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Additive Effects of Chlorinated Biocides and Water Temperature on Fish in Thermal Effluents with Emphasis on the Great Lakes

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ABSTRACT: We reviewed the literature on the effects of chlorine on selected Great Lakes fishes during the summer when chlorine is used to control biofouling in cooling systems at power generating stations. Mortalities of fish are usually not solely due to chlorine toxicity but to complex additive functions and interactions of various stressors, in particular temperature. Elevated temperature appears to be important in magnifying the effects of the toxicity of chlorine to fish. When chlorination is used at temperatures near the thermal maxima, but not sufficiently high to exclude fish, high mortality rates can be expected. Most of the fish that lose equilibrium during exposure do not survive. Fish exposed to sublethal levels of chlorine become lethargic and often gulp air and frequently suffer increased predation pressures from birds and other fish. Additionally, hematological and biochemical disturbances, and potentially irreversible gill damage, may impair the lifetime fitness of fish exposed to chlorine. The sensitivity of different species of fish to chlorine toxicity varies widely. As such, chlorination regimes should be evaluated on a daily basis to account for differences in species composition and water temperatures. Most of the chlorine exposure concentrations reported in the literature are for 50% mortality, but the highest concentration resulting in no mortality, loss of equilibrium, or sublethal effects, is a more appropriate value for management and conservation. We also advocate comprehensive ecological risk assessments to determine the scope of impact on all organisms, not just fish. Only a series of *in situ* and laboratory studies for each situation will provide biologically meaningful values and the basis for relevant regulations.

KEY WORDS: toxicity, thermal effluents, biofilm, mortality, tolerance.

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I. INTRODUCTION

Biofouling is the process where living organisms and a combination of organic and inorganic matter attaches to a substrate, although the exact mechanisms are poorly understood. The process initially requires slime-forming microorganisms, usually including capsulated bacteria and protozoa. These tightly adherent deposits trap inorganic particulate matter in the original slime layer (Burton and Liden, 1977) inhibiting heat transfer when present in condenser cooling tubes, reducing the efficiency of the power production process (Cairns et al., 1979). The invasion of zebra mussels (*Dreissena polymorpha*) has also led to increased needs for the use of biocides, especially in the laurentian Great Lakes.

Various biocides have been used, but chlorine is the most highly regarded by the utility industry (White 1975) for several reasons (Hall et al., 1981):

- 1. Chlorine effectively controls biofouling at reasonable concentrations.
- 2. Readily available, at low cost.
- 3. Availability of simple and controllable feed systems at low capital costs.
- 4. Minimal maintenance feed system.
- 5. Relatively short residual times.

Others have reviewed the ecological effects of power plant cooling water chlorination in freshwater (Brungs, 1973; Mattice and Zittel, 1976; Hall et al., 1981; Howells, 1983) and marine environments (Hall et al., 1982). These reviews include reference to numerous types of organisms ranging from phytoplankton to marine mammals. However, there is an absence of a recent synthesis of the ecological impacts of chlorine and chlorine residuals from laboratory and field studies on freshwater fish.

This review has three objectives. First, we synthesize the results of research on the ecological impacts of chlorine and chlorine residuals, originating from the use of chlorine as an antibiofouling agent in power plant cooling loops on selected species of freshwater fish in the Great Lakes. The species selected represent a diversity of life history strategies, apparent sensitivities, and sizes of fish common in the Great Lakes region. We also provide a general discussion on the effects of chlorine on fish, based on information about other species of fish from beyond the Great Lakes, to broaden the usefulness of this review. The second objective is to codify the links between temperature and the ecological effects of chorine on fish. This was deemed important because research suggests that a variety of environmental parameters may alter the ecological effects of chlorine on fish. In particular, temperature has been cited repeatedly as a significant cofactor. To this end, we tabulated various thermal values relevant to summer chlorination for selected Great Lakes fishes.

Our third objective is that of assessing the current status of understanding, provide management directions, and to recommend future research directions for understanding and minimizing the ecological effects of chlorine from condenser cooling systems biofouling control.

II. AQUATIC CHEMISTRY OF CHLORINATION, TEMPERATURE, AND DEFINITIONS

The chemistry of chlorine in natural waters is complex (White, 1972). Toxicological studies have primarily concentrated on the two forms which are apparently the most toxic to aquatic life (Heath, 1977). The first form, commonly referred to as free chlorine, is the portion of chlorine injected into the water that remains as molecular chlorine (Cl₂), hypochlorous acid (HOCl), or a hypochlorite ion (OCl-) after chlorine demand has been satisfied. The second form, called monochloramine or combined chlorine (NH₂Cl), is the portion of chlorine injected into the water that remains combined with ammonia or nitrogenous compounds after the chlorine demand has been satisfied. The sum of the free and combined forms generally is referred to as total residual chlorine (TRC). The relative proportion of free and combined chlorine present following a chlorination dose depends primarily on the concentration of nitrogenous materials in the water. The chlorine demand is defined as the difference between the amount of chlorine added to the water and the amount of TRC that remains at the end of a specified period (Mattice and Zittel, 1976).

Data on the relative toxicity of free and residual chlorine have been variable and contradictory. In general, however, most research reports suggest that free chlorine is the most toxic form (Merkens, 1958; Eren and Langer, 1973). The biocidal efficiency of chlorine depends on the amount of hypochlorous acid (HOCl) in the water, because HOCl can penetrate cells and react with cell enzymes (Moore, 1951). It is this property that makes chlorine particularly toxic to fish and other aquatic organisms.

Brungs (1973) and Cairns et al. (1975a,b, 1978) reviewed the influence of temperature on the toxicity of chemicals to aquatic organisms. These investigators concluded that temperature may have a marked effect on the toxicity of chlorine to fish, but also noted that direct studies supporting this supposition were lacking. Based on the mechanism of chlorine toxicity to fish, the authors surmised a significant influence of temperature on the toxicity of chlorine to fish.

For this review, high-acclimation temperatures representative of summer water conditions and thermal extremes in heated effluents were reported. The upper incipient lethal temperatures represent the temperatures above which mortality is greater than 50%. A standard 7-d exposure period is usually used. This temperature was chosen because it is analