are respectively 2.1, 5.2 and 10.4 mg/l at 15 C and 1.8, 2.4, and 2.3 mg/l at 23 C. Because the authors used the static bioassay, the ammonia excretion by the test fishes might change the pH values and concentrations of toxic un-ionized ammonia, causing some experimental errors.

Holland et al. (1960) found that 0.3-3.5 mg/l as NH_3 of NH_4OH in sea water with 19 per thousand chlorinity and pH 7.6-7.8 killed chinook salmon in 72 hours and 5.2 mg/l as NH_3 at pH 8.15 killed the fish in 20 hours. They suggested that 3.5-10.0 mg/l as NH_3 is the critical level of chinook salmon exposed for 72 hours to NH_4OH in aerated sea water. The Water Pollution Control Administration (1968) stated that the ammonia exerted a considerable toxic effect on all aquatic life within a range of less than 1.0 mg/l to 25 mg/l, depending on the pH and dissolved oxygen level present. It did not indicate what evidences there were and where they came from.

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Freshwater Fish

Residual components of detergents in sewage. After a synthetic detergent is used for a washing job and the waste water is released down the drain, it loses its identity as formulated. It becomes simply a "blend of residues" in waste water. The components which are not used up or decomposed during use fall into several groups, identified in the waste water by the type of their chemical structure, regardless of the particular detergent product from which they originated (Anon, 1962).

The most important group is surfactants or surface acting agents. The common household detergents are an anionic type whose surfactants are usually alkyl benzene sulfonate (ABS), linear alkylate sulfonate (LAS), alkyl aryl sulfonate, dodecylbenzene sulfonate, alkyl sulfate, sodium lauryl sulfate and other derivatives depending on the brands of the detergents. The ABS and LAS have been the most common surfactants in household detergents. Alkyl sulfate is decomposed during the sewage treatment process, but alkyl aryl sulfonate is not decomposed. It takes 14 days to decrease 25 mg/l to 10-15 mg/l (Degen et al. 1950). ABS is resistant to degradation in sewage treatment process and is often found in natural water (Payne, 1963).

Cationic detergents are less frequently used in household washing. In sewage they are neutralized by the anionic detergents that are almost certain to be present in excess in municipal sewage. The interaction of both detergents yields insoluble compounds that have neither detergent nor germicidal property (American Water Works Association, 1954). Non-ionic detergents are little used in household washing.

Except the surfactants which compose about 70 percent of constituents in synthetic detergents, the rest of the detergents are the inorganic builders. These builders usually contain pentasodium tripolyphosphate or tetrasodium phyrophosphate, sodium sulfate, sodium chloride, sodium silicate, sodium carboxymethyl cellulose, perfume and bleach (American Water Works Association, 1954; Ohio River Valley Water Sanitary Commission, 1963). The tripolyphosphate and pyrophosphate occupy the major portion of the builders and they break down to orthophosphate in natural water. Other constituents of the builders are small in quantity and appear to have no significant effects on water and sewage.

Detergents in surface water. Detergents are manufactured products and absent in unpolluted natural water. The presence of detergents in the surface water unquestionably indicates the occurrence of pollution, particularly sewage pollution. The concentrations of detergents in the surface

waters which have been reported varied greatly from zero mg/l in natural water to 5.1 mg/l in sewage polluted streams in the United States (Table 10). In British rivers, the detergent concentrations ranged from 0.2 mg/l to 4.0 mg/l. The concentration of detergents in the River Swine was between 1 mg/l and 2 mg/l (Prat. 1962).

Since replacement of hard ABS with soft LAS during 1965 (Pickering and Thatcher, 1970), the concentrations of detergents in many streams have been reported decreasing. In the Illinois River, Sullivan and Evans (1968) reported that detergent concentration was 0.55 mg/l during the ABS period from 1959 to 1965 and became 0.22 mg/l during the post ABS period from 1965 to 1966. The same reduction in detergent concentration in the Illinois River was reported by Sullivan and Swisher (1968). Tsai (1973) reported that in the sections of streams immediately below sewage outfalls in Maryland, Virginia, and Pennsylvania, in 1968 to 1971 the detergent concentrations ranged from zero mg/l to 1.2 mg/l, except for two extreme cases with 1.6 mg/l and 2.4 mg/l.

In sewage, the amounts of detergents have been reported to be 4 mg/l to 45 mg/l from various cities in the United States (American Water Works Association, 1954), 1 mg/l to 9.7 mg/l in Britain (Herbert et al. 1957; Lloyd and Jordan, 1963, 1964), and 10.7 mg/l in Europe (Mann, 1955). Roberts (1954) reported 20 mg/l detergents in sewage. The concentrations of ABS in domestic sewage were reported to be 5 mg/l to 10 mg/l (McGaukey and Klein, 1959) and 3.1 mg/l to 13.8 mg/l Ohio River Valley Sanitary Commission, 1963) and 1 mg/l to 10 mg/l (Mckinney and Donovan, 1959). Sullivan and Evans (1968) reported that in Germany the average concentration of detergents at 14 biological sewage treatment plants from 1962 to 1964, when the ABS was used as surfactant, was 5.4 mg/l and decreased to 1.2 mg/l in 1965 after LAS replaced ABS in detergent manufacture. In England during the same period the detergent in sewage decreased from 3.0 mg/l in the ABS years to 1.3 mg/l in the LAS years.

The effects of detergents on water, sewage treatment processes, and aquatic life have been studied extensively and reviewed by several investigators (Smith et al. 1954; U. S. Public Health Service, 1962; McKee and Wolf, 1963; Jong, 1964; Prat and Giraud, 1964; Marchetti, 1965a; Federal Water Pollution Control Administration, 1968; Banerji, 1970; Anon, 1971; Nietsch and Schöller, 1972.)

Modes of detergent intoxication. Gills of fish have been found to be the primary target organ of detergent intoxication (Mann, 1955; Schmid and Mann, 1961, 1962; Nehring, 1962; Klust and Mann, 1962; Mount, 1963; Lemke and Mount, 1963; Cairns and Scheier, 1963, 1966; Cairns et al. 1964; Swisher et al. 1964; Bock, 1965; Scheier and Cairns, 1966; Dooley, 1968; Brown et al. 1968; Swedmark et al. 1971).

TABLE 10. CONCENTRATIONS OF DETERGENTS REPORTED IN THE AMERICAN SURFACE WATERS

Conc. (mg/l)	Rivers	Year	References
0.05	Illinois River, Illinois	1965	Sullivan and Swisher (1968)
0-0.14	Rivers in America	--	Jente et al. (1961)
0.01-0.14	Drinking water at 32 cities in U.S.	--	Anon (1962)
0.04-0.12 (Average 0.078)	Kaskushia R. Illinois	1956-1957	Morgan and Engelbrecht (1957)
0.15	Ohio River at Willow Island	1957-1961	Ohio R. Valley Water Sanitation Commission (1963)
0.17	Ohio River at Anderson, Ohio	1957-1961	Ohio River Valley Water Sanitation Commission (1963)
0.22	Illinois River, Illinois	1965-1966	Sullivan and Evans (1968)
0.54	Illinois River, Illinois	1959	Sullivan and Swisher (1968)
0.5-5.1	Neosho River, Kansas	1956-1957	Culp and Stoltenburg (1953)
0.56	Illinois River, Illinois	1959-1965	Sullivan and Evans (1968)
0.59	Ohio River	--	Anon (1962)
0.5-1.3	Illinois River, Illinois	1959	Anon (1962), Sayers (1962)
0-2.4	Below sewage outfalls in Md. Va. Pa.	1968-71	Tsai (1973)

Brown et al. (1968) indicated that the pattern of gill damages by ABS proceeded from swelling and thickening of gill filaments to final destruction of the larmellar walls and total loss of mucous cells with lesion of the inner parts of the walls of the blood vessels. Destruction in the early stage is similar to that which occurs with acute heavy metal poisoning. The swelling and thickening of the gill in the bluegill sunfish by ABS was also described by Mount (1963) and Lemke and Mount (1963). Dooly (1968) reported that fish were killed in detergent solution and in many cases only skeletal portions of the gills remained. The gill damages in the bluegill sunfish by ABS was found at concentrations of 3 mg/l and low concentrations of LAS (Cairns et al. 1964). LAS attacks the gill membranes of bluegill sunfish at concentrations near or above the TLm value, but no damage at 2/3 the TLm value. The typical gill damage by ABS in bluegill sunfish, persisted even after eight weeks of recovery in clean water (Scheier and Cairns, 1966).

Gill damage impairs respiration and the salt balance of fish (Bock, 1965). Schmid and Mann (1961, 1962) showed that 5 mg/l sodium dodecylbenzene sulfonate reduced the epithelial cells of the rainbow trout and caused the respiratory folds of the gill lamina to stick together. Increased concentrations and prolonged exposure periods increased gill destruction. Uptake of oxygen by the gill was then diminished and the mechanisms of salt balance and urea metabolism were also impaired. Fish died of suffocation. The same destruction of the gill function was stated by Hassler et al. (1967) who indicated that lethal concentration of ABS greatly reduced the respiratory surface of the gill by lysis and clumping of the larmellae and edema in the epithelial cells of the larmellae and thrombi in the capillaries in the rainbow trout. The fish died of asphyxiation. However, Cairns and Scheier (1966) reported that in 60 mg/l NaCl and 18 mg/l ABS the regulatory ability and capacity for adjustment to sudden increases in salt in water remained unimpaired, despite considerable damage to the gill by ABS. Schmassman (1946) suggested that the strong wetting action of the detergents adversely affected the exchange of gas at the respiratory surface.

Besides gill damage, other organs have been reported to be damaged by detergents. Sierp and Thiele (1954) found the detergents in gills, intestines, and reproductive organs of fish exposed to detergents. Cairns and Scheier (1963) exposed pumpkinseed and bluegill sunfish to 5.6 mg/l ABS for three months and found that the fish increased oxygen consumption, erythrocyte count and parasitism in the mucous laden gill areas by lower invertebrates. The fish exhibited general pathological reaction was shown by increase in hemoglobin and erythrocyts and by decrease in leucocyte count (Krylov, 1969). The lethal concentration of ABS increased the microhematocrit values and total plasma protein

in rainbow trout (Hassler et al. 1967). Solon et al. (1969) suggested that certain surface active agents increased the permeability of biological membrane, and thus, uptake of pesticides in fish exposed to 1 mg/l LAS increased. Mann and Schmid (1965) found that common guppy exposed to 1 mg/l tetrapropylenebenzene sulfonate for six weeks showed signs of incipient liver damage. However, Lemke and Mount (1963) found that the bluegill sunfish exposed to ABS showed no damage to liver, spleen, kidney, or small intestine. Bock (1966) showed that 5 mg/l detergent destroyed carp gill epithelial membrane but did no damage to intestines. Pickering and Thatcher (1970) also indicated that the fat-head minnows killed by LAS showed no histopathological changes in organs.

The action of detergents on fish has been suggested to be primarily through the reduction of surface tension (Bock, 1966; Pitter and Matulova, 1967). Gloxhuber and Fischer (1968) indicated that the reduction in the surface tension of the water seemed to be the basic cause of the poisoning of the fish by anionic detergents. The anionic detergents act on the gills and the non-ionic detergents passed through the gills. The symptons of poisoning by the non-ionic detergents showed similarity to those due to local anesthetics like tetracaine and procaine. On the contrary, Marchetti (1964, 1966) found that there appeared to be no relationship between toxicity of anionic detergents and surface tension values to fish. He suggested that the toxicity of surface-active agents to fish depends on the characteristics of the medium and on the chemical structure, ionic character, and biodegradability of the individual surface active agent.

Bardach et al. (1965) studied the effects of ABS and LAS on the chemoreceptors of yellow bullhead. In a concentration as low as 0.5 mg/l of either ABS or LAS the fish suffered the erosion of the taste buds and the impairment of receptor function. The authors suggested that the fish that rely mainly on the chemical senses for finding food will go foodless in water containing around 0.5 mg/l detergents. The same damage or fatigue of the chemoreceptor cells was also found in the feet of starfish exposed to non-ionic detergent solutions (Mackie, 1970). Kisimoto and Adelman (1964) found that detergents altered squid giant axon conduction, because they affected the lipoprotein.

In the cellular levels, Manwell and Baker (1967) found that detergents disrupt macromolecular ensembles and facilitate the solubilization of certain proteins. The detergents remove lipid from lipoprotein comples and affect protein directly by irreversible binding or by causing a configuration change, or both. These changes may lead to activation or inhibition of enzymes. The anionic detergents cause inhibition and non-ionic detergents often cause activation. Pitter and Matulova (1967) also indicated that bacteriocidal action of detergents is due to their

chemical action on enzymes.

Toxicity and chemical structures of surfactants. Anionic detergents are less harmful to freshwater organisms than cationic detergents and the non-ionic detergents (Madai and Lan, 1964). In the anionic detergents, the hard ABS is much less toxic than soft LAS (Bock, 1964; Marchetti, 1964; Bock and Wickbold, 1966; Liebmann, 1966; Pickering, 1966; Kölbel and Kurzendörfer, 1969). Thatcher and Santner (1967) showed that LAS degrades faster and is two to four time more toxic than the slow degrading branched chain ABS. Kölbel and Kurzendörfer (1969) suggested that the surface tension is greatly reduced by detergents possessing long and unbranched alkyl chains which might explain the stronger effects of LAS as compared to ABS. Many authors have stated that rapid degradable detergents are generally more toxic to fish than those which undergo slow biological decomposition (Degens et al. 1950; Hiemitz and Pestin, 1962; Hirsch, 1963; Bock, 1964; Marchetti 1965b). However, Dooley (1968) found that ABS is more toxic than the ones which have short carbon chains (Pickering and Thatcher, 1970). The C_{14}-ABS affected the swimming activity of fish more adversely than C_{12}-ABS (Marchetti, 1968). Hirsch (1963) found that the position in which the benzenesulfonate radical is attached to the carbon chain affects the toxicity of the different isomers of ABS. The ABS containing 14-16 carbon atoms has LD_{50} values for goldfish at less than 1 mg/l, whereas for the ABS containing 10-13 carbon atoms the LD_{50} values are between 4 mg/l and 6 mg/l. The author concluded that the LD_{50} values increased with increasing decomposition. The same phenomenon was reported for LAS. Swisher et al. (1964) found that the alpha-C_{12}-LAS is less toxic than alpha-C_{14}-LAS to bluegill sunfish. Accordingly, the authors concluded that the methylene blue active substance (MBAS) determination would not indicate the chain length of the detergents. Thus, Pickering and Thatcher (1970) indicated that the establishment of water quality criteria of LAS for fish is made difficult by the non-specificity of the analytical methods used for the measurement of surfactant concentration and by the variation of the toxicity of the various surfactant chain length.

Because the toxicity of detergents decreases with the process of decomposition, the synthetic detergents which have been treated by sewage treatment processes are less toxic to fish than the equivalent concentration of original manufactured detergents in clean water (Robert, 1954; Herbert et al. 1957; Anon, 1965, 1957; Pentelow et al. 1957; Ohio River Valley Water Sanitation Commission, 1963). Hiemitz and Pestin (1962) found that for the tetrapropylenbenzene sulfonate fish would withstand much higher concentrations of methylene blue active substances in the effluents than in the crude sewage.

For the other varieties of detergents, there is no

difference in toxicity of dodecylbenzene sulfonic acid and its sodium salt (Marchetti, 1964). Dodecylbenzene sulfonate is more toxic to carp than tetrapropylenebenzene sulfonate (Ludemann and Kayser, 1963). Primary sodium alkyl sulfate is less toxic than secondary sodium alkyl sulfate (Arlet, 1959).

Toxicity of builders. Builders are relatively non-toxic to fish, although sodium tripolyphosphate is considerably more toxic than sodium sulfate (Henderson et al. 1959). For the other builders, the minimum lethal concentration to fish of sodium palmilate and sodium oleate was 10-12 mg/l in distilled water and 250-300 mg/l in hardwater (LeClerc and Delvaminck, 1952). Sodium nitrilotriacetic acid (NTA) has been used as a partial replacement for sodium tripolyphosphate. Its 24-hr. to 168-hr. TLm values for marine fish are 5,500 mg/l. NTA levels of 1,000 mg/l and lower are non-toxic to marine fish (Eisler et al. 1972).

The builders apparently decrease somewhat the toxicity of ABS to fish (Henderson et al. 1959). Hassler et al. (1967) found that fish exposed to ABS builder combinations were more sensitive to ABS but their 120-hr. TLm values were slightly higher than those fish exposed only to ABS. It appears that sodium builders made the fish more susceptible to ABS during the first 48 hours exposure and had little effect thereafter. The builders may even decrease the toxicity of ABS or make the fish sensitive to ABS after 48 hours.

Environmental factors affecting detergents toxicity. Effect of hardness on detergent toxicity differs greatly from that on soap. Toxicity of soap decreases greatly in hardwater, because it precipitates to form insoluble calcium salts (Klein, 1957; Henderson et al. 1959; LeClerc and Delvaminck, 1952; Ohio River Valley Sanitation Commission, 1960). For synthetic detergents, hardness of water appears to have no, or very little, effect on their toxicity to fish. There are several conflicting reports on the effects of water hardness on detergent toxicity. Henderson et al. (1959) and the Ohio River Valley Water Sanitation Commission (1960) reported that the average toxicity of detergents was slightly higher in hardwater than in soft water. Marchetti (1964) indicated that the toxicity of the anionic detergents increases with increasing hardness. On the contrary, Mann (1957) found that increased carbonate hardness decreased the toxicity of detergents. On the other hand, Schmassman (1946), LeClerc and Delvaminck (1952) and Cairns and Scheier (1963) stated that the toxicity of detergents was unaffected by hardness of the water. Mann (1962a) indicated that the content of lime in the test water was not an important factor in detergent toxicity. The above conflicting statements may be simply due to experimental variation and the water hardness may not be an important factor causing these variation.

High temperature increases the toxicity of LAS only

during the tests of short exposure (Hokanson and Smith (1971). The same effect of temperature on detergent toxicity was also indicated by Mann (1962a and 1968). Low dissolved oxygen is found to decrease the TLm value of detergents to fish (Herbert et al. 1957).

Lethal effects. There is a great amount of information available regarding the lethal effects of various detergents on fish (Table 11-13). The lethal concentrations reported for various species range from 0.5 mg/l to 22 mg/l for ABS, 0.64 mg/l to 15 mg/l for LAS, and 3.5 mg/l to 36 mg/l for the other anionic detergents. The differences are due to difference in test species, test condition, and the duration of the tests. McKee and Wolf (1963) stated that although there is a considerable amount of information with respect to the toxicity of detergents to various forms of aquatic life, it is still almost impossible to compare the works of different investigators. Unless standardized techniques are agreed upon and used, there appears to be little hope that comparison can often be made.

In addition to those listed in Table 11-13, there are many general statements on the toxic effects of detergents on fish. Schmassman (1946) reported that 20 mg/l of a German detergent killed fish in 12 hours. Mann (1955) stated that 10 to 25 mg/l synthetic detergents are toxic to fish and fish food. Marchetti (1965a) concluded from literature that concentrations of ABS between 0.25 mg/l to 26 mg/l are lethal to various species of freshwater fish. Marchetti (1964) showed that the toxic concentration of 17 anionic detergents to goldfish and Salmo iridus for six hours at 15 C ranged from 8.5 mg/l to 43/7 mg/l. Cairns et al. (1964) indicated that bluegill sunfish survived exposure to 9.8 mg/l ABS for several months. Mann (1957) showed that there is little difference in the toxic effects of the various detergents. Their limiting concentration varies from 10 mg/l to 50 mg/l for fish. Holland et al. (1960) found that critical levels of several industrial detergents to silver salmon and rainbow trout ranged from 5.6 mg/l to 112 mg/l in aerated lake water. Pohl and Svec (1963) found that the concentration of 20 mg/l Dubaral (an ABS type detergent) or more was lethal to relatively resistant aquatic organism. Jong (1964) stated that fish are sensitive to detergents, but man is unlikely to consume harmful quantities in drinking water.

Thatcher (1966) compared the susceptibility of several freshwater fish to ABS. He found that black bullhead, carps, and common shiners are more resistant than the emerald shiners which are the most sensitive. When the mortality occurred, it most often took place in the first 48 hours. The same phenomenon was found by Lemke and Mount (1963). However, in the LAS solution, the fish mortality continued to occur also on the third and fourth days (Thatcher and Santner, 1967).

The sensitivity of fish to detergents varies at

TABLE 11. LETHAL EFFECTS OF ALKYL BENZENE SULFONATE (ABS) TO FRESHWATER FISHES

Concentrations mg/l	Fish	Test condition	Survival time	References
0.5	Rainbow trout	11.8-16.5 C, pH 9.5, Hardness 290 mg/l as $CaCO_3$	72-hr. TLm	Brown et. al (1968)
0.56	Rainbow trout	11.8-16.5 C, pH 7.5, Hardness 290 mg/l as $CaCO_3$	48-hr. TLm	Brown et al. (1968)
2.4	Fathead minnow fry	--	9-day TLm	Pickering (1966)
2.53	Rainbow trout	--	120-hr. TLm	Hassler et al. (1967)
3.0	Rainbow trout	--	8-weeks TLm	Herbert el a. (1957)
3.48	Rainbow trout	--	24-hr. TLm	Hassler et al. (1967)
3.5-6.0	Freshwater fish	--	Average TLm values	Ohio River Water Sanitary Commission (1960-1963)
4.3	Fathead minnow	Hardness 360 mg/l as $CaCO_3$	96-hr. TLm	Henderson et al. (1959)
4.9	Fathead minnow eggs	--	9-day TLm	Pickering (1966)
5.6	Bluegill sunfish	Hardness 360 mg/l as $CaCO_3$	96-hr. TLm	Henderson et al. (1959
6.6	Fathead minnow	pH 7.4, hardness 18 mg/l as $CaCO_3$	96-hr. TLm	Henderson et al. (1959)
7.4	Emerald shiner	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)

TABLE 11. LETHAL EFFECTS OF ALKYL BENZENE SULFONATE (ABS) TO FRESHWATER FISHES (continued)

Concentrations mg/l	Fish	Test condition	Survival time	References
7.5	Bluegill sunfish	Hardness 360 mg/l as $CaCO_3$	48-hr. TLm	Henderson et al. (1959)
7.7	Bluntnose minnow	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
8.2	Bluegill sunfish	Hardness 360 mg/l as $CaCO_3$	24-hr. TLm	Henderson et al. (1959)
8.2	Bluegill sunfish	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
8.9	Stone roller	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
9.0	Sand shiner	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
9.2	Silverjaw minnow	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
9.5	Rosefin shiner	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
11.3	Fathead minnow	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
12.0	Rainbow trout	Hardwater	8 hours	Herbert et al. (1957)
15.5-18.3	Bluegill sunfish	25 C, pH 7.1-7.3, DO 7.4-7.6 mg/l, hardness 19-21 mg/l as $CaCO_3$	30 days TLm	Lemke and Mount (1963)

TABLE 11. LETHAL EFFECTS OF ALKYL BENZENE SULFONATE (ABS) TO FRESHWATER FISHES (continued)

Concentrations mg/l	Fish	Test condition	Survival time	References
15.8-21.2	Bluegill sunfish	25 C, pH 7.1-7.3, DO 7.4-7.6 mg/l, hardness 19-21 mg/l as $CaCO_3$	96-hr. TLm	Lemke and Mount (1963)
16.5-24.8	Bluegill sunfish	25 C, pH 7.1-7.3, DO 7.4-7.6 mg/l, hardness 19-21 mg/l as $CaCO_3$	24-hr. TLm	Lemke and Mount (1963)
17.0	Common shiner	23 C, pH 7.5, DO 6.9 mg/l, hardmess 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
17.2-17.5	Bluegill sunfish	20 C, soft and hard water	24-,48-, and 96-hr. TLm	Cairns and Scheier (1963)
18.0	Carp	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)
21.9-22.4	Pumpkingseed sunfish	20 C, soft water	24-,48-, and 96-hr. TLm	Cairns and Scheier (1963)
22.0	Black bullhead	23 C, pH 7.5, DO 6.9 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher (1966)

TABLE 12. LETHAL EFFECTS OF LINEAR ALKYLATE SULFONATE (LAS) TO FRESHWATER FISHES

Concentrations mg/l	Fish	Test Condition	Survival time	References
0.64(C_{14}-LAS)	Bluegill sunfish	--	96-hr. TLm	Swisher et al. (1964)
0.9	Fathead minnow fry	--	9-day TLm	Pickering (1966)
2.2	Bluegill sunfish fingerling	24-25 C, DO 25 mg/l	24- and 48-hr. TLm	Hokanson et al. (1971)
2.23 and 2.54	Bluegill sunfish	25 C, DO saturated, hard water	24-hr. TLm	Hokanson (1968)
2.3	Fathead minnow eggs	--	9-day TLm	Pickering (1966)
2.8 to 2.9	Bluegill sunfish fingerlings	Hardwater, hardness 290 mg/l as $CaCO_3$	Lethal threshold	Hokanson et al. (1971)
3.1(C_{12}-LAS)	Bluegill sunfish	--	96-hr. TLm	Swisher et al. (1964)
3.2 and 5.6	Bluegill sunfish sac fry	15 C, DO 8.2 mg/l	24- and 48-hr. TLm	Hokanson et al. (1971)
3.3	Emerad shiner	23 C, pH 7.5, DO 6.5 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher and Santner (1967)
3.5	Fathead minnow	--	96-hr. TLm	Solon et al. (1969)
3.7 and 4.0	Bluegill sunfish eggs	--	TLm value	Hokanson et al. (1971)

TABLE 12. LETHAL EFFECTS OF LINEAR ALKYLATE SULFONATE (LAS) TO FRESHWATER FISHES (continued)

Concentrations mg/l	Fish	Test condition	Survival time	References
4.0	Bluegill sunfish	23 C, pH 7.5, DO 6.5 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher and Santner (1967)
4.0 to 4.5	Bluegill sunfish fingerling	Soft water, hardness 15 mg/l as $CaCO_3$	Lethal threshold	Hokanson et al. (1971)
4.2	Fathead minnow	23 C, pH 7.5, DO 6.5 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher and Santner (1967)
4.2 and 5.0	Fathead minnow	pH 7.5-9.0, hardness 200 mg/l as $CaCO_3$	96-hr. TLm	Pickering and Thatcher (1970)
4.25	Bluegill sunfish	25 C, DO Saturated, soft water	24-hr. TLm	Hokanson (1968)
4.9	Common shiner	23 C, pH 7.5, DO 6.5 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher and Santner (1967)
5.7 and 5.4	Bluegill sunfish sperms	--	50% no motile	Hokanson and Smith (1971)
6.4	Black bullhead	23 C, pH 7.5, DO 6.5 mg/l, hardness 50 mg/l as $CaCO_3$	96-hr. TLm	Thatcher and Santner (1967)
15	Bluegill sunfish sperms	--	No motile	Hokanson and Smith (1971)

TABLE 13. LETHAL EFFECTS OF THE SURFACTANTS OTHER THAN ABS AND LAS TO FRESHWATER FISHES

Surfactants	Concentration mg/l	Fish	Test Condition	Survival time	References
Alkyl aryl sulfonate	4.2 and 3.7	Bluegill sunfish	pH 6.9-7.5, hardness 84-136 mg/l as $CaCO_3$	24- and 48-hr. TLm	Turnbull et al. (1954)
Alkyl aryl sulfonate	15	Fish	--	100% motality in 6 hours	Sierp and Thiele (1954)
Alkyl aryl sulfonate	18	Roach, Rhodeus sp.	--	Limiting concentration	Degens et al. (1950)
Alkyl aryl sulfonate	36	Carp, Idus. sp.	--	Limiting concentration	Degens et al. (1950)
Alkyl sulfate	15	Fish	--	100% mortality in 20 hours	Sierp and Thiele (1954)
Dodecyl benzene sulfonate	5	Trout eggs	--	35 days	Mann and Schmid (1961)
Dodecyl benzene sulfonate	10	Trout sperm	--	Minimum concentration for mobility	Mann and Schmid (1961)
Korenyl benzene sulfate	5	Trout sperm	--	Minimun concentration for mobility	Mann and Schmid (1961)

TABLE 13. LETHAL EFFECTS OF THE SURFACTANTS OTHER THAN ABS AND LAS TO FRESHWATER FISHES (continued)

Surfactants	Concentration mg/l	Fish	Test condition	Survival time	References
Sodium alkyl aryl sulfonate	7-12	Rainbow trout	9-14 C, pH 6.9, alkalinity 22 mg/l as $CaCO_3$	6 hours	Wurtz-Arlet (1960)
Sodium alkyl sulfate	9	Roach, Rhodeus, Idus	--	Limiting concentration	Degens et al. (1950) (1950)
Sodium alkyl sulfate	18	Carp	--	Limiting concentration	Degens et al. (1950)
Sodium dodecyle benzene sulfonate	6-7	Fish	18-23C, soft and hard water	Minimum lethal concentration	LeClerc and Devlaminck (1952)
Sodium dodecyle benzene sulfonate	4.0, 3.5 and 3.5	Fish	--	24-, 48-, and 96-hr. TLm	Ohio River Water Sanitary Commission (1960, 1963)

different development stages of the life cycle (Hokanson, 1968). In the early development stage, rainbow trout is especially sensitive to detergents (Marchetti, 1965b). Arlet (1959) found that the trout eggs hatched normally after 24 hours exposure to anionic detergents concentration (sodium alkyl sulfate) up to 10 mg/l. However, none of the alevins could withstand exposure for 15 hours to water containing 5 mg/l or more. Six weeks-old alevins were more sensitive than the young ones. Maldura (1961a and b) found that the most sensitive stage of rainbow trout to ABS is the feeding sac fry which almost completely absorbed the vitaline vesicle just prior to self feeding. After the sac was absorbed, resistance increased with age. Hokanson and Smith (1971) indicated that the most sensitive stage of development in the bluegill sunfish to LAS is the feeding sac fry, while the most tolerant stage is newly hatched sac-fry. Eggs and fingerlings had similar intermediate tolerance levels. The lethal threshold concentrations of the bluegill sunfish varied from 1.9 to 2.9 mg/l LAS. For the fathead minnows, the most sensitive stage in the life cycle to LAS is newly hatched fry (Pickering and Thatcher, 1970). The eggs are only one half as resistant as the adult (Pickering, 1966). Dooley (1968) found that in detergents young mosquito fish die earlier than old fish and females consistently withstand concentrations that are lethal to males.

Mann and Schmid (1961) investigated the effect of eight synthetic detergents on the mortality of trout sperm. Minimum harmful concentrations range from 5 mg/l to 100 mg/l. Fertilized eggs grown in a solution of dodecylbenzene sulfonate were all killed in 35 days in a concentration of 5 mg/l.

Damage caused by detergents to fish is often irreversible. Bardach et al. (1965) exposed yellow bullhead to ABS and LAS and then they were transferred to clean water. The fish were not fully recovered from the damages from chemoreceptors after 6 weeks. The gill damages of pumpkinseed sunfish in 18 mg/l ABS for 24 hours persisted even after 8 weeks of recovery in clean water. On the contrary, Schmassman (1946) immersed fish for a short time in 100 mg/l of German detergents and they recovered completely when they were transferred to freshwater (20 mg/l killed the fish in 12 hours). Wurtz-Arlet (1960) found that the concentrations of sodium alkyl aryl sulfonate and octophenol at which the rainbow trout might not have apparent damage after one hour exposure and 24-hour follow up period in clean water were 10-12 mg/l and 10-25 mg/l. After six hours exposure the concentrations for both detergents were 5-7 mg/l. It appears that recovery of fish in clean water after being exposed to lethal concentrations of detergents depends on the concentration and exposure period.

Fish appear to increase their resistance to detergent by slow acclimation (Mann, 1962a; Degens et al. 1950). Anon (1961) stated that roach will die in water containing 5 mg/l of detergents but the fish may gradually be acclimated to

live in water containing 20 mg/l detergents. The bluegill sunfish acclimated at 13 mg/l ABS for 30 days had a 48-hr. TLm value of 25.4 mg/l, 63 percent higher than the control which had 16 mg/l (Lemke and Mount, 1963). Hokanson and Smith (1971) found that lethal threshold concentration of bluegill sunfish acclimated to sublethal concentration of LAS was about 50 percent higher at air saturation and 400 percent higher at 2 mg/l DO than the control.

Many authors have stated that the concentrations of detergents found in the natural surface water are much lower than the concentration found to be harmful to fish. Herbert et al. (1957) concluded from their studies on toxicity of detergent on rainbow trout that in rivers in Great Britain, synthetic detergents are unlikely to cause serious damage to fish, as most of them will be in the form of residual materials remaining after sewage treatment, and their concentration in rivers water are usually less than 3 mg/l. Mann and Schmid (1961) found that the concentration of 5 mg/l of Dodecylbenzene sulfonate which killed fertilized trout eggs in 35 days may be exceeded by the amount found locally in polluted water. The Ohio River Valley Water Sanitation Commission (1963) concluded from literature that the TLm values of ABS to fish is 3.5-6 mg/l, and the levels are many times higher than those normally occurring in surface water (Table 10). Bock (1966) stated that soap and detergents in their normal use are not toxic to fish unless present in excessive quantities. In the Upper Mississippi River, Hokanson (1968) also found that there was no evidence that the detergents present in polluted reaches had any effects on young bluegill sunfish. In Maryland, Virginia, and Pennsylvania, Tsai (1973) indicated that the detergent concentrations in the river water immediately below sewage outfalls were much lower than the concentration harmful to fish. However, Webb and Casey (1961) showed that fish kills which occurred below the sewage outfall were attributed to 8 mg/l ABS from a sewage treatment plant effluent.

Fish behavior. In the lethal concentration of detergents fish showed a strong sliming of skin and gills, disturbance of equilibrium, turning to a side position and finally death (Mann, 1955; Maldura, 1961a; Hassler et al. 1967). The fish also showed a violent and erratic swimming and initial increase in respiration (Maldura, 1961a; Hassler et al. 1967).

Marchetti (1968) showed that the concentration ABS and LAS have adverse effects on the swimming ability of fish. In contrast, the marked damage to the gills by ABS does not affect the swimming performance of sunfish (Lemke and Mount, 1963; Cairns and Scheier, 1963) and oxygen consumption was altered only slightly (Cairns and Scheier, 1963).

Flagfish decreased breeding behaviors and did not produce viable eggs in 11.5 mg/l and 24 mg/l ABS (Foster and Cairns,

1966). After the four days of exposure to 11.5 mg/l to 24 mg/l ABS the flagfish also showed changes in feeding behavior. The changes suggest that the specific effect of the ABS is to inhibit the input of sensory information by which the fish distinguish between palatable-edible material and unpalatable-inedible material, probably by damaging the olfactory epithelium in the nasal capsule (Foster et al. 1966).

Rainbow trout avoided the ABS at concentrations as low as 0.37 mg/l (Sprague and Drury, 1969). However, at 10 mg/l, a nearly lethal level, fish were confused and unable to avoid. Perhaps this resulted from a lag in rinsing the detergent from the sensory receptor. Korpela (1969) also indicated that stickleback can detect quite low concentrations of detergent which could be harmful, but if the concentration of detergent is high, rapid damage is caused to the fish which are then unable to take avoiding action.

Fish growth. Mann and Schmid (1965) found that 1 mg/l of tetrapropylenebenzene sulfonate impaired growth and weight of common guppy when they were exposed for six weeks. Lemke and Mount (1963) found the gill damage to bluegill sunfish by ABS was coincident with abrupt change in growth rate of the fish.

Effects of detergents on toxicity of other chemicals. Sublethal concentrations of detergents in sewage effluents may increase the toxicity of some other pollutants (Pentelow et al. 1957; Federal Water Pollution Control Administration, 1968). Anon (1956) and Herbert (1956) found a possible synergistic relation between detergents and ammonia. Calamari and Marchetti (1970) found the synergistic reaction to goldfish between copper and 1 mg/l of ABS. The same reaction was found between ABS and mercury to rainbow trout. Solon et al. (1969) found that the 96-hr TLm value of fathead minnow to parathion decreased from 1.4 mg/l to 0.72 mg/l in sublethal concentration of 1 mg/l LAS. However, toxicity of endrin has no synergistic action with LAS. The synergistic reaction with 1 mg/l LAS was also found with methyl parathion, ronneltrithion and trichloronat, but not with dicapthon, guthion and EPN (Solon et al. 1970). The synergistic reaction of detergents with insecticides was suggested to be a biochemical interaction in which the LAS activated the enzymes responsible for oxidation of the thiophosphate compounds to their oxygen analogues or deactivating the enzymes responsible for detoxication, as well as purely physical interaction in which the LAS increased the solubility of the pesticides on the gills or skin membrane. The latter appears to be more acceptable. Solon et al. (1969) gave another hypotheses that detergents increases the permeability of biological membrane to increase the uptake of insecticedes in fish. The same concept was expressed by Mann (1962b, 1965) who suggested that the detergents increase the uptake of phenol and oil in the fish and thus the taste of fish flesh is adversely affected.

Anon (1956) found that trout increases susceptibility to low concentrations of oxygen in the presence of snythetic detergents, even in concentrations as low as 1.8 mg/l. Lemke and Mount (1963) attributed this increase in susceptibility to low DO to the damage of fish gills by detergents.

Dugan (1967) showed that goldfish which had a prior history of long term exposure to sublethal concentration of ABS were more susceptible to the toxic effects of dieldrin and DDT than were fish that had not previously been exposed to ABS. In contrast, Cairns and Scheier (1964) found that the pumpkinseed sunfish which had prior exposure to a concentration of ABS (12 mg/l) for five weeks, and which caused marked gill damage, did not undergo any gross changes in tolerance to either zinc or temperature increase. On the other hand, Brown et al. (1968) showed that fish exposed to zinc at a concentration of 0.8 mg/l for 100 days were more sensitive to the mixture of zinc and detergents, but were not more sensitive than the controls when exposed to detergents alone.

Safety concentration. The threshold concentration of detergents proposed for several freshwater species by various authors are shown in Table 14. They range from 1 mg/l to 6.31 mg/l for ABS and 0.2 mg/l to 1 mg/l for LAS. Pickering and Thatcher (1970) estimated that the LAS concentrations between 14 and 28 percent (application factor) of the 96-hr. TLm value for a given species is the maximum for long-term exposure of that species without affecting survival. The Federal Water Pollution Control Administration (1968) recommended that the concentration of ABS and LAS should not exceed 1/7 of the 48-hr. TLm values.

Estuarine and Marine Fishes

There are few studies of the effects of detergents, particularly of household anionic detergents, on estuarine and marine fishes. The lethal effects of ABS and LAS studied by Eisler (1965), Eisler et al. (1972) and Swedmark et al. (1971) are summarized in Table 15. Eisler (1965) indicated that toxicity of ABS was affected by salinity. Eel exposed to 10 mg/l ABS for 96 hours and mummichogs exposed to 20 mg/l for 120 hours were relatively unaffected at salinities below 15 per thousands, but dies at salinities approaching that of sea water. No biological effects (morphology, growth, red blood cell count, gonadosomatic index, and liver condition) of 10 mg/l ABS on mummichogs for 150 days at salinities 10, 20, 30, and 40 per thousands. TLm values change only slightly between 24 hours and 96 hours, suggesting that toxic properties of a detergent are manifested within the first 24 hours. Eisler et al. (1972) also found that syndet GA (LAS as surfactant) was least toxic to mummichogs at the lowest salinity (5%) with increasing mortality at high salinity. This may be due to stress associated with hypersaline environment rather than to a

TABLE 14. THRESHOLD CONCENTRATIONS OF SYNTHETIC DETERGENTS PROPOSED FOR FRESHWATER FISH

Detergents	Concentration mg/l	Fish	References
ABS	1	Fish	Federal Water Pollution Control Administration (1968)
ABS	1-2	Sensitive fish	Henderson et al. (1959)
ABS	3	Bluegill sunfish	Lemke and Mount (1963)
ABS	5.15-5.23	Bluegill sunfish	Cairns and Scheier (1963)
ABS	6.31	Pumpkinseed sunfish	Cairns and Scheier (1963)
LAS	0.2	Fish	Federal Water Pollution Control Administration (1968)
LAS	0.6-0.9	Fathead minnow	Pickering (1966)
LAS	0.63-1.2	Fathead minnow	Pickering and Thatcher (1970)
LAS	1	Bluegill sunfish	Hokanson and Smith (1971)
Haleo	0.37	Bluegill sunfish	Turnbull et al. (1954)
Akyl aryl sulfonate	0.86	Bluegill sunfish	Turnbull et al. (1954)
Dubaral (ABS type)	2	Fish	Pohl and Svec (1963)
Detergents	5	Freshwater organisms	Madai and Lan (1964)

change in physical and chemical property of syndet. Mann (1972) indicated that the toxicity of tetrapropylenebenzene sulfonate to eel and Gammarus titrinus increased with increasing salinity. He suggested that the increasing toxicity was due to a change in surface tension under the influence of salt content. Concentrations became lethal for the various species of marine life when the surface tension was reduced to 40 dyn/cm, corresponding to tetrapropylenebenzene sulfonate concentrations of 5 mg/l to 10 mg/l. This means that the detergents will be harmful only at outlets of concentrated and biologically undegraded wastewater. In the vicinity of industrial sewage effluent outfalls near Cuxhaven, Emdem, and Tonning, the maximum concentration of detergents at coastal water was 0.3 mg/l. The effect of salinity on the toxicity of synthetic detergents is contrasted to that of soap which becomes less toxic in high salinity, because the soap becomes insoluble white precipitate in salt water. Eisler and Devel (1965) found that there was high mortality among mummichogs exposed to 750-1,500 mg/l soap at salinity of 21 per thousand or less, but at higher salinities few or no deaths were observed.

Swedmark et al. (1971) studied the effects of five detergents (the anionic ABS, LAS and LES3EO, and the non-ionic NP10ED and TAE10ED) on marine organisms. The soft anionic LAS and LES3EO were more toxic to marine fish than the hard ABS and non-ionic detergents. Fish were more susceptible than bivalves, while crustacea were considerable more resistant. Within each of these three systematic groups, more active species were more sensitive than were less active species. Developmental stages were more sensitive than adults. The first reaction to the surfactants was increased activity, indicating avoidance reaction, followed by inactivation, immobilization, and death. The detergents affected gill membrane where gas and ion exchanges and urea metabolism take place. Swelling of the gill epithelium and increase in mucus secretion were found.

Holland et al. (1960) showed that the critical concentration of Gamlen CW to chinook salmon in aerated sea water was 17.8 ng/l. Portmann (1972) found that 48-hr TLm of octylphenol 11 ethylene oxide and Dobes 055 (non-ionic detergents) for flounder was 33-100 mg/l and 10-33 mg/l respectively. Thus, he concluded that the detergents discharged to the marine environment are relatively harmless since concentrations in excess of 20 mg/l are unlikely even in raw sewage. Bellan et al. (1972) compared the action of 20 non-ionic and 15 anionic detergents on marine invertebrates. The former are 1.5 to 2.0 times more toxic than the latter. Eisler et al. (1972) showed that 168-hr TLm values for syndets (NTA containing detergents) for marine organisms ranged from 4.6 mg/l to 36 mg/l total packaged product.

Eisler et al. (1972) found that NTA (Na-nitrilotriacetic acid), the new type of builder, was considerably less toxic than tripolyphosphate. The 168-hr TLm of sodium tripolyphosphate to marine fish was 4,600 mg/l to 5,000 mg/l. The

TABLE 15. LETHAL EFFECTS OF DETERGENTS ON ESTUARINE AND MARINE FISHES

Detergents	Concentration mg/l	Fish	Test condition	Survival time	References
ABS	3.5	Cod	6.5 C	96-hr. TLm	Swedmark et al. (1971)
ABS	6.5	Flounder	6.5 C	96-hr. TLm	Swedmark et al. (1971)
ABS	7.0	Silverside	20 C, 20% salinity	96-hr. TLm	Eisler (1965)
ABS	7.2	Silverside	20 C, 24% salinity	24-hr. TLm	Eisler (1965)
ABS	7.5	Eel	20 C, 20% salinity	96-hr. TLm	Eisler (1965)
ABS	8.0	Eel	20 C, 24° salinity	24-hr. TLm	Eisler (1965)
ABS	8.2	Winter flounder	20 C, 20° salinity	96-hr. TLm	Eisler (1965)
ABS	10.1	Mullet	20 C, 20% salinity	96-hr. TLm	Eisler (1965)
ABS	12.0	Mullet, winter flounder	20 C, 24% salinity	24-hr. TLm	Eisler (1965)
ABS	22.5	Mummichogs	20 C, 20% salinity	96-hr. TLm	Eisler (1965)
ABS	23.5	Mummichogs	20 C, 24% salinity	24-hr. TLm	Eisler (1965)
LAS	1.0	Cod, flounder	15-17 C	96-hr. TLm	Swedmark et al. (1971)
LAS	1.0-5.0	Plaice	6.5 C	96-hr. TLm	Swedmark et al. (1971)
LAS	1.0	Cod	6.5 C	96-hr. TLm	Swedmark et al. (1971)

TABLE 15. LETHAL EFFECTS OF DETERGENTS ON ESTUARINE AND MARINE FISHES (continued)

Deter-gents	Concentra-tion mg/l	Fish	Test condition	Survival time	References
LAS	1.5	Plaice	6.5	96-hr. TLm	Swedmark et al. (1971)
LAS	2.1	Mummichogs	20 C, 20% salinity	168-hr. TLm	Eisler et al. (1972)
Syndet CH (LAS as surfactant)	7.2, 4.6 and 4.6	Striped bass	20 C, pH 8.0, DO 4.0, mg/l, 20% salinity	24- 96- and 168-hr. TLm	Eisler et al. (1972)
Syndet CH	15.0, 9.1 and 7.5	Mummichogs	20 C, pH 8.0, DO 4.0 mg/l, 20% salinity	24-, 96-, and 168-hr. TLm	Eisler et al. (1972)
Syndet G (LAS as surfactant)	12.7, 8.7, and 8.7	Striped bass	20 C, pH 8.0, DO 5.0 mg/l, 20% salinity	24-, 96-, and 168-hr. TLm	Eisler et al. (1972)
Syndet G	23.6, 13.1, and 12.7	Mummichogs	20 C, pH 8.0, DO 4.0 mg/l, 20% salinity	24-, 96-, and 168-hr. TLm	Eisler et al. (1972)

24-, 96-, and 168-hr. TLm of NTA were 5,500 mg/l for both striped bass and mummichogs. Pathological changes in striped bass and mummichogs were observed in proximal kidney tubules at NTA levels greater than 3,000 mg/l. However, significant intestinal lesion was noted in mummichogs even at NTA levels as low as 1 mg/l. The mixture of syndet GA and CH (LAS as surfactant and NTA as builder) and Cd^{2+} and H^{+} caused additive effects of mummichogs and could be expressed as a simple summation of the toxicity of individual components.

Swedmark et al. (1971) found that fish may recover after exposure to lethal concentrations of LAS for a time less than fatal immersion time which decreases rapidly with increasing concentrations. Complete recovery of normal behavior in cod fish occurred within 24 hours. The maximum exposure time which permitted cod fish to recover completely varied from two days in 10 mg/l to three days in five mg/l non anionic NP10EO. The effects of anionic detergents are found to be irreversible in fish sooner than those of non-ionic detergents. This may be due to the property of anionic detergents to form protein salts which might inhibit the penetration of these surfactants into the gills, rather than passing through the gill epithelium as it does in non-ionic detergents.

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HYDROGEN SULFIDE

Freshwater Fish

Hydrogen sulfide in water. Hydrogen sulfide seldom occurs in natural water where dissolved oxygen is sufficient. It is produced almost entirely by the anaerobic decomposition of organic materials, particularly in the bottom sediments, such as in acid lakes in Texas during the summer, where as high as 2.13-7.25 mg/l was recorded (Bonn and Follis, 1967) and ice covered lakes in Minnesota, where 0.3-0.6 mg/l hydrogen sulfide was found during the winter fishkills (Scidmore, 1957). In streams it may occur naturally at the sediment-water interface (Adelman and Smith, 1970) particularly in those which have sludge deposited from paper mills and sewage effluents (Colby and Smith, 1967; Smith and Oseid, 1972). The concentrations of 0.02-0.8 mg/l hydrogen sulfide were reported within 1 to 20 millimeters of the bottom in a Minnesota river below paper mills, when they were undetectable above these strata (Colby and Smith, 1967). In the water columns of streams or lakes receiving sewage, hydrogen sulfide may be caused by anaerobic decomposition of organic effluents (Ellis, 1937; Longwell and Pentelow, 1935; Ziebel et al. 1970). The other direct sources of hydrogen sulfide in water are industrial wastes such as from tanneries, textile mills, chemical plants, paper mills and gas-manufacturing works (McKee and Wolf, 1963; Anon, 1972).

Mode of toxic action. Hydrogen sulfide (H_2S) in water dissociates into H^+, HS^- and S^{--}, according to pH and temperature. The degree of its dissociation is greater in higher pH. At pH value of 9 about 90 percent of the sulfide is in the form of HS^-, at pH 7, un-ionized H_2S and HS^- are about equally divided, and at pH 5 more than 99 percent is present as un-ionized H_2S (McKee and Wolf, 1963). The toxicity of hydrogen sulfide to fish is known to be associated with the concentration of un-ionized H_2S (Bonn and Follis, 1967; Longwell and Pentelow, 1935). The un-ionized molecules of hydrogen sulfide penetrate living tissues more readily than do sulfide ions. The rate of entrance of H_2S into cells of Valonia macrophyta is proportional to the concentration of molecular H_2S in the external solution (Jacques, 1936).

Jones (1947) indicated that the toxic action of hydrogen sulfide on fish resembled that of hydrogen cyanide, affecting the respiration of stickleback. The hydrogen sulfide caused a layer of edema in gills under their epithelium which swelled in the larmella to a non-functional state (Wood, 1960). Chief symptoms of fish exposed to a high concentration of hydrogen sulfide were found to be irritation, frenzied rushing, respiratory paralysis, stupor, loss of equilibrium, and oxygen hunger (Belding, 1927)

In a fish pond the low H_2S was found to promote the

respiration of algae, while photosynthesis was reduced to a minimum, and gas vacuoles developed in the cells causing flotation of a dense mass of algae which diminished the oxygen supply. As the H_2S concentration increased, all aquatic organisms, whose metabolism depended on iron-containing enzymes, were destroyed (Vamos, 1965). Chromatograms carried out on the amino acids produced by aquatic plants exposed to H_2S showed vivid blue bands between those for valine and the amino acid (Vamos, 1967). In humans, at high concentrations of H_2S, it immediately reduced the oxyhemoglobic, and at lower concentrations it inactivated respiratory enzymes, primarily the cytochrome system (Goodman and Gilman, 1955).

Environmental factors affecting hydrogen sulfide toxicity. Dissolved oxygen, temperature, and pH are known to affect hydrogen sulfide toxicity to fish. Hydrogen sulfide becomes more toxic in low DO to fish (Shelford, 1917; Adelman and Smith, 1972) and to fish eggs and fry (Smith and Oseid, 1972; Colby and Smith, 1969). Smith and Oseid (1970) indicated that low DO aggravated the condition by lowering fish tolerance to hydrogen sulfide and by exerting a direct effect on the eggs. However, no increase in mortality of northern pike eggs from hydrogen sulfide with decreased oxygen concentration was found, but there was an increase in mortality in their sac fry (Adelman and Smith, 1970). Adelman and Smith (1972) showed that decreasing DO concentration increased toxicity with lowered oxygen was not very great. Since complete mortality or survival occurred within a narrow range, the DO would not have an important effect on hydrogen sulfide toxicity in the environment, except at very low DO concentration. Also, prior acclimation of the goldfish to DO had little effect on acute toxicity of hydrogen sulfide to goldfish.

It is expected that hydrogen sulfide would become more toxic to fish at low pH, because more toxic un-ionized hydrogen sulfide is formed (Doudoroff and Katz, 1950; Anon, 1972). From pH 6.8 to 6.0 the hydrogen sulfide toxicity to brown trout increased slightly (Longwell and Pentelow, 1935). For temperature, Adelman and Smith (1972) found that the relation of hydrogen sulfide toxicity to temperature was negatively logarithmic over the range of 6.5-25 C for goldfish.

Lethal effects. Lethal effects of hydrogen sulfide on freshwater fish are shown in Table 16. Perhaps due to the difference in experimental methods, conditions, and species, there is a great discrepancy among species as well as among the same species by various authors. As the table shows the lethal concentrations of hydrogen sulfide to several species of fish found by Smith and his colleague (Smith and Oseid, 1970; Adelman and Smith, 1970, 1972) who used continuous flowing bioassay which enabled them to maintain constant concentrations of hydrogen sulfide throughout their experiments, are much lower than those found by other investigators in the past, who used a static bioassay method.

TABLE 16. LETHAL EFFECTS OF HYDROGEN SULFIDE ON FRESHWATER FISH

Concentration mg/l	Species	Test conditions	Survival time	References
0 018-0.020	Walleye (juvenile)	16-18 C, DO 6 mg/l	96-hr. TLm	Smith and Oseid (1970)
0.020-0.032	Bluegill sunfish	16-18 C, DO 6 mg/l	96-hr. TLm	Smith and Oseid (1970)
0.039-0.43	Fathead minnow (adult)	16-18 C, DO 6 mg/l	96-hr. TLm	Smith and Oseid (1970)
0.04-0.071	Goldfish	17-25 C, DO 1.5% to mean saturation, pH 2.7	96-hr. TLm	Adelman and Smith (1970)
0.086	Brook trout	--	Lethal	Belding (1929)
0.1	Trout	--	Harmful	Stroede (1933)
0.53	Goldfish	6.5 C, DO mean saturation, pH 7.8	96-hr. TLm	Adelman and Smith (1972)
0.53-0.8	Channel catfish	--	24-hr. TLm	Bonn and Follis (1967)
0.8-1.4	Channel catfish	25-30 C, pH 6.8-7.8	3-hr. TLm	Bonn and Follis (1967)
1.0	Trout	--	Lethal	Schaut (1939), Jones (1948), Dimick (1952)
1.0	Emerald and Steel-colored minnows	--	Minimum lethal concentration	Van Horn et al. (1944)
1.2	Silver salmon	15 C	Lethal	Dimick (1952)
1.38	Fathead minnow	--	48-hr. TLm	Black et al. (1957)

TABLE 16. LETHAL EFFECTS OF HYDROGEN SULFIDE ON FRESHWATER FISH (continued)

Concentration mg/l	Species	Test conditions	Survival time	References
1.4	Killies fish	DO 4% saturation	24-hr. TLm	Schaut (1939)
3.8	White sucker	--	Lethal	Belding (1929)
4.3	Goldfish	--	Lethal	Belding (1929)
4.9-5.3	Orange-spotted sunfish	--	1 hour	Shelford (1917)
5	Goldfish	--	200 minutes	Ellis (1937)
5-6	Carp	--	24 hours	Schaut (1939)
6.3	Carp	--	Lethal	Belding (1929)
8-12	Carp and tench	--	Harmful	Stroede (1933)
10	Goldfish	--	96-hours or less	Ellis (1937)
10	Trout	--	15 minutes	Weigelt et al. (1885)
100	Tench	--	3 hours	Weigelt et al. (1885)

Due to a short half life of hydrogen sulfide in water in the presence of oxygen, it is impossible to maintain a constant level of hydrogen sulfide in static bioassay and thus the amounts of hydrogen sulfide lethal to fish found by static bioassay were found to be very high. Schaut (1939) stated that the amount of hydrogen sulfide which affects fishes was so high, 5-6 mg/l, that a body of water containing it would produce such a disagreeable odor that no one would care to be in its vicinity for any length of time.

Bonn and Follis (1967) showed that small catfish were killed more quickly than large fish when exposed to hydrogen sulfide. Dandy (1967) found that fish transferred to freshwater recovered rapidly from the effects of sulfide.

Fish behavior. Shelford (1917) indicated that fish reacted positively to a weak concentration of hydrogen sulfide, which would produce death in a few days, but were negative to strong concentrations. Hoglund (1961) found that spent sulphite liquor containing hydrogen sulfide repelled fish at low non-toxic concentrations, but at high toxic concentrations the ability of the fish to avoid spent sulphite liquor diminished. Jones (1948) also found that sticklebacks reacted negatively to 0.001 N solution of sodium sulphide solution of which pH was 6.8 by addition of sulphuric acid (to produce H_2S) immediately. At 0.0008 N the reaction time was about 47 minutes. Over the concentration range tested, the reaction time was always shorter than the survival time. The swimming endurance of bluegill sunfish was reduced after chronic exposure to 0.004 mg/l hydrogen sulfide (Oseid and Smith, 1972).

Fish reproduction. Lethal levels of hydrogen sulfide occur near the soil-water interface where organic material is undergoing decomposition and where the early stage of many species of fish and aquatic food organisms must be passed in streams and ponds (Smith and Oseid, 1970, 1972). Smith and Kramer (1963) indicated that walleye eggs could not survive when they were placed in a river bottom where hydrogen sulfide was known to be evolved, but at the same location the eggs suspended 12 inches above the bottom survived normally. In the Rainy River, Minnesota, mortality of the walleye eggs at the stream bottom was 96 percent in six days at 0.58 mg/l hydrogen sulfide and 95 percent in 13 days at 0.27 mg/l (Colby and Smith, 1967). In laboratory studies, the concentrations of hydrogen-sulfide found to be lethal to fish eggs and fry respectively are shown in Table 17 and Table 18. In general, the sac fry of fish are more vulnerable to hydrogen sulfide than eggs (Colby and Smith, 1967; Smith and Oseid, 1970, 1962). Among trout, walleye, northern pike, and sucker, the eggs of the sucker were less tolerant to hydrogen sulfide than the other three species (Smith and Oseid, 1970).

The sac fry of walleye and northern pike hatched from eggs exposed to hydrogen sulfide were smaller in size than those in control. Also their sac fry subjected to hydrogen sulfide decreased their growth rate (Adelman and Smith 1970; Smith

TABLE 17. LETHAL EFFECTS OF HYDROGEN SULFIDE ON FRESHWATER FISH EGGS

Concentration mg/l	Species	Test condition	Survival Time	References
0.049	Rainbow trout	16-18 C, DO 6 mg/l	96-hr. TLm	Smith and Oseid (1970,1972)
0.055	Rainbow trout	16-18 C, DO 6 mg/l, pH 7.6-8.0	72-hr. TLm	Smith and Oseid (1970,1972)
0.022-0.036	Walleye	12-18 C, DO 3.1-6.1 mg/l	10- and 19-days TLm	Smith and Oseid (1970,1972)
0.052-0.087	Walleye	12-18 C, DO 3-6 mg/l pH 7.6-8.0	96-hr. TLm	Smith and Oseid (1970,1972)
0.075	Walleye	16-18 C, DO 6 mg/l	72-hr. TLm	Smith and Oseid (1970)
0.21-0.26	Walleye	pH 7.6-8.0	144-hr. TLm	Colby and Smith (1967)
0.42-0.46	Walleye	--	48-hr. TLm	Colby and Smith (1967)
0.34-0.52	Walleye	--	Mortality 98% in 6 days and 100% in 72-hrs.	Colby and Smith (1967)
0.025-0.032	Northern Pike	16-18 C, DO 2-6 mg/l, pH 7.6-8.0	9-days TLm	Smith and Oseid (1970)
0.034-0.38	Northern Pike	13-18 C, DO 2-6 mg/l, pH 7.3-8.0	96-hr. TLm	Smith and Oseid (1970,1972) Adelman and Smith (1970)
0.038-0.041	Northern Pike	16-18 C, DO 2-6 mg/l pH 7.3-8.0	72-hr. TLm	Smith and Oseid (1970) Adelman and Smith (1970)

TABLE 17. LETHAL EFFECTS OF HYDROGEN SULFIDE ON FRESHWATER FISH EGGS (continued)

Concentration mg/l	Species	Test conditions	Survival time	References
0.046-0.076	Northern Pike	DO 2-6 mg/., pH 7.3-8.0	48-hr. TLm	Adelman and Smith (1970)
0.181-0.411	Northern Pike	DO 2-6 mg/l, pH 7.3-8.0	24-hr. TLm	Adelman and Smith (1970)
0.018	Sucker	16-18 C, DO 6 mg/l, pH 7.0-8.0	12-days TLm	Smith and Oseid (1970)
0.028	Sucker	15-18 C, DO 6 mg/l	96-hr. TLm	Smith and Oseid (1970,1972)

TABLE 18. LETHAL EFFECTS OF HYDROGEN SULFIDE ON FRESHWATER FISH FRY.

Concentration mg/l	Species	Test conditions	Survival time	References
0.02	Rainbow trout	15-18 C, DO 6 mg/l	72-hr. TLm	Smith and Oseid (1970,1972)
0.007	Walleye	15 C, DO 6 mg/l	96-hr. TLm	Smith and Oseid (1970,1972)
0.017	Walleye	DO 6 mg/l	72-hr. TLm	Smith and Oseid (1970)
0.12-0.18	Walleye	--	24-hr. TLm	Colby and Smith (1967)
0.05	Walleye	DO 8 mg/l	96-hr. TLm	Colby and Smith (1967)
0.14	Walleye	--	48-hr. TLm	Colby and Smith (1967)
0.009-0.026	Northern Pike	DO 2-6 mg/l	96-hr. TLm	Smith and Oseid (1970), Adelman and Smith (1970)
0.012-0.030	Northern Pike	DO 2-6 mg/l	72-hr. TLm	Smith and Oseid (1970), Adelman and Smith (1970)
0.016-0.047	Northern Pike	DO 2-6 mg/l	48-hr. TLm	Adelman and Smith (1970)
0.035-0.160	Northern Pike	DO 2-6 mg/l	24-hr. TLm	Smith and Oseid (1970), Adelman and Smith (1970)
0.018	Sucker	DO 6 mg/l	120-hr. TLm	Smith and Oseid (1970)
0.017-0.026	Sucker	13-15 C, DO 3-6 mg/l	96-hr. TLm	Smith and Oseid (1970,1972)
0.024	Sucker	DO 6 mg/l	72-hr. TLm	Smith and Oseid (1970)
0.034	Sucker	DO 6 mg/l	24-hr. TLm	Smith and Oseid (1970)

and Oseid, 1972).

Northern pike eggs subjected to sublethal concentrations of hydrogen sulfide resulted in increasing percentages of sac fry with anatomical malformation. The types of malformation included lordosis, congestion, gelatinous lesions, and their various combinations (Adelman and Smith, 1970). For the walleye, malformation of the fry was one to 54 percent at the concentrations of hydrogen sulfide of 0.024 mg/l and 0.059 mg/l, and 16-20 percent at 0.025 mg/l and 0.062 mg/l. For the sucker, 63.5-50.8 percent deformities occurred at 0.023 mg/l and 0.049 mg/l. For the northern pike, 6.9-19 percent deformities occurred at 0.025 mg/l and 0.06 mg/l (Smith and Oseid, 1970).

Threshold concentration. The concentration of hydrogen sulfide tolerated without harm by juvenile salmon, coho salmon and cutthroat trout were found to be 0.3, 0.7, and 0.5 mg/l respectively (Haydu et al. 1952). For steel-colored and lake emeralds, the minimum lethal concentration for 120 hours was found to be 1.0 mg/l (Van Horn et al. 1949). In contrast to the above high threshold concentrations, Smith and Oseid (1972) suggested that the safe level of hydrogen sulfide for development of eggs and fry of walleye, sucker, trout, and northern pike is below 0.006 mg/l. Anon (1972) indicated after literature review that a level of un-ionized hydrogen sulfide assumed to be safe for all aquatic organisms including fish is 0.002 mg/l.

Estuarine and Marine Fishes

In contrast to freshwater streams and lakes where hydrogen sulfide may occur, but usually in small quantities at the bottom, hydrogen sulfide is a very important gas in salt lakes, especially those with a thermocline, and in the thermocline arms and estuaries of the sea. Marine organisms encounter hydrogen sulfide much more often and in greater degree than freshwater water organisms (Shelford, 1917). In the brackish water lake of Faro, Italy, the concentrations of hydrogen sulfide at 10 meters below the surface where the red water zone was located were 54.74 to 66.15 mg/l (Genoverse, 1964).

Despite this fact, there are almost no studies on the effects of hydrogen sulfide on marine and estuarine fishes In Carquinez Strait of California, 1-6 mg/l hydrogen sulfide was detected and suspected to be the most probable cause of the striped bass mortality in the strait (Silvey and Irwin, 1969). Also, in San Francisco Bay, changes in the species diversity index was found to be related to the levels of waste constituents in the sediments. High concentration of sulfide and a high index of putrescibility were associated with decreased benthic animal diversity (Storrs et al. 1969).

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SEWAGE SLUDGES

Freshwater Fish

Sewage sludges are the insoluble portions of the sewage treatment plant effluents. In receiving streams they are generally regarded as suspended and settleable solids (or simply as silts) like those originating from soil erosion, irrigation, dredging, mining, industrial wastes and other human activities. There is a vast accumulation of literature in the effects of suspended and settleable solids on freshwater organisms and fisheries. It has been reviewed recently by McKee and Wolf (1963), Cairns (1968), Alabaster (1972), Sherk (1971) and the European Inland Fisheries Advisory Commission (Anon, 1964). Recently there were a few new publications (Owens et al. 1970; Gammon, 1970; Ogunrambi and Dobbins, 1970; Garland and Hart, 1971). On the basis of information available up to now, the suspended and settleable solids affect fish by causing direct mortality through abrasive injuries and clogging gills, by blanketing the stream bottom to kill fish eggs, fry, and food organisms of fish, by destroying spawning ground, and by changing the fish behavior and reducing the feeding and growth of the fish. Indirectly, they cause turbidity of the water, screening out the light penetration and decreasing the primary productivity, promoting oxygen depletion and formation of toxic hydrogen sulfide, and developing noxious condition of the stream.

However, past studies of the effects of the so-called suspended and settleable solids on fish life mostly concern those from other sources rather than sludges from sewage treatment plant effluents. It is still a question whether there is a difference in the effects on fish life between sewage sludges and other suspended solids. To date there are very few field studies on the effects of sewage sludge on fish and virtually no laboratory studies.

Hubbs (1933) indicated that the accumulation of sludge in streams is one of the most baneful effects of untreated sewage. The septic bottom putrifies under anerobic conditions, producing substances toxic to fish and obnoxious to man. Liepolt (1963) reviewed the biological effects of sludges and suspended matter on fish and lower organisms. He described a case of severe fish mortality in the river March in 1962, following the discharge of suspended mineral matter in waste water from a sugar refinery plant, which caused severe proliferation and thickening of the gill lamellae, preventing oxygen uptake. However, Pescheck (1963) showed that mineral solids and fibers did not damage fish directly, but affected the environmental and nutritional conditions, causing fish mortality and fish migration. Klinke (1966) investigated the effects of putrifying sludges on the expec-

tancy of fish life in lakes and rivers. He indicated that the effect resulting from environment with mineral substances (phosphorous and nitrogen) in sludges was an excess growth of sewage fungi, which in turn increased the amount of sludge deposited in the water and reduced the food availability to fish. Tsai (1973) indicated that the turbidity of streams and fish may become accustomed to it. There is no correlation between species diversity indices and turbidity (up to 85 JTU) caused by the clay type suspended solids in natural streams. There are also no damaging effects of a continuous turbid condition caused by the suspended algae from sewage lagoons on fish. However, in streams receiving continuous sewage effluents and having a constant turbid condition due to the presence of sludge as suspended matter, there is a significant negative correlation between the species diversity indices and turbidity increments. The 50 and 25 percent reduction in the indices were found at turbidity increments caused by sludge at 20 JTU and 8 JTU respectively. It appears that there might be a difference in the effects on fish of different forms of suspended solids, clay, sewage sludges, and algae.

Estuarine and Marine Fish

The effects of suspended and settleable solids on estuaries and marine fish have been reviewed by Alabaster (1972) and Sherk (1971). However, for sewage sludge, very few field studies and virtually no laboratory study has been done. Buelow (1968) found the possible sludge drifting into the head of the Hudson Canyon might endanger the breeding areas of lobster and red crab. Also, clam beds off the Delaware Bay may be in line to receive sludge carried by prevailing currents. Anon (1965) and Domenowske and Matsuda (1969) studied the effects of sludge disposal near the submarine outfall on marine fauna in Puget Sound, Seattle. They found no measurable sludge deposits around the outfall, and the marine fauna in the immediate vicinity appeared normal. Both fish and zooplankton appeared undistrubed. Hume et al. (1962) studied the effects of the Hyperion outfall which discharges digested and screened sludge into Santa Monica Bay, California. The standing crop of plankton and benthic fauna are affected; a reduction in diversity and an increase in absolute numbers was not observed around the outfall, while maximum populations occur at some distance from the outfall. Grigg and Kiwala (1970) made the survey at the area below the Los Angeles sewer outfall at White Point. It was found that the number of species enumerated at each station is negatively correlated with the amount of fine grain organic-laden sediments present in the cores. Fish appear to be particularly affected. The reduction of benthic species is probably due to decreased settlement and survival of their larvae caused by fine grained sediments which cover the bottom. Since these benthic species make up the diet of many resident fishes, the numbers of resident

fishes in turn would be expected to decline. Mackay and Topping (1972) made the preliminary survey in the relatively confined water of the Firth of Clyde just south of the Isle of Bute, which has received sewage sludge from the City of Glasgow since 1904. They found that the discharge sludge becomes disturbed over a wide area, but little obvious harm to the environment has resulted and there was no evident deterioration in commercial fisheries in the area.

However, there is some indication of qualitative changes in the benthic fauna and of the build-up of organic materials and heavy metal residues on the sea bed. Bascom (1974) indicated that sewage sludge contains a high concentration of heavy metals which may be toxic, particularly when it combines with organic materials to create an environment poor in DO content. Few animals can live in that condition.

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DISSOLVED OXYGEN

Freshwater Fish

Dissolved oxygen in water. The availability of oxygen in water is one of the most critical requirements for aquatic life. The oxygen supply in water comes chiefly from two sources, diffusion from air and photosynthesis of aquatic plants. The air contains 21 percent oxygen, but its diffusion into water is slow, unless there is wind or water movement. Normally, the amount of oxygen saturated in water during the summer at 20 C is 9.2 mg/l. Water temperature and dissolved salts are two major factors affecting solubility of oxygen in water. The DO content increases when temperature and salinity decrease (Raymont, 1963).

Photosynthesis, the second major source of oxygen in water, is limited to the depth near the water surface of light penetration. The depth of a photosynthetic layer depends on water turbidity and light absorption. High intensity of photosynthetic activity may result in supersaturation of DO in water. As high as 30-32 mg/l of DO or 300 percent saturation was reported in lakes of heavy algal bloom, such as Lake Waubesa, Wisconsin (Woodbury, 1941), several ponds and lakes in Iowa (Wiebe, 1931), and Westlake Park, California (Anon, 1938).

The diurnal variation of DO in water is largely a result of temperature fluctuation and photosynthesis-respiration cycle of aquatic plants. This causes the photo-layer to have its maximum DO content usually during the afternoon and its minimum oxygen content at about dawn. The annual cycle in oxygen content is closely correlated with temperature, highest in winter and lowest in summer. Sewage effluents and other pollutants containing high concentrations of organic matters tend to eutrophy the water, causing a reduction in DO content by bacterial decomposition and/or by algal respiration at night. Also, supersaturation of DO may be caused by intensification of photosynthesis in eutrophicated water.

Modes of effects of DO deficiency and supersaturation. In water containing DO below the concentration of minimum tolerant level, fish usually show hypoxia and die of asphyxiation. Asphyxiation occurred in trout in water containing DO content of 2.5 mg/l or less. The fish might recover from asphyxiation in water containing DO as low as 2.65 mg/l. At 1.3 mg/l or less, all trout were asphyxiated (Gutsell, 1929). Bluegill sunfish and largemouth bass showed stress from changes in serum proteins under low oxygen content in water (3 mg/l for eight hours per day for nine days), but yellow bullheads were not affected. It was noted that low oxygen concentration adversely affected the digestion of food and accelerated clotting of the blood of the fish (Bouck and

and Ball, 1965). Following hypoxia from reduced DO level for 4.9 mg/l to 2.8 mg/l, the rainbow trout excreted abnormally high concentration of Na, K, Mg, Cl and inorganic phosphates (Hunn, 1969).

There was a parallelism between the oxygen tension of water and that in the tissues of trout, although the latter appears to lag by approximately four hours. The oxygen tension in the carp did not show any cyclic changes and their values were distinctly less than those for the trout (Garey and Rahn, 1970). Itazawa (1957) demonstrated that the oxygen content of arterial blood increased with oxygen tension up to about 80 mm of mercury and ranged from zero percent to 9.54 percent by volume. When the DO content of the arterial blood is less than two percent by volume, carp showed symptons of dyspnoea. This concentration of DO in the blood corresponded to the oxygen tension in the water of about 15 mm of mercury. The oxygen content in the arterial blood decreases in fish kept in still water, but it recovered in running water after a short period of time. Later, Itazawa (1959) found that the maximum concentration of oxygen reached in the fish blood decreased in order of rainbow trout, carp, and Japanese eel. This order was positively correlated with the activity of the fishes. The critical tension of oxygen in water required to maintain the minimum concentration of oxygen in the fish blood also varied with the species of fish, and appeared to be negatively related to the resistivity of the fish to oxygen deficiency. Complete deoxygenation of arterial blood resulted in the failure of oxygen consumption in the brown bullhead at low levels of ambient oxygen. The arterial deoxygenation apparently resulted from inability of the blood to load oxygen across the diffusion barrier of gills, since the gill ventilation and perfusion continued after oxygen uptake ceased. Thus the oxygen affinity of the blood was strongly implicated as a factor limiting oxygen uptake at low level of ambient oxygen (Grigg, 1969). The fish living in a healthy condition at extremely low DO concentration showed a physiological compensation through a high red blood cell count (Rounsefell and Everhart, 1953). The blood sugar values of carp were influenced by low DO concentration. Under condition of asphyxia for several hours, the blood sugar level in some fish increased twice the normal level (Muennich, 1958). Trout reared in water containing less than 8 mg/l DO were more susceptible to disease than those in high DO concentration (Hewitt, 1938).

Tench responded to hypoxic condition by increasing breathing rate and ventilation volumes. Their arterial partial pressure of oxygen and carbon dioxide decreased, but pH tended to remain steady. There was also a significant increase in blood lactate. The reasons enabling the tench to withstand severe hypoxic conditions were attributed to blood of high oxygen affinity and the ability to maintain a favorable acid-base status in the blood for oxygen transport (Eddy, 1974). Hughes (1973) discussed the conditions

under which fish cells suffer a lack of oxygen. From a consideration of the transfer of oxygen from the water to the cellular site as a series of resistances, it was suggested that hypoxia can result from an increase in resistance anywhere along the chain. Detailed discussion was given in relation of hypoxia to interference with gas transfer at the gill surface, reduction in the blood oxygen carrying capacity, modifications in the cardiac and ventillatory frequencies and the time course of the toxic stimulus.

Fish kills in the Westlake Park of Los Angeles, California caused by 300 percent supersaturation of DO in the water was due to bubbles of oxygen in the gills (Anon, 1938). Characteristic lesions of the dead fish by superconcentration of DO in Lake Waubesa, Wisconsin, consisted primarily of gas emboli in the gill capillaries and gas bubbles in the subcutaneous tissues. Death of the fish was attributed to the blocking of the blood circulation through the gills, resulting in respiratory failure (Woodbury, 1941). Lassleben (1951) found that maxillary disease and dropsy occurred in carp in water containing DO of 150 percent saturation. There was a connection between dropsy and damage to the fins, especially of the tail fin, by gas bubbles. Alikunhi et al. (1954) demonstrated that a mixed collection of carp fry reared in jars with supersaturated water developed bubbles of gas on the lower lip and fins, and later in the stomach. Death occurred due to distension of the stomach, and subsequent choking as the fish were buoyed up to the surface. Muennich (1958) found that the blood sugar level in carp increased in water containing a considerably supersaturated dissolved oxygen.

Environmental factors affecting the effects of DO deficiency. Several environmental factors, such as temperature, carbon dioxide, and pH are known to affect significantly the effects of DO deficiency on fish (Creaser, 1930).

I. Temperature. The minimum tolerant limit of DO to fish usually increases with increasing water temperature. Asphyxial point for trout increased from 1.14 mg/l DO at 6.5 C to 3.4 mg/l at 25 C. For goldfish it was 0.56 mg/l at 11 C and 0.6 mg/l at 27 C (Gardner and King, 1922). The bluegill sunfish of four to six grams survived the sudden change in DO from saturation to 1.5 mg/l at 25 C, but to 1.9 mg/l at 35 C (Anon, 1958). Increase in temperature from 10 C to 20 C reduced the resistance of rainbow trout, perch, roach and carp to lack of DO (Downing and Merkens, 1957). In the experiments on the resistance of trout and several species of coarse fish to low DO, it was found that the higher the temperature, the higher DO concentration is necessary for fish survival (Anon, 1957). Largemouth bass (50 to 75 grams) withstood DO concentration of 1.2 mg/l to 1.5 mg/l for 24 hours at 35 C, while the small fish (10 grams) withstood a concentration as low as 0.92 mg/l for 25 hours at 25 C. Channel catfish (75 to 100 mg/l) were able to survive in 1.65 mg/l DO at 25 C, but in 1.8 mg/l

DO at 35 C (Anon, 1960). FOr 1.5 to 7 month old sturgeon, the critical oxygen level (the concentration below which oxygen uptake of the fish decreases) varied from 1.8 mg/l at 11 C to 6.0 at 20 C (Lozinov, 1952). Optimal DO concentration for trout is 10 mg/l to 11.43 mg/l, but the fish show some discomfort at the DO of 7.86 mg/l in warm water (Plehn, 1924). Burdick et al. (1957) indicated that lethal DO concentration for yellow perch increased from 0.5 mg/l at 12.2 C to 0.76 mg/l at 21.1 C. Blazka (1958) found that at 5 C Crucian carp tolerated for two months at low DO, which they could withstand for only a few hours at 16 C. In field studies on the oxygen requirements of several species of freshwater fish, Moore (1942) showed that, in general, oxygenation of less than 3.5 mg/l at temperature of 15-20 C (summer) were fatal to most of eight species tested within 24 hours, while oxygen tension of less than 2.0 mg/l at temperature of 0.4 C (winter time) were fatal to the fish within 48 hours.

Davison (1954) found that the minimum DO content tolerated by juvenile silver salmon was not affected by increasing the temperature from 12 C to 16 C. However, the fish increased their minimum tolerant limit of DO from 1.4 mg/l to 1.7 mg/l, when the temperature increased from 18 C to 22 C. Davison et al. (1959) suggested that critical oxygen concentration for the salmon may increase by only about ten percent with a rise of temperature from 12 to 20 C, and may be almost independent of temperature, when temperatures are below 16 C. However, there is a marked increase in the critical DO requirement for the fish at high temperature, particularly above 22 C.

II. Carbon dioxide. Carbon dioxide plays an important role in vital processes of organisms, such as photosynthesis, respiration, and decomposition in the aquatic system. The amount of carbon dioxide present in water greatly influences the effects of low DO on fish (McKee and Wolf, 1963; Black et al. 1954).

Berg (1969) stated that the low estimates of low oxygen tolerance of yellow perch may be due to the presence of low carbon dioxide. When whitefish were exposed to water of a pH of 6.3, obtained by adding carbon dioxide directly, the fish died earlier in the acid water than in the alkaline water with a low DO content. A high DO content antogonizes the CO_2 present, prolonging the life of the fish (Hall, 1925). The concentration of carbon dioxide could increase the minimum concentration of DO necessary for the survival of rainbow trout for 24 hours by more than 100 percent. In water containing more than 59 mg/l of carbon dioxide, perch were more resistant than rainbow trout to low lethal concentrations of DO, but less so in higher lethal concentrations (Alabaster et al. 1957). If the DO concentration was less than 30 percent of the air saturation value, carbon dioxide in concentrations between 15 mg/l and 66 mg/l was lethal to trout (Anon, 1955a). High concentration of carbon dioxide

caused pronounced increase in the minimum DO requirement of coho salmon. There was also found a noticeable increase in the minimum DO requirement of salmon at free carbon dioxide concentration between 59 mg/l and 100 mg/l, depending on the period of acclimation and the values of bicarbonate alkalinity of the water (Anon, 1959). Irving et al. (1941) showed that carbon dioxide decreased the affinity of the fish hemoglobin to oxygen. Increasing the temperature decreased the degree of oxygen saturation of the hemoglobin in the presence of carbon dioxide.

On the other hand, McNeil (1956) found that the resistance of DO deficiency of resting coho salmon was not markedly reduced at carbon dioxide concentrations below 50 mg/l. Wilding (1939) also found that the concentration of carbon dioxide of 13 mg/l had no effect on the asphyxial oxygen concentration of yellow perch and two species of cyprinid fishes.

III. pH value. There was correlation between the utilization of oxygen by fish at low oxygen tension and the pH value and carbonate system of the water (Power, 1928). Wells (1913) indicated that low oxygen (0.1 mg/l) in alkaline water causes death of fish sooner than in acid water. A specific range of optimal pH values for bluegill sunfish to tolerate low DO was between 7.0 to 8.5. However, there was little difference in the lethal DO concentration for rainbow trout in pH values between 5.2 and 6.5. At pH of 6.0 the least tolerable concentration of DO for bluegill sunfish was 7.5 mg/l (Wiebe et a. 1934). Salmon in a low pH water containing no initial free carbon dioxide increased their lethal DO concentration at pH values less than 4.45, while for the bluegill sunfish their concentration markedly increased at pH values less than 4.0 (Anon, 1959).

Mookerjee and Bhattacharya (1949) found that in the tank where microcystis was abundant, there was an increase in the mortality of young carps, when photosynthetic activity was greatest, DO content was high, carbonates were formed, and pH values increased to 9.55 at noon. They concluded that young carp cannot tolerate high alkalinity accompanied by high concentration of organic matter, even when the content of DO is appreciably higher than normal. In contrast, Creaser (1930) found that variation of the hydrogen concentration over the range of pH 4.1 to 9.5 seems to have little or no influence upon the temperature or DO limits of the brook trout.

Lethal effects. Fish mortalities resulting from the dramatic reduction of dissolved oxygen in water have been reported frequently by many investigators (Olson, 1932; Costin, 1954; Abeliovich, 1967; Saleem, 1962). The oxygen concentrations lethal to fish are shown in Table 19. The salmonid fishes were generally more susceptible to oxygen deficiency than warm water fishes (Downing and Merkens, 1957; Anon, 1957; Plehn, 1924).

With regard to different susceptibilities to lethal oxygen

TABLE 19. LETHAL EFFECTS OF DISSOLVED OXYGEN DEFICIENCY ON FRESHWATER FISHES

Concentration mg/l	Fish	Temperature C	Survival time	References
0.2	Trout	-	1 minute	March (1908)
0.3	Golden shiner, black bullhead	4	48 hours	Moore (1942)
0.25-0.50	3 spined stickleback	20	Lethal	Pruthi (1927)
0.3	Gambusia, Mollienesia	Spring	Lethal	Odum and Caldwell (1955)
0.528-0.545	Yellow perch	12	Lethal	Burdick et al. (1957)
0.56-0.6	Goldfish	11 and 27	Lethal	Gardner and King (1922)
0.57-0.71	Roach	-	Lethal	Kupzis (1901)
0.626-0.78	Yellow perch	15.5-21	Lethal	Burdick et al. (1957)
0.8	Bluegill	4	48 hours	Moore (1942)
0.9	Banded killifish, pumpkinseed	4	48 hours	Moore (1942)
1.0	Carp	--	1 day	Basu (1949)
1.0	Roach	--	7 days	Jenkins (1965)
1.0-1.3	Long-fin dace, gila sucker, speckled dace	23-30	8.5-18-hr. LD_{50}	Lowe et al. (1967)
1.1-1.6	Walleye	24	160-250 minutes	Hoff and Chittenden (1969)

TABLE 19. LETHAL EFFECTS OF DISSOLVED OXYGEN DEFICIENCY ON FRESHWATER FISHES (continued)

Concentration mg/l	Fish	Temperature C	Survival time	References
1.14	Trout	6.5	Lethal	Gardner and King (1922)
1.3-2.5	Trout	9	Lethal	Gutsell (1929)
1.4	Black crappie, orangespotted sunfish	4	48 hours	Moore (1942)
1.5	Green sunfish, yellow perch	4	48 hours	Moore (1942)
2.0	Trout	--	Few hours	Lloyd (1964)
2.0	Rainbow trout	15-17	Lethal	Southgqte (1933)
2.0	Young American shad	21-23	Lethal	Tagatz (1961)
2.2	Salmon	8	8 days	Lindroth (1949)
2.3	Rock bass, northern pike, largemouth bass	4	48 hours	Moore (1942)
2.3-2.7	Trout	--	7 days	Jenkins (1965)
2.5-3.0	Salmon	--	Lethal	Chapman (1940)
2.9	Trout	--	Lethal	Paton (1904)
3.0	Black bullhead	22 and 15	24 hours	Moore (1942)
3.1	Pumpkinseed, largemouth bass, northern pike, yellow perch, bluegill	25	Lethal	Ellis (1937)
3.4	Trout	25	Lethal	Gardner and King (1922)

TABLE 19. LETHAL EFFECTS OF DISSOLVED OXYGEN DEFICIENCY ON FRESHWATER FISHES (continued)

Concentra-tiong mg/l	Fish	Temperature C	Survival time	References
4.3	Black crappie, freshwater drum	26	24 hours	Moore (1942)
4.5	Walleyes fingerlings		--	Smith and Kramer (1965)
5.0	Walleye, channel catfish, bluegills	winter	Lethal	Moyle and Clothier (1959)

concentrations, Moore (1942) showed that the black bullhead appeared to be the hardiest one of the eight species tested in the field under summer condition, while the pumpkinseed and possibly the black crappie were the least tolerant. Under winter condition, the oxygen concentration of 1.0 mg/l or less was found to be fatal for all species tested, except some black bullhead survived in this low DO concentration. In the study of Prairie Lake, two walleyes and perhaps channel catfish and bluegill sunfish did not survive in winter when DO levels reached below 5 mg/l, while northern pike, crappies, and some rough fish were more tolerant and evidently survived in DO levels near 1 mg/l (Moyle and Clothier, 1959). Mass mortalities of fish occurred frequently in summer at night in eutrophicated ponds because of oxygen deficiency (Abeliovitch, 1967; Olson, 1932; Lovett and Brown, 1949).

In the open and closed systems for studying the tolerance and potential natural selection of four native Arizona freshwater fishes subjected to low DO concentrations, Lowe et al. (1967) found that there were different rankings of species. In the closed air systems the degree of tolerance to low DO by the four species was in order of desert pupfish, gila sucker, longfin dace and speckled dace. The order of survival of the four species in the open air system was desert pupfish, longfin dace, speckled dace and gila sucker. The difference in their survival in the open air system appeared to be related to the metabolic capacity and scope of the fishes (Fry, 1947; Brett, 1966), as well as to their tolerance to low oxygen level (Lowe et al. 1967). Small fish were less tolerant to low oxygen tension than large fish of the same species (Moore, 1942).

Through acclimation to low DO, fish may become more resistant to DO deficiency. The minimum tolerance level of DO for young speckled trout was 1.75 mg/l at 9 to 10 C. After 20 to 33 hours of acclimation to low DO, the fish could survive at the DO level of 1.05 mg/l (Shepard, 1955). Prosser et al. (1957) found that the point where goldfish switched from oxygen-independent metabolism to oxygen-dependent metabolism was at 2.17 mg/l DO for non-acclimated fish and 1.05 mg/l for acclimated fish. In the investigation of DO requirements of bluegill sunfish, largemouth bass, and channel catfish at 25, 30, and 35 C, Moss and Scott (1961) indicated that the critical level of DO for each species was lower for the acclimated fish than non-acclimated fish at any given temperature.

Mass fish mortality caused by supersaturation of DO resulting from algal bloom was reported in Westlake, California (Anon, 1938), Lake Waubesa, Wisconsin (Woodbury, 1941), fish ponds at Fairport, Iowa (Wiebe, 1931) and Roundhay Park, Leeds (Lovett and Brown, 1949). The finerling largemouth bass tolerated DO concentrations ranging from 29 to 40 mg/l for 23 hours at 17 C (Wiebe, 1933) and various centrachids, salmonids, and cyprinids

withstood prolonged exposure to 20 to 34 mg/l at 14.5 to 17 C (Wiebe and McGavock, 1932).

Fish behavior. The ability of fishes to avoid low dissolved oxygen concentrations has been reported through field observations (Allen et al. 1958; Alabaster, 1959; Alabaster and Robertson, 1961) and laboratory studies (Jones, 1952; Whitmore et al. 1960; Höglund, 1961; Stott and Cross, 1973; Hill et al. 1973). Shelford and Allee (1913) first indicated that fish reacted negatively to a deficiency of DO by moving away, rising to the surface, and gulping. Whitmore et al. (1960) found juvenile chinook salmon showed marked avoidance of DO concentrations below 4.5 mg/l at summer temperatures and they decreased in the avoidance reaction at low temperatures. At summer temperatures, juvenile coho salmon avoided dissolved oxygen concentration as high as 6 mg/l. Bishai (1962) indicated that the response of salmonids to poorly oxygenated water depended on their ages. Yolk sac fray were unable to avoid water of low DO content but to the same concentration the feeding fry showed a marked negative reaction. Some responses took place by trial, or choice, not by random swimming. The fish seemed to improve avoidance with the passage of time, suggesting that learning or conditioning must have taken place. Jones (1952) found that at 13 C stickleback showed no hesitation in entering water containing less than 3 mg/l, but if they remained in this water, respiration became difficult and they began to swim rapidly and returned to water of a normal DO content. The fish generally took one to three minutes to leave the poorly oxygenated water. At 20 C, and with concentrations of DO less than 2 mg/l, the response of fish to DO deficiency was so rapid that the fish usually turned away from the poorly oxygenated water. Similar results were obtained with minnow and trout fry.

Juvenile largemouth bass and bluegill sunfish showed avoidance to DO concentrations near or below 1.5 mg/l. The prompt avoidance by some fish to DO levels below those known to be lethal to fish cannot be attributed entirely to the stimulation or increase in activity as the result of DO deficiency (Whitmore et al. 1960). Roach could avoid low DO concentration by taking appropriate action and the reduction in DO level in water was apparently a distinct stimulus for their avoidance (Stott and Cross, 1973). Cave fish, Chologaster agassizi, avoided light more pronounced than they did low oxygen condition. The fish seemed to be able to sense an unfavorable condition of the water and to search for a more desirable area (Hill, 1968).

Fish in water containing low DO may reduce their activity. In a warm spring, Hubbs et al. (1967) and Hubbs and Hettler (1964) found that a low DO level resulted in reduction of fish activity. Such reduction in activity could be at a site where DO level was limited, while nearby populations were active in localities that had more DO. Apparently, at 32 C fish activity was adversely affected by DO concen-

trations lower than 2 mg/l. Reuction in DO concentration decreases swimming performance of juvenile coho and chinook salmon (Davis et al. 1963). The same phenomenon was found in goldfish. This reduction in the swimming activity of the fish in water containing low DO was not due to fatigue, but to an oxygen-sensing mechanism of the fish, since the fish began to swim steadily immediately when DO concentration was increased (Kutty, 1968). Swimming ability of largemouth bass at low DO shows a marked seasonal change. During September and December the swimming performance of the fish was reduced perhaps due to seasonal changes in fish activity associated with falling water temperature (Katz et al. 1959). Sams and Conover (1969) observed that no salmon passed over Willamette Falls, Oregon, until DO level rose above 5 mg/l. The sustained swimming speed of juvenile largemouth bass and coho salmon was markedly reduced at DO concentrations below 5 or 6 mg/l at 25 C (Dahlburg et al. 1968). Such recovery of activity of the fish was also reported for salmon by Townsend et al. (1938) and Dandy (1967). In contrast to the above findings, Whitworth and Irwin (1964) indicated that most fish can remain active at 2 mg/l DO at 21.1 - 26.7 C. Juvenile chinook and coho salmon were able to swim continuously against the current of 0 8 ft./sec. for a day at a DO concentration of 2 mg/l, only slightly higher than the concentration necessary for quiescent fish to survive for the same period of time.

Fish showed "gasping reaction" when DO was less than 1 mg/l (Townsend et al. 1938). Japanese dace came up to the water surface snapping air when DO content was less than 0.9 mg/l (Shirahata, 1963). In low DO water, fish increased ventilation for a given rate of oxygen consumption and increased locomotory activity (Fry, 1969). The young salmonid fish show "yawning" and "coughing," increase in gill ventilation and in activity, to sudden change in DO content (Höglund and Hördig, 1969). Petrosky and Magnuson (1973) simulated winterkill condition and found that gill ventilation rate of bluegill sunfish, yellow perch, and northern pike increased in response to lower DO. Maximum ventilation rate occurred at 0.5 mg/l DO for northern pike and yellow perch, and 1.0 mg/l for bluegill sunfish. The movement of fish upward to the surface of water containing little or no DO is called "nosing." This is a part of the vertical response of the fish in adaptation to utilize a thin layer of oxygenated water present at the interface with the atmosphere (Lewis, 1970). Hyperbiomedical ventilation and random agitated swimming are characteristic behavior patterns of the eel in poorly oxygenated water. This behavior presumably is attributed to the fish sensing the unfavorable nature of the water and searching for a more desirable area. As the fish penetrates a high DO area, rest and recovery form its behaviorial pattern (Hill, 1969).

Fish growth. The growth rate of juvenile coho salmon kept at 20 C on a diet of marine amphipods decreased slightly with reduction of DO from 8.3 mg/l to 5 mg/l, and then, decreased sharply with further reduction in DO concentration. Food conversion rate was slightly depressed at DO near 4 mg/l.

At this concentration many fish died and those that survived consumed little food and lost weight (Hermann, et al. 1962). Young Salmo salar grew fast in DO concentrations higher than 10 mg/l, but old fish required only 6 mg/l (Nikiforov, 1952). However, the growth rate of Windermere char appeared to be not affected by oxygen concentrations over the ranges to 50 to 200 percent of the air saturation value (Swift, 1964). There was no difference in the growth rate of brown trout at oxygen concentrations of 50 percent of air saturation or higher (Swift, 1963).

For the warm water fishes, the growth and food consumption rates of juvenile largemouth bass increased as DO concentration increased to near saturation level, but its gross food conversion efficiency was considerably reduced only at concentrations well below 4 mg/l (Stewart et al. 1967). Young sturgeon could live and grow at DO concentrations below 5 mg/l at 18 C, but a DO concentration of that amount and above were required for their optimal growth (Lozinov, 1956). For channel catfish low DO level of 3 mg/l caused seeming retardation of the growth, although they signaled their stress by surfacing when DO values were less than 1 mg/l (Simco and Cross, 1966). In the experiment with young carps reared in water containing various levels of DO concentrations at 20-23 C, Chiba (1966) found that growth, feeding, food conversion, and oxygen consumption rates of the fish decreased in DO concentrations less than 3 mg/l. In DO concentrations about 3 mg/l the rates increased slightly or remained constant with the increase in DO concentrations. Adelman and Smith (1970) also found that growth and food consumption rates of juvenile northern pike decreased gradually as the DO levels were reduced to 3 mg/l, and were followed by a sharp decrease in the rates as the DO levels were further reduced. The growth of the fathead minnow fry was reduced significantly at all test concentrations of DO below 7.9 mg/l (Brungs, 1971). Growth rate and food consumption of channel catfish were drastically reduced in water containing 36 percent oxygen saturation as compared to those at 100 and 60 percent saturation (Andrews et al. 1973). However, the weights of Idus idus reared at the three levels of DO concentrations were not significant, but the length of the fish clearly increased with the increase in oxygen tension in water (Florez, 1972).

Diurnal fluctuation of DO restricted the growth and development of the salmonid fishes (Fisher, 1963). Brook trout were unable to tolerate DO fluctuation to 2 mg/l. There was a difference in growth rate of the fish in waters containing constant DO levels and fluctuating oxygen levels (Whitworth, 1968). However, Dorfman and Whitmore (1969) indicated that fluctuation of DO concentrations between 6.5 mg/l and 10.7 mg/l probably did not affect the growth of brook trout. Doudoroff and Warren (1965) pointed out that growth rate of fish subjected to fluctuating oxygen concentrations may be largely dependent on the DO minima occurring at night or early in the morning.

Fish reproduction. There was a possible correlation between the survival rate of coho salmon and steelhead trout embryos and the mean DO concentrations on the beds of two Oregon coastal streams. The DO requirement for the survival of the salmonid embryos might exceed 8 mg/l (Phillips and Campbell, 1962).

Steelhead trout and chinook salmon embryos held at 9.5 and 11 C died at a DO concentration of 1.6 mg/l. At 2.5 mg/l large numbers of embryos survived as the result of a reduction in respiration rate and, consequently, the rates of growth and development were reduced. Sac fry reared at low and intermediate oxygen concentrations were smaller and weaker than those from embryos reared at the high DO concentration (Silver et al. 1963). For the lake trout embryos the low DO levels (2.5-4.5 mg/l) retarded the rate of their development, resulting in delayed hatching, reduction of vitelline circulation, and abnormalities of head and trunk. The low oxygen levels at 10 C caused total mortalities just before hatching (Garside, 1959). Coho salmon embryos exposed to hypoxial stress (5-3 mg/l) were smaller at the time of hatching and emergence and underwent higher mortality during the development than the embryos and fry of the same parents exposed to 11 mg/l DO. They were prone to emigrate. Competition for food and space, and the effect of size-related order put smaller individuals at a disadvantage (Mason, 1969).

Water velocities through stream bed gravels in which salmonid eggs are deposited must be strong enough to transport sufficient exygen to all embryos and the surface of the chorion enveloping the individual embryo (Doudoroff and Warren, 1965; Silver et al. 1963). Wickett (1954) found a high percentage of salmon eggs dead in certain heavily-silted areas of the Nile Creek, Vancouver Island, which may be explained on the basis of a very low concentration of DO (0.2 mg/l) or very low velocity of water on the gravel (2mm/hr.) Coble (1961) found that in two streams in Oregon, there was a correlation between percentage survival of embryos of steelhead trout and the DO concentration and water velocity, but there was no relationship to gravel permeability. Also, reduction in either DO concentration or water velocity increased the length of the egg incubation period and reduced the size of the newly hatched fry in steelhead trout, chinook salmon, and coho salmon and other species (Silver, 1960; Silver et al. 1963; Shumway et al. 1964; Doudoroff and Warren, 1965). Shumway (1960) indicated that the relative efficiencies of yolk conversion to embryonic tissue in coho salmon were impaired at decreased levels of DO and water velocities. However, there was insufficient evidence that it is also the case in sac fry under the same conditions.

Devilless and Rosenberg (1953) found that the oxygen requirement for embryonic development of Salmo irideus was low during the first stage of segmentation, but it increased rapidly in the beginning of gastulation, In water deficient in oxygen, the second stage of development did not take place. Reduction in the oxygen concentration in water hindered the development

of rainbow trout fry (Mechanik, 1957). The Pacific salmon embryos at various developmental stages tolerated exposure of limited duration to DO levels well below 2 mg/l. In early stages of the development, DO levels of 0.3 mg/l or less might result in monstrosities. The low oxygen concentration stimulated the eggs in an advanced stage of development to hatch prematurely. Critical levels of DO were found to range from about 1 mg/l in early stages to over 7 mg/l shortly before hatching (Alderdice et al. 1958).

Similar effects of DO deficiency on embryonic development were found in warm water species. DO near the bottom muds of lakes and sluggish rivers may approach zero, and under such conditions, the hatching of fish eggs has been delayed, or the fish hatched from such eggs have been deformed (McKee and Wolf, 1963). Walleye eggs and sac fry in a stream over a bottom of wood fiber sludge deposits were killed when DO concentration dropped below 3 mg/l (Colby and Smith, 1967). Walleye eggs incubated at a DO concentration of 1 mg/l at 10 C were poorly developed upon hatching (Van Horn and Balch, 1956). Oseid and Smith (1971a) incubated walleye eggs at different oxygen levels and found that the hatching period of the eggs was substantially longer and body length of fry was shorter at low DO concentrations. They suggested that if a short incubation period and large fry are considered to be advantageous for a large year class in a natural system, the minimum oxygen level for the incubation of walleye eggs appeared to be at 5 to 6 mg/l. A similar case was found for white sucker at low DO concentration (Oseid and Smith, 1971b). Siefert et al. (1973) incubated northern pike eggs at 15 and 19 C and at water flows of 3.3 and 1.6 cm/min. It was found that 50 percent oxygen saturation was sufficient for survival and development of northern pike eggs from fertilization until larval stage. However, oxygen tension of about 33 percent saturation appeared to be inadequate for the fry survival. Studies on the embryonic development of sturgeon (Acipenser guldenstadti) show that larvae hatched at low oxygen concentrations developed more slowly and gained weight less rapidly than the controls. Lack of oxygen caused the morphological peculiarities in fish. The minimum concentration of DO for normal development of sturgeon eggs was suggested to be 5.5 mg/l mg/l (Yourovitskii, 1964).

In continuous exposure of fathead minnows at various concentrations of DO for 11 months, Brungs (1971) found that the number of eggs produced by each female was reduced at 2 mg/l of DO and spawning ceased at 1 mg/l. The incubation period of eggs increased successively at lower oxygen concentrations as much as 50 percent. The fry survival rate was reduced at 4 mg/l; 18 percent of the survivors were deformed.

Respiration and oxygen consumption. Active respiration of fish is often dependent on the DO concentration in the water. The rate of active respiration of lake trout becomes dependent on the oxygen pressure at approximately two-thirds of the air saturation (Gibson and Fry, 1954). The rate of oxygen consumption of active brook trout becomes dependent on oxygen

concentration at levels near or even well above the air saturation (Job, 1955; Basu, 1959). A 75 percent air saturation of oxygen in water would affect the activity of the speckled trout above 20 C as measured by cruising speed, and fully saturated water was required to maintain full activity (Graham, 1949). The partial pressure of ambient DO and aerial respiration are negatively related in young spotted gar (Hill et al. 1972). However, the oxygen consumption of the largemouth bass fingerling appears to be independent of oxygen tension at least within the range of the tension between 4.4 ml/l to 10.6 ml/l (Wiebe and Fuller, 1933).

The effects of low DO on respiration have been studied for various species of fishes (Podubsky, 1958; Randall and Shelton, 1963; Holeton and Randall, 1967, Randall and Smith, 1967; Beamish, 1964a,b,c,d; Serfaty and Peyraud, 1965; Garey, 1967, Ruff and Zippel, 1968). The trout and tench responded to decreasing oxygen tension in water by increasing the breathing rate and its amplitude and ventilation volume. These changes were associated with a marked bradycardia (Holeton and Randall, 1967; Randall and Shelton, 1963; Randall and Smith, 1967). Carp showed respiratory difficulty, when the DO was reduced to 4.3 mg/l or lower (Plehn, 1924). Respiratory activity increased gradually until the oxygen concentration fell to a critical level, where a rapid increase in respiration occurred (Serfaty and Peyraud, 1965). At low partial pressure of DO, the standard oxygen consumption rate of eastern brook trout increased, probably due to an increase in the respiratory volume, and it reached the maximum rate which was less than the active oxygen consumption rate of the fish. Further reduction in the partial pressure of DO, the oxygen consumption started to decline, suggesting that anerobic respiration might occur (Beamish, 1964c). During hypoxic conditions (4 mg/l-0.6 mg/l) the juvenile catfish increased ventilation, primarily as a result of increasing opercular stroke volume associated with an increase in gill efficiency. It is suggested that a part of the energy used by the fish in increasing the ventilation volume may come from anerobic oxidation (Gerald and Cech, 1970).

The life of Crucian carp can be sustained at low DO concentrations largely or entirely by anaerobic metabolism at low temperatures. The lowest DO concentration which did not decrease the oxygen consumption of the fish was 1.7 mg/l at 5 C (Blazka, 1958). The resting largemouth bass, bluegill sunfish, and channel catfish held in a respirometer at 25 to 35 C, in which DO concentration was gradually reduced, showed no reduction in their oxygen consumption until a lethal DO concentration of the fish was reached (Moss and Scott, 1961). There is a rhythmic fluctuation of DO consumption in species inhabiting quiet water (bullhead, sucker, golden shiner, and white crappie), but not in the species living in rapid flowing water (stone roller, common shiner, and greensided darter) (Claussen, 1936). For young sturgeons, the

critical oxygen level (the concentration below which oxygen uptake decreases) varies from 1.8 mg/l to 6 mg/l at 20 C. Carp resting in continuously renewing water exhibiting a rhythmic alteration in the periods of respiratory activity and of respiratory cessation. This periodic change in respiration disappeared when the flow of water with a sufficient amount of available DO was induced, indicating that DO concentration in water is an essential regulating agent for carp respiration (Peyraud and Serfaty, 1964). Goldfish in water containing DO pressure in a range between 140 Torr and 200 Torr showed no change in their oxygen consumption. However, in the oxygen pressure above or below the range, their oxygen consumption increased (Ruff and Zippel, 1968).

There are several factors influencing the oxygen consumption of fish. Oxygen consumption of many aquatic animals almost doubles with each rise of 10 C within their physiological limits (Ruttner, 1926). Oxygen consumption increased with increases in temperature in Etroplus maculatus (Parvatheswararao, 1965) and in trout and salmon (Shaw, 1946). The standard oxygen consumption rate of fish increases linearily on a semi-logarithmic grid over the temperature range of 10 to 35 C as shown in goldfish (Beamish and Mookherjil, 1964). Goldfish increase their oxygen consumption with increased swimming activity (Smith, 1965). Oxygen uptake of carp increased by uptake of food (Krayukhin, 1962). The channel catfish showed a distinctly higher rate of oxygen consumption in the afternoon than in the morning (Higginbotham, 1947). Standard oxygen consumption of brook trout and white sucker decreased rapidly during the firs two to three days of starvation (Beamish, 1964b). The male brook trout has the highest standard oxygen consumption in the late autumn and lowest in spring (Beamish, 1964a). Absolute oxygen consumption of stellate sturgeon increased with their ages (Konovalov, 1963).

The ability of fish to utilize DO in the presence of carbon dioxide varies from species to species (Fry and Black, 1938). For speckled trout, brown bullhead, carp and goldfish, there is a linear relation between the logarithm of the rate of oxygen consumption and the concentration of carbon dioxide for each level of oxygen concentration. The oxygen concentration determines the active level of oxygen consumption in the absence of carbon dioxide, except when DO approaches the low lethal level. The ability of the blood of the fishes to take up oxygen in the presence of carbon dioxide shows no direct relation to the activity of the fish to transport oxygen from the external medium under activity (Basu, 1959). There is no significant effect of carbon dioxide concentration on standard oxygen consumption at a constant DO partial pressure for brook trout at 10 C and carp at 25 C (Beamish, 1964c).

The active oxygen uptake rate of brook trout is influenced by acclimation of the fish in low oxygen concentration (Shepard, 1955). At the high partial pressures of DO, acclimation of the fish to low DO had little effect on the oxygen uptake rate

of the fish, but at low pressure the standard rate of oxygen consumption was generally less than that of non-acclimated fish. This is attributed either to a reduction in standard metabolic rate or to an enhancement of anaerobic respiration of the acclimated fish (Beamish, 1964b).

Threshold concentration. The threshold concentrations of dissolved oxygen have been suggested by many authors for various species of salmonid fish (Table 20) and of warm water fish (Table 21). In both groups of fishes, there is a great variation in the threshold concentrations for each species suggested by different authors. For salmonid fish the threshold concentrations vary from 0.94 mg/l for brown trout at 10 C (Gardner and Leetham, 1914) to 7-8 mg/l for brook trout and yearling coho salmon at 10-20 C (Davis et al. 1963). For warm water fishes the concentrations range from 0.3 mg/l for brown bullhead at 30 C (Basu, 1949) to 6 mg/l at 17 C for northern pike (Moore, 1942). These variations may be due to differences in test conditions, test populations, and perhaps in the definition of the so-called threshold concentration or minimum concentration.

Basu (1959) demonstrated that the threshold concentration of goldfish increased from 0.5 mg/l at 10 C to 0.7 mg/l at 30 C. Also under the same experimental condition, there was a difference in threshold concentrations among speckled trout, brown bullhead, carp and goldfish. At 16 C the concentrations are 0.35-0.52 mg/l for tench and 2.4-3.7 mg/l for rainbow trout. The salmonid fish have higher threshold concentrations of DO than warm water fish under the same experimental conditions.

There are several recommendations for the minimum required oxygen concentrations for aquatic life. Ellis (1937) suggested that to maintain a varied fish fauna in good condition the DO concentration should remain at 5 mg/l or higher. Patrick (1949) proposed that DO standards for polluted streams be the monthly average of 4 mg/l and daily minimum of 3 mg/l. Hewitt (1938) and Tarzwell (1958) suggested that the minimum DO concentrations for trout in soft water is 6 mg/l. The Aquatic Life Advisory Committee of the Ohio River Valley Water Sanitation Commission (Anon, 1955b) recommended that the minimum oxygen concentration for a well-rounded warm-water fish population be as follows: the dissolved oxygen concentration of warm water fish habitats shall be not less than 5 mg/l during at least 16 hours of any 24-hour period. It may be less than 5 mg/l for a period not to exceed eight hours within any 24-hour period, but at no time shall the oxygen content be less than 3 mg/l. To sustain a coarse fish population, the DO concentration may be less than 5 mg/l for a period of not more than eight hours out of any 24-hour period, but at no time shall the concentration be lower than 3 mg/l. The same standard was cited by Lagler (1959) and also recommended by Tazwell (1958). The National Technical Advisory Committee of the Federal Water Pollution Control Administration (Anon, 1968) recommended that for a diversified warm-water biota, including game fish, DO concentrations should

TABLE 20. THRESHOLD CONCENTRATIONS OF DISSOLVED OXYGEN PROPOSED FOR SALMONID FISHES

Concentration mg/l	Fish	Temperature C	References
0.94	Brown trout	10	Gardner and Leetham (1914)
1-3.5	Young Atlantic salmon	-	Privol'nev (1947)
1.1	Rainbow trout, brook trout	11	Burdick et al. (1954)
1.13	Brown trout	6.4	Gardner (1926)
1.16	Brown trout	9.5-10	Gardner (1926)
1.25	Silver salmon, steelhead trout, cutthroat trout	--	Townsend et al. (1938)
1.3	Coho salmon	16 and 18	Davison et al. (1959) Davison (1954)
1.35-2.35	Speckled trout	15.6	Burdick et al. (1954)
1.4	Cutthroat trout	--	Townsend et al. (1938)
1.4-1.7	Silver salmon, Coho salmon	18-22	Divison (1954), Davison et al. (1959)
1.42	Brown trout	9	Burdick et al. (1954)
1.5	Steelhead trout	--	Townsend et al. (1938)
1.50-1.54	Coho salmon	11.1-12.5	Townsend and Earnest (1940)

TABLE 20. THRESHOLD CONCENTRATIONS OF DISSOLVED OXYGEN PORPOSED FOR SALMONID FISHES (continued)

Concentration mg/l	Fish	Temperature C	References
1.51-2.85	Salmo salar	15-25	Privol'nev (1963)
1.52	Speckled trout	3.5	Graham (1949)
1.75	Speckled trout	9-10	Shepard (1955)
1.82	Rainbow trout	22	Burdick et al. (1954)
1.99-3.36	Pink salmon	17-25	Privol'nev (1963)
2.0-2.2	Speckled trout	10-15	Basu (1959)
2.13	Brown trout	18	Gardner (1926)
2.4	Speckled trout	23	Graham (1949)
2.4-3.7	Rainbow trout	16	Jones (1964)
2.5	Speckled trout, rainbow trout	20	Basu (1959), Gutsell (1929)
2.53	Brown trout	21	Burdick et al. (1954)
2.8	Brown trout	24	Gardner (1926)
2.9	Brown trout	--	Paton (1904)
3.48	Brown trout	25	Gardner and Leetham (1914)
6.0	Trout	--	Hewitt (1938)

TABLE 20. THRESHOLD CONCENTRATIONS OF DISSOLVED OXYGEN PROPOSED FOR SALMONID FISHES (continued)

Concentration mg/l	Fish	Temperature C	References
6.0	Brook trout, yearling Coho salmon	--	Graham (1949), Allen (1969)
7-8	Brook trout, yearling Coho salmon	10-20	Davis et al. (1963)

TABLE 21. THRESHOLD CONCENTRATIONS OF DISSOLVED OXYGEN PROPOSED FOR WARM WATER FISHES

Concentration mg/l	Fish	Temperature C	References
0.3	Brown bullhead	30	Basu (1959)
0.35-0.52	Tench	16	Jones (1964)
0.37-0.88	Yellow perch	15.5	Burdick et al. (1957)
0.4-1.4	Perca fluviatilus	15 and 25	Privol'nev (1963)
0.5	Goldfish	10	Basu (1959)
0.57-1.1	Dace	16	Jones (1964)
0.59-2.5	Mirror carp	16	Jones (1964)
0.6	Goldfish	20	Basu (1959)
0.6-1.6	Rutilus rutilus	15-23	Privol'nev (1963)
0.67-0.69	Roach	16	Jones (1964)
0.68-1.44	Bleak	16	Jones (1964)
0.7	Goldfish	30	Basu (1959)
0.72-1.4	Esox lucius	15-29	Privol'nev (1963)
0.75-1.23	Bluegill	25-35	Moss and Scott (1961)
0.9-1.2	Nile Tilapia	21.1-26.7	Whitworth and Irwin (1964)

TABLE 21. THRESHOLD CONCENTRATIONS OF DISSOLVED OXYGEN PROPOSED FOR WARM WATER FISHES (continued)

Concentrations mg/l	Fish	Temperature C	References
0.92-1.40	Largemouth bass	25-35	Moss and Scott (1961)
0.95-1.08	Channel catfish	25-35	Moss and Scott (1961)
1.0	Northern pike, crappie	4	Moyle and Clothier (1959)
1.0	Eel	17	Van Dam (1938)
1.1	Black bullhead	4	Moore (1942)
1.1	Carp	30	Paton (1904)
1.1-1.3	Perch	16	Jones (1964)
1.15	Smallwouth bass	15.6	Burdick et al. (1954)
1.2-1.7	Stone roller	21.1-26.7	Whitworth and Irwin (1964)
1.3-1.6	Common shiner	21.1-26.7	Whitworth and Irwin (1964)
1.4	Golden shiner, pumpkinseed	4	Moore (1942)
1.4-3.1	Young sturgeon	11-20	Lozinov (1952)
1-5	Fathead minnow	15-25	Brungs (1971)
1.5	Black crappie, banded killifish	4	Moore (1942)
1.5-1.6	Bluegill, silver chub	25	Anon (1958), Whitworth and Irwin (1964)

TABLE 21. THRESHOLD CONCENTRATIONS OF DISSOLVED OXYGEN PROPOSED FOR WARM WATER FISHES (continued)

Concentration mg/l	Fish	Temperature C	References
1.6-1.8	Northern hogsucker	21.1-26.7	Whitworth and Irwin (1964)
1.7	Crucian carp	5	Blazka (1958)
1.9	Bluegill	35	Anon (1958)
2.0	Orangespotted sunfish	4	Moore (1942)
2	Spring cavefish	21	Hill (1968)
2-4	Young American shad	21-23	Tagatz (1961)
2-5	Largemouth bass	--	Doudoroff and Warren (1965) Allen (1969)
2.0-2.5	Bluntface shiner, smallmouth bass, hornyhead chub	21.1-26.7	Whitworth and Irwin (1964)
2.2	Carp, buffalo	--	Thompson (1926)
2.3-2.6	Brook silverside	21.1-26.7	Whitworth and Irwin (1964)
2.25	Bluntnosed minnow, steel-colored shiner, yellow perch	20-26	Wilding (1939)
2.5	Carp, buffalo	--	Pearse (1933)
2.5	Eel	21.0	Hill (1969)

TABLE 21. THRESHOLD CONCENTRATIONS OF DISSOLVED OXYGEN PROPOSED FOR WARM WATER FISHES (continued).

Concentration mg/l	Fish	Temperature C	References
2.7	Fish	--	Kussat (1969)
3.0	Young striped bass	17	Chittenden (1971)
3.0	European perch	--	Fry (1957), Allen (1969)
3.2	Northern pike, Rock bass	4	Moore (1942)
3.4	Bluegill, black bullhead	23	Moore (1942)
3.6	Bluegill, green sunfish	4	Moore (1942)
4.3	Yellow perch, pumpkinseed	26	Moore (1942)
4.8	Yellow perch, largemouth bass	4	Moore (1942)
5.6	Largemouth bass, black crappie	15.5	Moore (1942)
6.0	Northern pike	17	Moore (1942)

be above 5 mg/l, assuming normal seasonal and daily variations are above this concentration. Under extreme conditions, however, they may range between 5 and 4 mg/l for a short period during any 24-hour period. For cold-water biota, it is desirable that DO concentrations be at or near saturation. This is especially important in spawning areas where DO levels must not be below 7 mg/l at any time. For good growth and the general well-being of trout, salmon, and their associated biota, DO concentrations should not be below 6 mg/l. Under extreme conditons, they may range between 6 and 5 mg/l for a short period.

The Committee on Water Quality Criteria, Environmental Studies Board, National Academy of Science and National Academy of Engineering (Anon, 1972) recommended a floor value of 4 mg/l for freshwater aquatic life, except in those situations where the natural level of DO is less than 4 mg/l, in which case no further depression is desirable. Several formulae are established to calculate the minimum dissolved oxygen required for aquatic life in any season on the basis of minimum natural seasonal concentration of DO. For example, in a trout stream where oxygen concentration is at the saturated level at 20 C during the summer, the minimum permissible concentration of DO is 7.8 mg/l for a high level of protection and 4.5 mg/l for a low level of protection. For warm water fish such as large mouth bass, during the summer at 30 C the recommended minimum oxygen concentration is 5.4 mg/l. The European Inland Fisheries Advisory Commission (Anon, 1973) also suggested that, in general, a minimum of 5 mg/l DO would be a satisfactory limit for most of the processes required for a successful fish life cycle.

McKee and Wolf (1963) indicated that on the basis of available information it is not feasible to suggest an optimal DO content of water for fish and other aquatic life. They considered that the recommendation of the Aquatic Life Advisory Committee of the Ohio River Valley Water Sanitation Commission (Anon, 1955b) appears to be logical. Doudoroff and Shumway (1967) concluded that standards such as a minimum of 5 mg/l for warm water fish habitats and 6 or 7 mg/l for cold-water fish habitats, which have been recommended and widely accepted for a long time, cannot be overthrown, although none was based initially on a firm factual foundation.

The Committee of Water Quality Criteria, Environmental Studies Board, National Academy of Science and National Academy of Engineering (Anon, 1972) suggested that there is no single arbitrary recommendation can be set for DO concentration that will be favorable for all kinds of water or even one kind of fish in a single kind of water. Any reduction in DO may be harmful by affecting fish production and potential yield of a fishery Doudoroff (1959) stated that standards adopted by various regulatory agencies for controlling DO concentrations vary widely, largely owing to differences of opinion on the objectives to be achieved. Because of these differences of opinions and the factors to be considered in establishing standards for DO concentrations,

the determination of water quality standards is an administrative problem and not a purely scientific problem. A similar statement was made by Doudoroff and Shumway (1967).

Estuarine and Marine Fishes

Lethal effects and destruction of fisheries. Mass fish kills or destruction of fisheries by DO deficiency in estuarine and coastal water due to decomposition of organic matters in sewage, or mass decomposition of dead algae after their massive bloom, have been reported in many parts of the world - Gray Harbor, Washington (Townsend et al. 1938), New York Harbor (Torpey, 1967), Delaware Estuary (Chittender, 1971), the west coast of Florida (Gunter et al. 1948), Offats Bayou, Texas (Connell and Cross, 1950), Walvis Bay, Southwest Africa (Copenhagen, 1953), False Bay, South Africa (Taylor, 1962; Grindley and Taylor, 1962, 1964), Lake Mariut, Egypt (Saad, 1972), Usk estuary (Freeman, 1969), the upper Lane Cover River, Australia (Costin, 1954), the Adriatic Coast, Italy (Froglia, 1970), the Thames estuary (Wheeler, 1969, 1970, Torpey, 1967), Bay of Bengal, India (Ramamurthy, 1970), Kulti estuary, India (David, 1959), Bonne-Fojord (Braarud, 1955) and Oslofjord (Foryn, 1959). Renfro (1963) reported fish kills by supersaturation of DO by algal bloom in Galveston Bay, Texas.

There are several studies of the lethal effects of DO on estuarine and marine fishes in the laboratories. They are listed on Table 22. Hoff et al. (1966) found that the lethal concentrations of DO for common silverside, American shad, winter flounder, and northern wellfish were influenced by environmental and physiological factors such as temperature, DO, pH and stress of fishes. They suggested that, for a given body of water, the DO standard should be high enough to meet the changes of these factors. The high concentrations of carbon dioxide impaired the oxygen-combining ability of the blood in marine fish (Root et al. 1938; Root and Irving, 1940). Near the low limit of DO tolerance, increase in the hydrogen ion concentration produces the same effect as lowering the oxygen concentration on fish (Townsend and Cheyne, 1944).

The effects of DO deficiency on migratory salmon were reduction in swimming stamina, respiratory efficiency, oxygen consumption, urine flow, and ammonia excretion, increase in lactate in blood, and long-term disruption in haematology and lipid metabolism. Most of these effects occurred at DO concentrations below 5 mg/l (Smith et al. 1971). The juvenile striped mullet (Magil cephalus) exposed to hypoxic condition increased ventilation volume and frequency, and stroke volume, but decreased in percentage utilization of oxygen without change in the routine rate of respiratory metabolism. These responses probably represent ventilatory adjustment which serves to maintain a constant oxygen supply to the gills under conditions of oxygen depletion (Cech and Wahlschlag, 1973). The resistance of the larvae of herring, lumpsucker, salmonids, and plaice to low concentrations of DO decreased with increase

TABLE 22. LETHAL EFFECTS OF DISSOLVED OXYGEN ON ESTUARINE AND MARINE FISHES

Concentration mg/l	Fish	Test condition	Survival time	References
0.66-0.77	Winter flounder, common silverside, northern swellfish	12 C	24-hr. TLm	Hoff (1967)
0.72-1.23	Skilletfish	25 C, 19.8-20.7% salinity	24-hr. TLm	Saksena and Joseph (1972)
0.80	Mummichog	20.0 C, pH 8.0, 30-32% salinity	6 hr. TLm	Voyer and Hennkey (1972)
0.87-0.93	Winter flounder, common silverside, northern swellfish	18.5 C	24-hr. TLm	Hoff (1967)
1.03-1.36	Winter flounder, common silverside, northern swellfish	25 C	24-hr. TLm	Hoff (1967)
1.30	Naked goby	26.7 C, 19.8-20.7% salinity	24-hrs. TLm	Saksena and Joseph (1962)
1.5-2.0	Fish in Black Sea		Lethal	Kahinina (1961)
2.50	Striped blenny	25.6 C, 19.8-20.7% salinity	24-hr. TLm	Saksena and Joseph (1972)

in their ages. This was due to the mode of respiration, degree of activity, and presence of carbohydrate. All the fish survived in water containing high concentrations of DO up to 300 percent saturation with no apparent ill effects, as long as the total gas pressure in the water did not exceed the hydrostatic pressure (Bishai, 1960). When juvenile Pacific rockfish were exposed to hyperbaric oxygen tension equal to that in their swim bladders (1.5-5 atm. absolute), the fish exhibited symptoms characteristic of oxygen poisoning in mammals, and finally died. It appears that the central nervous system of these fish is sensitive to high oxygen pressure. The cells of the gas gland in the swimbladders must be insensitive to the partial pressure of DO they produce (D'Aoust, 1969).

Fish behavior. The cycle of abundance of starry flounder, English sole, and staghorn corresponded to changes in dissolved oxygen concentration near the bottom, suggesting the avoidance reaction of these fish to low dissolved oxygen (Gibbs and Isaac, 1968). Striped bass, white perch, shad, blueback herring and alewives responded to oxygen concentrations below 2.0 mg/l by moving toward the surface of the test chambers. These fish spent more time in high oxygen concentrations when exposed to gradients of dissolved oxygen. They, however, apparently did not have an instinctive ability quickly to recognize and avoid waters of low oxygen concentrations (Dorfman and Westman, 1970). In laboratory experiments, Deubler and Posner (1963) showed that marine flatfish (Paralichthys lethostigma) removed themselves from water of low oxygen concentration when the values fell below 3.7 ml. There was no difference in sensitivity to oxygen depletion at temperatures of 6.1 to 25.3 C.

Growth and reproduction. The growth rate of the striped bass was significantly impaired by daily exposure to diurnal fluctuations of DO concentration less than 4.0 mg/l on the average. For white perch, it was less than 3.8 mg/l (Dorfman and Westman, 1970). The development of the plaice embryos was delayed in water containing DO levels less than 50 percent saturation at 6 to 7 C (Johansen and Krough, 1914). For the Pacific cod larvae, their development was retarded at DO concentrations of 4-5 mg/l and total dissolved solids exceeded 1,000 mg/l (Alderdice and Forrester, 1971). The mummichog tolerated a low concentration of DO of 0.8 mg/l for up to six hours, but their embryonic development was retarded or inhibited at 2.4-4.5 mg/l. A 50 percent hatch of the mummichog's eggs occurred at 3.9 mg/l of DO (Voyer and Hennkey, 1972). The desert pupfish embryos' development was retarded by exposing them in water containing 70 percent saturation of DO and 35 per thousand of salinity at temperatures greater than 18 C. The developmental arrest was reversible, if the ambient dissolved oxygen was increased within hours or a few days. Longer periods of exposure of the embryos to reduced oxygen levels were lethal (Kinne and Kinne, 1962a,b).

Threshold concentrations. Chittenden (1971) suggested

that the minimum DO level at any time for reasonably normal existence of striped bass was about 3 mg/l near 17 C, although this concentration might not be enough to maintain optimal population. In experimental studies of the minimum DO requirement of the Black Sea fishes, Kalinina (1961) recommended that the DO content should not be lower than 4 mg/l at 9-10 C for coastal and pelagic fishes, not lower than 5 mg/l for whiting, and not lower than 2.5-3 mg/l for benthic species. Collette (1967) indicated that salmon and trout enter estuaries at either end of the passage, if not exposed to toxic chemicals, or to DO levels lower than 60 percent saturation, or to water temperatures higher than 22-26 C; they would survive and the fisheries would be maintained. Fales (1934) proposed that water for fish life must contain at least 3 mg/l DO.

The National Technical Advisory Committee on Water Quality Criteria, Federal Water Pollution Control Administration (Anon, 1968) recommended that the minimum dissolved oxygen level in estuaries and tidal tributaries shall not be less than 4 mg/l. The surface dissolved oxygen concentrations in coastal waters shall not be less than 5 mg/l, except when natural phenomena cause this value to be depressed. The Committee on Water Quality Criteria, Environmental Studies Board, National Academy of Science and National Academy of Engineering (Anon, 1972) recommended that to protect marine fish at a maximum seasonal temperature of 16 C, the minimum DO is 137.1 mg/l for a high level of protection, 5-8 mg/l for a moderate level of protection, and 4.3 mg/l for a low level of protection.

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Toxic Dinoflagellates and Fish Mortality

Red tides resulting from the bloom of dinoflagellates have been observed in coastal and estuarine waters in different parts of the world. They often inflict catastrophic mortality of fish and other marine organisms, and/or cause paralytic shellfish poisoning. The species of red tides which have been implicated in causing fish kills are summarized in Table 23.

The red tide bloom of G. breve. which has been responsible for the mass mortality of fish on the west coast of Florida and coasts of Texas and Mexico, has also caused mortality of other marine life including shrimps, crabs, oysters and barnacles (Gunter et al. 1948). The toxicity of G. breve to oysters has been proved; the reason for relative infrequency of oyster mortality of G. breve bloom on the west coast of Florida is that oysters usually grow in Gulf areas with salinity levels of 25 per thousands or less, a concentration which inhibits development of this dinoflagellate (Ray and Aldrich, 1967). On the other hand Sievers (1969) found that the cultured G. breve was not toxic to crustacea and mollusks, but toxic only to fish. He suggested that mortality of oysters and crabs reported by Gunter (1948) was an indirect result of oxygen deficiency rather than a direct result of the toxic bloom.

Toxicity of G. breve to fish has been studied in the laboratory. Galtsoff (1948) immersed killifish in redwater extract and the fish died in two and one half hours. Ray and Wilson (1957) found that bacteria-free culture was as toxic as uni-algal culture containing bacteria, indicating that this dinoflagellate is the direct cause of the mass mortality of fish and other marine animals associated with bloom. The toxicity of G. breve, increased by physical and chemical procedures, effectively destroyed the organisms and does not depend on the presence of the living cells (Ray and Wilson, 1957; Starr, 1958).

In Offats Bayou, Texas, Connell and Cross (1950) found that fish usually died when G. monilata bloom submerged and suggested that the decrease in oxygen concentration was a contributing factor to the fish mortality. On the other hand Gtates and Wilson (1970) found that G. monilata produces a substance or substances toxic to Mugil cephalus, and the toxicity of the culture medium increased if the dinoflagellates cytoses by heat or freezing. They suspected that the submergence of G. monilata reported by Connell and Cross (1950) was actually the decomposition of the flagellate cells and that fish would die at that time because more toxins would be released.

Abbott and Ballantine (1957) extracted a toxin from G. veneficum. It was found to be toxic to fish, slightly so to mulluses and crustaceas, and not at all to annelids. The toxin acts on nervous systems of animals by depolarizing excitable membranes. The death of fish is due to respiratory

TABLE 23. FISH KILLS ASSOCIATED WITH "RED TIDE" BLOOMS OF DINOFLAGELLATES IN COASTAL AND ESTUARINE WATERS

Dinoflagellate species associated with red tide	Locations	Fish and other animals killed	References
Entomosigana sp.	Coast of Fukuyama, Japan	Fishes	Iwasaki et al. (1968)
Exuviella baltica	Coast of Angola	Fishes, crabs	Paredes (1964)
Glenodinium foliaceum	Baltic coast of Germany	Fishes	Lindemann (1926)
Glenodinium trichoideum	Brackish water Lagoa Rodrigo de Freitas and Bay of Guarahara, Brazil	Fishes	Oliverra et al. (1956)
Gonyaulax polyedra	Coast of California	Stingray, guitar fish, dogfish, perch, smelt, octopi, benthic invertebrates	Torrey (1902), Reish (1963)
Gonyaulax polygrama	Bays of Tobe and Aga, Japan, and False Bay, Capetown, South Africa	Fishes, mollusks, shrimps, other invertebrates	Mishikawa (1903); Taylor (1962); Grindley and Taylor (1962,1964)
Gonyaulax monilata	Offats Bayou near Galveston, Texas and Melborne, east coast of Florida	Fishes	Connell and Cross (1950); Howell (1953)
Gonyaulax tamarensis	North east coast of England	Sand eels, birds, starfish, uncommercial bivalves	Clark (1968)

TABLE 23. FISH KILLS ASSOCIATED WITH "RED TIDE" BLOOMS OF DINOFLAGELLATES IN COASTAL AND ESTUARINE WATERS (continued)

Dinoflagellate species associated with red tide	Locations	Fish and other animals killed	References
Gymnodinium breve	West coast of Florida and coast of southern Texas and eastern Mexico	Fishes, barnacles, shrimps, crabs, oysters	Gunter et al. (1948); Davis (1948); Galtsoff (1948); Odum et al. (1955); Lackey and Hynes (1955); Finucane (1960); Finucane et al. (1964); Berg (1971); Cummins et al. (1971); Ray and Wilson (1957); Wilson and Ray (1956)
Gymmodinium sp.	Omura Bay, Nagasaki, Japan	Fishes	Iizka and Irie (1969)
Gyrodinium aurolum	Coast of southern Norway	Fishes	Braarud and Heimdal (1970)
Noctiluca scintillans	Madras, Malabar and Kamara coasts of India and coasts of South Africa	Fishes	Aiyar (1936); Bhimachar and George (1950); Grindley and Heydorn (1970)
Peridinium depressum	Adriatic Coast, Italy	Fishes	Froglia (1970)
Peridinium foliaceum	Moray Firth, Scotland	Fishes	Rae et al. (1965)
Peridinium sp.	West coast of India	Fishes	Carter (1858)
Peridinium sp.	Narragansett Bay, Rhode Island	Menhaden, flatfish, eels, tautag, shrimps, crabs	Meade (1898)

TABLE 23. FISH KILLS ASSOCIATED WITH "RED TIDE" BLOOMS OF DINOFLAGELLATES IN COASTAL AND ESTUARINE WATERS (continued)

Dinoflagellate species associated with red tide	Locations	Fish and other animals killed	References
Prorocentrum micans	Mejillones, Chile, and False Bay, Capetown, South Africa	Fishes, invertebrates	Villar (1966); Grindley and Taylor (1962,1964)
Trichodesmium erythraeum	The Bay of Bengal	Fishes	Ramamurthy (1970)

failure. The primary site of entry of the toxin is the gills and not orally. G. monilata produce a toxin different from the one produced by G. breve. Fundulus grandis killed by the former tended to sink and those killed by the latter tended to float (Ray and Aldrich, 1967).

The other species of dinoflagellate, Noctiluca scintillan, caused fish kills in the coastal waters of India and South Africa. However, when it bloomed off the coast of Hong Kong, its toxicity was so minimal that it did not lead to fish kill (Morton and Twentyman, 1971). In the False Bay, South Africa, mass mortality of fish and marine invertebrates by red tide bloom of G. polygrama and P. micans was mainly due to oxygen depletion in the water by decaying of dead dinoflagellates, and living dinoflagellates did not appear to have any harmful effect (Taylor, 1962; Grindley and Taylor, 1962 and 1964). Brongersma-Sanders (1947) attributed the fish kills by the red tide in South Africa to the toxins liberated from the dinoflagellates. Grindley and Taylor (1964) suspected that it is possible that a type of toxin may be released by decaying plankton.

In the Moray Firth, Scotland, Rae et al. (1965) found that during red tide there was below normal oxygen content and large numbers of toxic Peridinium foliaceum. Although either of these factors was sufficiently severe to kill the fish, evidence suggested that their combined effect could have caused a toxic condition. The oxygen deficiency as the result of red tide bloom was also reported for Peridinium depressum in the Adriatic coast of Italy (Froglia, 1970) and for Trichodesnium erythraeum in the Bay of Bengal (Ramamurthy, 1970). The latter dinoflagellate is nontoxic and its bloom is common in the tropics and gives the Red Sea its name (Hutner and McLaughlin, 1958).

Fish reaction to the red tide has been described by several investigators. Mead (1898) observed the reaction of fish and other marine organisms to the red tide of Peridinium sp. in Narragansett Bay, Rhode Island. Blue crabs, myriad shrimps, great numbers of eels, menhaden, tautog, and flatfish came up to the surface and to the edge of the shore as though struggling to get out of the noxious water. In the west coast of Florida, fish entering the red tide zone of G. breve acted strangely, coming to the surface, whirling around, then overturning and sinking to the bottom (Galtsoff, 1948). Abbott and Ballantine (1957) immersed Gobius virescus in the culture medium of G. veneficum. The fish showed a violent attempt to swim away for about two minutes and then subsided, causing vasodilation, expansion of chromatophors, slow breathing, vomiting reaction, irregular thrust of violence, and finally died of respiratory failure. Mugil cephalus immersed in the culture medium of G. monilata first exhibited distress by frenzied activity, followed by the loss of equilibrium. The fish then turned upside down with slow opercular movement and eventually died. Immediately before death there

was a violent burst of activity and the fish died with mouth and opercula open (Gtates and Wilson, 1960).

Besides causing the mass mortality of fish mentioned above, many species of dinoflagellates have been implicated in the paralytic shellfish poisoning (Table 24). The toxin has a peripheral action on the muscle membrane and also blocks conduction in motor and sensory nerves. The patients who ate the toxic shellfish show ataxia, dysmetria, intention tremor, and occasionally nystagmus and temporary hypertension (McCollum et al. 1968) and peripheral paralysis and death by paralytic shellfish poisoning observed on the northeast coast of England, the symptoms were excessive vomiting, loss of equilibrium and muscular coordination, paralysis, respiratory failure and lung congestion (Clark, 1968).

Except for a few species of the dinoflagellates such as G. breve, N. scintellans, and G. polyedra which are implicated in both fish mortality and shellfish poisoning, most of the dinoflagellates cause either fish kill or paralytic shellfish poisoning. It appears that the toxin produced by dinoflagellates which causes paralytic shellfish poisoning may be different from that liberated by other dinoflagellates associated with mass mortality of fish (Sommer and Meyer, 1937). Clam and mussel toxin is identical with that produced by Gonyaulax (Burke et al. 1960; Schantz, 1960).

The outbreak of shellfish poisoning resulting from the bloom of G. tamarensis in 1968 in northeast England was preceded by a mass mortality of sand eels, sea birds and noncommercial shellfish, a week after the bloom (Robinson, 1968). Clark (1968) and Wood (1968) suggested that the sand-eels were killed by ingesting a toxin by feeding on copepods and other planktonic habivoric fishes which formed the link between the toxic dinoflagellates and the sand-eels. The birds were killed by eating the toxic sand-eels. However, Ingham et al. (1968) could not detect the toxin in plankton-feeding herrings, and in the dead sand-eels in the infected area. Recently, Prakash (1967) found that the toxin of G. tamarensis is chiefly an intracellular constituent and the actual ingestion of toxic dinflagellates is unlikely to cause death in cold blooded animals. As the culture of G. tamarensis, aged cells died, releasing liberated toxin into the medium. Accordingly, he suspected that the death of the sand-eels on the northeast coast of England in 1968 occurred about a week after the bloom and thus low oxygen resulting from mass decay of the bloom could have contributed an added stress to the sand-eels. Death of birds and marine mammals by the way of shellfish which had ingested toxic dinoflagellates also occurred on the Atlantic Coast of Canada. The shellfish themselves appeared unaffected. There is no evidence of toxicity to other invertebrates (Medcof et al. 1947).

In the Chesapeake Bay, most of the genera of Dinophyceae, which have been reported elsewhere in the world as the causative organisms of red tide, are present. They are Exuviella,

TABLE 24. "RED TIDES" ASSOCIATED WITH PARALYTIC SHELLFISH POISONING IN COASTAL AND ESTUARINE WATERS

Dinoflagellate Species	Location	Affected shellfishes and other animals	References
Exuviella mariaelebouriae	Japan	Shellfish poisoning	Nakazima (1968)
Gonyaulax acatenella	Strait of Georgia, British Columbia	Shellfish poisoning	Prakash and Taylor (1966)
Gonyaulax catanella	San Francisco and Monterey, California	Shellfish poisoning	Sommer, et al. (1936), Sommer and Meyer (1937); Hutner and McLaughlin (1958)
Gonyaulax tamarensis	Bay of Fundy, St. Laurence River estuary, and Head Harbour, New Brunswick, Canada, coasts of Scotland, and northeast coast of England	Shellfish poisoning and mortality of sand eels, birds, starfish, bivalves	Gemmill and Manderson (1960); Clark (1968); Ingham et al. (1968), Coulson et al. (1968); Adams et al. (1968); Wood (1968); Robinson (1968); McCollum et al. (1968); Prakash and Medcof (1962)
Gonyaulax polyedra	Coast of California	Shellfish poisoning	Schradie and Bliss (1962)
Gonyaudax washingtonensis	Sequim Bay, Washington	Shellfish poisoning	Dupuy and Sparks (1968)
Gonyaulax sp.	Southern California	Shellfish poisoning	Reish (1963)
Gymnodinium breve	West coast of Florida	Shellfish poisoning	Cummins et al. (1971); Eldred et al. (1964); McFarren et al. (1965)

TABLE 24. "RED TIDES" ASSOCIATED WITH PARALYTIC SHELLFISH POISONING IN COASTAL AND ESTUARINE WATERS (continued

Dinoflagellate Species	Location	Affected shellfishes and other animals	References
Noctiluca scintillans	British Columbia, Canada	Shellfish poisoning	Quayle (1969)
Pyrodinium phoneus	France		Koch (1939)

Prorecentrum, Gymnodinium, Gyrodinium, Glenodinium, Gonyaulax, and Peridinium. The species found in the Bay and recorded to have caused red tides and fish kills and/or paralytic shell fish poisoning elsewhere in the world are Prorecentrum micans, Gonyaulax catenella, G. polyedra, G. polygrama, and Peridinium depressum (Griffith, 1967). Of them, G. polyedra is most common throughout the bay and its tributaries (Mulford, 1972). Also, incidents of mass fish mortality have been often reported from the bay. It is unknown whether there has been an outbreak of red tide by these organisms and associated with the incidents of fish mortalities in the Chesapeake Bay.

Toxic Phytoflagellates and Fish Mortality

Prymesium parvum is a typical brackish water phytoflagellate whose blooms have been implicated in mass fish mortality in diked marshes in the Netherland (Liebert and Deens, 1920) and brackish water ponds in Denmark (Sproston, 1946; Otterstoem and Steemann-Nielson, 1939). In 1947 it first appeared in fish ponds in Israel and rapidly spread through the region where the water was brackish and caused extensive damage to the fish (Shilo and Shilo, 1953, 1955). Reich and Aschner (1947) observed that carps became sluggish, tried to leap out of the poisonous water, became listless and died by the thousands. Gambusia affinis and carps immersed in pond water containing high numbers of this phytoflagellate died in two to four hours. Loss of equilibrium by intoxicated fish was always followed by the death of the fish (Shilo and Aschner, 1953). A low concentration of ammonium sulfate which exerts lytic effect on this phytoflagellate has been used effectively to control the blooms of this toxic algae in the brackish ponds in Israel (Komarovsky, 1952; Shilo and Shilo, 1953, 1955; Reich and Aschner, 1947; Kessler, 1960).

Toxicity of P. parvum was detected by immersion of eel (Otterstroem and Steemann-Nielson, 1939) and of carp and Gambusia affinis in the water infected with this toxic phytoflagellates. The fishes died in a few hours. Shilo and Aschner (1953) found that cell free fluid of cultured medium of P. parvum was as toxic as the orginal cell suspension to G. affinis indicating that the toxin was liberated by organism into the cultured medium. The toxicity of the P. parvum culture medium was decreased by mild acidity, oxidizing agents and bacterial growth (Shilo and Aschner, 1953), and exposure to light (Reich and Parnas, 1962). More toxin was formed in five percent sea water than in 30 percent sea water in bacteria-free culture, confirming the fact that mass mortality of fish has been observed only in brackish water, although it also occurs in the sea (Reich and Rotberg, 1958). In the supernatant of P. parvum culture medium, there are two hemolysins potent against red cells of various species of animals (Yariv and Hestrin, 1961). The toxicity of P. parvum to fish was reviewed by Parnas (1963). Recently, the toxin of

P. parvum was isolated and found to be a glycolipid containing about 70 percent sugars (Paster, 1968). In contrast to the above evidences of toxicity of P. parvum to fish, Reich and Kahn (1954) showed that bacteria-free culture of P. parvum had no poisonous effect on fish, or only slightly so.

Sewage, Nutrients, and Red Tides Outbreak

There are many meterological and oceanographical conditions which have been suspected of being associated with red tide breakout. Of them, nutrient enrichment due to upwelling of bottom water and to freshwater drainage from the land has been regarded as being among the most important causative factors.

Brongersma-Sanders (1947, 1957) indicated that the red tide bloom on the southwest coast of South Africa is the result of the presence of upwelling water which supplies an unusually high concentration of nutrients to dinoflagellate, N. scintillans. The same phenomenon was suggested to have occurred in the red tide outbreak by E. baltica along the coast of Angola; where the upwelling was related to the occurrence of a southerly wind (Paredes, 1964). In the coastal water of sourthern Norway, the bloom of G. aurolum was accompanied by a local upwelling (Braarud and Heimdal, 1970). The upwelling of bottom water has been also implicated in the red tide outbreak in bays, such as in the cases of G. polygrama in False Bay, South Africa (Taylor, 1962) and of Gymonidinium sp. in Omura Bay, Nagasaki, Japan, (Iizuka and Irie, 1967). Germination of spores and multiplication of Gymonidinium sp.may be accelerated in water containing nutrients and some unknown growth-promoting substances related to bottom mud.

The red tide outbreak of G. breve on the west coast of Florida and Gulf of Mexico has been suggested as due to terrigenous nutrient supply (Gunter et al. 1948; Ketchum and Keen, 1948; Lackey and Hynes, 1955), particularly inorganic phosphorous (Smith, 1948). It was suspected that during the rainy season the introduction of large volumes of land drainage with heavy loads of organic materials can produce the initial stimulus for heavy blooms of G. breve, which in turn may augment themselves with both inorganic and organic materials produced by the decomposition products of dead fish (Gunter et al. 1948; Collier, 1958; Wilson and Collier, 1955). This concept was stressed by Odum et al. (1955) who indicated that the blooms of G. breve with the resultant fish mortality occurred in the presence of high concentrations of nutrients. The fertile water with high nitrate and phosphates in the red water zone, was possibly maintained by drainage of rivers containing high concentrations of phosphorous derived from phosphate bearing rocks or from sewage. The continual localization of G. breve red tide blooms in the same area of the southwestern coast of Florida implied that the important inorganic and organic nutrient

conditions are being supplied from estuarine sources. Feinstein (1956) found that the red tide breakout of G. breve on the west coast of Florida correlated with prior storms and was related to the nutrients discharged from rivers.

In contrast to the above studies, Chew (1956) indicated that there is no correlation between rainfall and runoff magnitudes and the red tide incidents on the west coast of Florida. However, he suggested that estuarine conservation of runoff is necessary to provide the proper incubatory environment into which G. breve may be seeded from the source. Bein (1957) studied the total concentration of phosphorus in sea water before, during, and after outbreaks of red tide in the area and found that the blooms are not associated with an abnormally high concentration of phosphates. He concluded that sea water in the area contains sufficient phosphorus at all times of the year to support the red tide.

The outbreaks of dinoflagellate blooms are often associated with high concentrations of nutrients by heavy rains or land washing down from sewage and low salinity (Hutner and McLaughlin, 1958; Long, 1953; Ryther, 1955; Ketchum and Keen, 1948; Feinstein, 1956; Rounsefell and Nelson, 1966). Numann (1957) indicated that the red tides which occurred in the Bay of Luoanda, Portugese West Africa and the Bay of Izmir, Smyrna, were caused by nutrients brought into the bays by freshwater. Iwassaki et al. (1968) suggested that in coastal water off Fukuyma, Japan, the red tide of bloom of Entomosigma sp. occurred after rainfall, suggesting that the growth of this dinoflagellate was promoted by low salinity and possibly by nutrients washed into the sea from the land. In the laboratory, the authors found that this dinoflagellate prefers low salinity, and its growth was accelerated by trace metals or nitrilo-triacetic acid and also by supplements of nitrogen and phosphorus. The outbreak of red tide bloom of Gymnodinium sp. in July in Omura Bay, Magasaki, Japan was also suggested to be associated with inflow of freshwater containing nutrients in the rainy season (Iizuka and Irie, 1969). Morton and Twentyman (1971) indicated that red tide outbreak by N. scintillans in the water off Hong Kong Harbor occurred just after typhoon which stirred up nutrient rich bottom deposit and sewage discharged in the harbor.

Besides the nutrient requirement, there are several growth-promoting substances which have been suggested as stimulating the red tide outbreak. Collier (1958) showed that Vitamin B_{12} produced by bacteria associated with unialgal culture of G. breve was found to be a growth promotor of this dinoflagellate. The bacteria do not develop to a position of dominance in the absence of G. breve, suggesting that the dinoflagellate probably supply substrate for the bacteria growth, which in turn, supply vitamin B_{12} for the red tide bloom. He also found that carbohydrates produced by Prorecentrum sp. could serve as substrates for vitamin B_{12}-producing bacteria and thus play a part in the conditioning of water for succeeding populations of the dinoflagellates. Gandhi and Freitas (1964) found that in the experimental culture the marine microorganisms produced growth promoting substances containing

vitamin B_{12}. Gold (1964) also found that the dinoflagellate Glenodinium halli responsible for the red tide bloom in Long Island Sound, New York, required vitamin B_{12}. Prakash and Taylor (1966) showed that during an extensive bloom of the dinoflagellate Gonyaulax acatenella in the Straights of Georgia, British Colombia, a very high concentration of vitamin B_{12} was present in the infected water. Besides the sources of vitamin B_{12} produced by the microorganisns in sea water, Hunter and McLaughlin (1958) suggested that quantities of vitamin B_{12}, which is essential to dinoflagellate metabolism, may be washing into the ocean from the soil and from salt marshes where it is manufactured by bacteria and blue-green algae. Iwasaki et al. (1968) indicated that the dinoflagellate Entomosigna needs vitamin B_{12}, but all the analogues containing benzinidazole can replace this vitamin. Gibberellic acid is also found to be a growth promotor for T. erythraeum (Ramamurthy, 1970). Thiamine is found to stimulate growth of G. halli (Gandhi and Freitas, 1964).

The connection between sewage pollution, which contains high concentrations of nutrients and vitamin B_{12}, and red tide outbreak has been suggested by several authors. Nitta (1972) indicated that the frequent occurrence of red tides in bays such as Tokyo, Ise, Osaka, and Hakata, Japan, were caused, under certain conditions, by a high concentration of nitrogen and phosphorus as the result of eutrophication by sewage. It is thought that the red tides are produced in waters where COD values are about 1 mg/l in the areas where the river waters are somewhat diluted. Cole (1972) suspected that there may be some connection between the build-up of nutrients due to sewage and the occurrence since 1968 of the toxic blooms of dinoflagellates off the northeast coast of Britain and the west coast of Denmark. Hutner and McLaughlin (1968) indicated that the outbreak of red tide associates with high concentration of nutrients by heavy rain on land washing down from sewage and the low salinity. Morton and Twentyman (1971) implicated the sewage pollution as one of the contributing factors in the outbreak of red tide bloom of N. scintillans in the coastal water of Hong Kong by making nutrients available to the dinoflagellate or by faecal bacteria acting as an immediate source for this holozoic organism.

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Utilization of sewage in fish culture. Utilization of domestic sewage as a fertilizer in fish culture has been practiced in many parts of the world (Vass, 1949, 1952; Boss, 1944; Ganapati and Chacko, 1950; Wolney, 1963; Allen, 1969, 1972; Bardach, 1968; Bardach and Ryther, 1968; Thorsland, 1971). Cities as large as Munich, Germany dispose of most of their sewage in fish ponds to produce edible fish for local consumption and for export to neighboring countries (Neess, 1949). Sewage-recycled fish have been commonly acceptable food in most countries in Europe and Asia. In the Orient, use of sewage in fish culture is an ancient art started thousands of years ago. There is no question of the nutritional value of domestic sewage in fish culture (Schaeperclause, 1933; Prowse, 1965; Bardach, 1968). Recently, perhaps due to pollution problems and protein deficiency in many countries, there is a world-wide increase in the use of the fertilizing potential of domestic sewage for fish and shellfish culture (Allen, 1972) and in the recognition of sewage as a valuable natural resource (Allen, 1972; Huguenin and Kildow, 1974). Actually, sewage is a better fertilizer than chemical fertilizers for fish ponds, because of its longer lasting effect on the growth of phytoplankton (Komarovsky, 1952).

At the present time, there are three major methods of sewage utilization in fish culture: fish stocking directly into sewage lagoons, addition of sewage to fish ponds, conversion of sewage nutrients to phytoplankton and then discharging into fish ponds. The first method has been experimented with and used since lagoon treatment of sewage was adopted. Goldfish and carp (Bay and Anderson, 1965) and prickly sculpin and turtle perch (Cook, 1964) were stocked in sewage lagoons to control chironomid larvae. Mackenzie and Campbell (1963) stocked Tilapia fish to control mosquitos in a lagoon in Natal, South Africa. Although they were mainly for insect control in the sewage lagoons, they proved the lagoons to be useful as fish culture ponds.

In Ames, Iowa, Huggins and Bachmann (1969) stocked channel catfish in a series of four tertiary sewage treatment ponds. During the summer, catfish grew well in the third and fourth ponds and the survival rate, growth, and production of the fish progressively increased from the first to fourth ponds. In the same ponds, Konefes and Bachmann (1970) propagated fathead minnow and found that the growth of the fish was comparable to that of hatching ponds. They claimed that the use of tertiary sewage treatment ponds for fish production seems to be a useful approach to the conservation of natural resources. The same fathead minnow propagation in a municipal wastewater stabilization pond was done by Trimberger (1972) in Michigan.

In Tucson, Arizona, channel catfish, rainbow trout and

Malacca-Tilapia hybrids were stocked in a series of four tertiary sewage treatment ponds for recreational purpose. However, in some instances there was less than one percent survival, because of dissolved oxygen depletion at night. When survival occurred, there was high production of fish (Hallock and Ziebell, 1970). In Santee, California, in a series of ponds receiving sewage from the community, a reasonably well-balanced ecological system was established to become a socially accepted recreational center including sport fishery (Merrell and Katko, 1966).

In Bhilai Steel Works, India, carp were stocked in a sewage lagoon and it was found that the fish survived under conditions in which dissolved oxygen was about 8.4 mg/l, hydrogen sulfide was absent and low levels of ammonia nitrogen occurred (Chatterjee et al. 1967). An oxidation pond at Arcata, California, was found to be marginal for fish life (Hansen, 1967). In brackish sewage ponds in North Carolina, crabs and small fish were often found dead because of low oxygen concentration (Beeston, 1970). Lake Tunis, receiving sewage from 300,000 inhabitants, produced 147 kg per hectare of fish, but once a year there was a mass mortality because of dissolved oxygen deficiency and production of hydrogen sulfide (Stirn, 1972). Aschner et al. (1967) reported that carp raised in sewage ponds readily acquire objectionable taste and odor from their environment.

On the basis of the above reports, it is clear that sewage lagoons may be used as fish ponds, but the degree of success of fish culture cannot be assured in advance. The chemical characteristics of the sewage effluents in the ponds cannot be controlled, and their variance from day to day and from season to season affects the quality of the water that is ideal for the culture of fish.

The second method, adding sewage to fish ponds, is most commonly practiced in many parts of the world. With this method one can control and regulate the amount of sewage to be added to the pond and the technique is very effective in fish culture. It prevents deterioration of water quality in the ponds by over-enrichment by sewage and enables the fish to utilize the nutrients more fully.

Ganapati and Chacko (1950) reported that in India and Germany crude sewage must be diluted with four to ten volumes of freshwater, so that sufficient dissolved oxygen is provided for the survival of fish. In Calcutta, India, sewage was mixed with 100-120 million gallons of water over a period of 5-10 days to reduce the possibility of sudden pollution and maintain dissolved oxygen at 6.6 mg/l to 9.8 mg/l (Basu, 1950). For carp culture, Falck (1934) diluted sewage with three times its volume of ground water and Kisskalt and Lizhöfer (1937) diluted sewage five times with stream water. Bhaskaran (1952) added sewage to fish ponds at a rate which would maintain dissolved oxygen at 3 mg/l. Cludicus (1955) settled sewage in two chamber tanks, mixed it with five times its volume of freshwater, and discharged it into two fish ponds. Chatterjee et al. (1967) found that carp survived in the

diluted crude sewage lagoon which had a dissolved oxygen above 8.4 mg/l, was free of hydrogen sulfide, and low in ammonia. Prowse (1965) indicated that very diluted sewage supports good fish crops because of the beneficial use of inorganic nutrients.

The third method, conversion of sewage nutrient into algae and then discharge of them into fish ponds, actually is an extension of the current lagoon sewage treatment process by adding fish ponds to utilize algae produced for fish production and further sewage effluent purification. This method appears to be a very promising line for future research and development of aquaculture-sewage treatment system.

Since 1969 Woods Hole Oceanographic Institute has conducted extensive research in using sewage to grow marine algae and then to produce useful marine food organisms (Ryther, 1971; Ryther et al. 1972). The results of the studies which have been reported indicate that treated secondary sewage treatment plant effluents are a good and complete fertilizer for algae which form the base of the marine food chain (Dunstan and Menzel, 1971). The algae were successfully fed as food to marine bibalve mollusca (oyster, clam, and mussel) (Dunstan and Tenore, 1972). There is no adverse effect on the growth of the bibalves from sewage (Tenore and Dunstan, 1973). Sewage treatment-aquaculture systems can be made to remove sewage nutrients effectively and to produce rapid growth of useful marine organisms (Huguenin and Ryther, 1974).

At the Oklahoma State Department of Health, a preliminary project has been conducted since 1970 in attempt to remove algae and to upgrade the quality of sewage stabilization pond effluent (Coleman et al. 1974). The result of the study indicates that water quality greatly improved in the fish ponds to reach water quality standards, with great production of the stocked fish (channel catfish, fathead minnow, golden shiner, and Tilapia). In California, Allen and O'Brien (1967) and Allen et al. (1972) reared salmonid fish to migratory size in brackish water ponds receiving effluent from a sewage stabilization pond. The South Pacific Commission in Noumea discharged sewage into a narrow pond for purification and then fed it into the ponds for fish culture (Chan, 1972). They all obtained very satisfactory results.

Effects of sewage on yields in fish culture. Great yields have been reported in many parts of the world in this application of sewage in aquaculture. The Bavaria Power Company which owns two hundred acres of sewage fish ponds near Munich, Germany, produces 500 kg of carp per acre per year and makes great profit (Bardach, 1968; Randal, 1968). The fish production per man-year exceeds 30 tons, higher than those produced by intensive pig farming in developed nations around 25 tons of live pigs (Randal, 1968). In West Java, in the rapidly flowing streams containing high concentrations of sewage, carp production in bamboo cages yields 50 or more

kilograms of fish per square meter of cage surface or 500 metric tons per hectare (Bardach, 1968). In Taiwan, using sewage as fertilizer without supplemental feeding, milkfish ponds produce 2,400 to 2,600 kilograms per hectare per year (Bardach and Ryther, 1968). Thorslund (1971) grew two year old carp in undiluted sewage effluents of an activated sludge treatment plant and obtained over 1,000 kilograms per hectare without aritifcal feeding.

Various marine aquatic invertebrates, feeding chiefly on microscopic organisms, show production exceeding the best harvest from intensively managed fish ponds. In a Spanish estuary subjected to sewage fertilization 120 tons (meat weight) per hectare of mussels were produced by rope culture, much higher than the five tons per hectare produced in natural, relatively unfertilized water (Bardach and Ryther, 1968). Also in Japan 100 tons of oyster meats per hectare per year were produced with raft culture in eutrophied estuaries (Ryther et al. 1972). Ryther (1971) estimated that an algal farm of 50 acres, operated in conjunction with a shellfish farm of one acre, would be capable of providing tertiary sewage treatment for a population of 11,000 people, with a by-product of one-million pounds (meat weight) per year of commercially valuable shellfish. Ganapati and Chacko (1950) also estimated that one acre of fish pond receiving sewage of 1,000 people will produce 3,000 pounds of fish per year.

Coleman et al. (1974) reported the results of the study at the Oklahoma State Department of Health during five summer months. The fish grew rapidly and the weight of Tilapia increased from 4 pounds to 163 pounds, the channel catfish from 600 pounds to 4,400 pounds and golden shiner from 85 pounds to 535 pounds. Cost-benefit analysis indicated that the total estimated cost is 15 cents/1,000 gallons of domestic sewage, cheaper than the 40 cents/1,000 gallons by the present use of mechanical separation of algae from effluent. Cost recovery based on local market value of the fish was 2.3 cents/1,000 gallons of sewage. This suggests that using fish ponds to remove algae from the effluents of sewage stabilization is cheaper than present engineering methods.

Fish ponds for sewage purification. In many cases, the use of fish ponds is a very satisfactory method for the purification of sewage (Falck, 1934; Kisskalt and Lizhofer, 1937; Neess, 1949; Bozko et al. 1967; Bosshard, 1970). Actually utilization of sewage in aquaculture is in itself an advanced sewage treatment process with a valuable food as its by-product (Ryther et al. 1972). Cities as large as Munich, Germany, dispose of most of their sewage in this manner (Neess, 1949). The city of Tainan, the third largest city in Taiwan, disposes of most of its sewage into milkfish ponds; the amount of sewage available has been found actually insufficient for fish culture demands and chemical fertilizer has been added.

It has been found that the self-purification of sewage in fish ponds is more effective than in the stream (Schiemenz,

1955). Bozko et al. (1967) made comparative studies of five sewage treatment ponds of which three were stocked with fish and the other two were stocked with Daphnia. The ponds stocked with fish provided suitable conditions for phytoplankton development of Cladocera and Tendipedidae. The retention period of sewage in fish ponds for ten days gave the best results of sewage purification as judged by BOD and bacteria reduction. Other researchers have found that 40 hours retention of sewage in fish ponds would reduce by 40-50 percent BOD (Kisskiat and Lizhofer, 1937; Ganapati and Chacko, 1950), 99.6 percent of the total bacterial count 99.6 percent of E. coli, about 87 percent of turbidity, and 76 percent of ammonia (Kisskalt and Lizhofer, 1937). Ganapati and Chacko (1950) estimated that sewage from a population of 1,000 would require a fish pond of one acre.

In the sewage-algae-oyster-seaweeds system at the Woods Hole Oceanographic Institute, it was found that 95 percent of the influent inorganic nitrogen was assimilated by algae. The oysters in turn, removed 85 percent of the algae. The regenerated nutrients in oyster ponds (16-18 percent) were assimilated by seaweeds. The total inorganic nitrogen removal was 45-60 percent (Goldman et al. 1974). Comparison of feeding rates and conversion rates among mussels, oysters and clams in the experimental ponds indicated that the ecological efficiency (net production/ingested food) is highest for clams, lowest for mussels and medium for oysters (Tenore et al. 1973).

The results obtained by the Oklahoma Department of Public Health study indicated that a great purification of sewage stabilization ponds effluents was achieved in a series of fish ponds (Coleman et al. 1974). BOD was reduced from 184 mg/l to 6 mg/l, suspended solids (algae) from 131 mg/l to 6 mg/l, ammonia nitrogen from 12.6 mg/l to 0/12 mg/l, and fecal coliform from 3.05×10^6/100 ml to only 20/100 ml. Also fish grown in the ponds were free of pathogenic contamination and it was confirmed that the lagoon treatment of sewage has a very effective bacteriocidal action (Carpenter, 1974).

Present status of fish culture and sewage utilization. At present, the fishery industry in the United States as well as in many parts of the world, as Bowers (1966) stated, is much the most primitive of all protein producing activities. It is still based on virtually indiscriminate capture with little management of fish stock in the nature. Hunting of fish must give way to farming of stocks, if the yield of protein is to be increased. Among several possibilities for increasing the world supply of protein, is the development and expansion of aquaculture - the rearing of aquatic organisms under controlled conditions, using the techniques of agriculture and animal husbandry (Ryther and Bardach, 1968). Randal (1968) stated that fish farming could be a definite step in the direction of a solution to the food supply problem facing man today. We cannot afford to continue to accept what economists are fond of saying: that commercial fishing is

still in the hunting and gathering stage, and letting it go at that. In the United States there is competent talent in the relevant scientific and technological fields, but it has not been applied to aquaculture. In Maryland, except for a couple of small goldfish farms, there is virtually no fish culture research and industry.

Besides the primitive stage of our fishing industry, there is the arduous practice of sewage nutrient removal and chemical fertilization of fish ponds (Konefes and Bachmann, 1970; Ryther et al. 1972). It has become customary to consider sewage as one of the troublesome waste products of our society, which leads only to degradation of our aquatic environment through acceleration of eutrophication. In order to eliminate this problem much time, money, and effort has been devoted to research and construction of more and more sophisticated and expensive sewage treatment plants to remove nutritional elements of sewage. At the same time, fish culturists spend money to purchase chemical fertilizers containing the same nutritional elements as those in sewage and disperse them in their ponds to increase productivity. Even the fishery management agencies in most states in this country offer publications teaching how to use chemical fertilizers to increase fish production in farm ponds.

Considering the primitive stage of our commercial fishery, wasteful methods of sewage disposal and its deteriorating effects on natural aquatic systems and the possibile deficiency in protein supply in the future, research and development of aquaculture with utilization of sewage is urgent. Many questions and objections need to be answered and solved through research before the commercial operation of sewage-fish culture may be realized in Maryland as well as elsewhere in this country. These include problems of the biology of the fish in the ponds, energy flows in the ponds, pond engineering design, fish species selection for culture, public health, and marketing obstacles. The last three problems in particular must be solved, otherwise there is no chance for success in utilizing sewage in fish culture in this country. However, as Huguenin and Kildow (1972) stated, due to the required long lead time for planning, research and development, the time for choice is now.

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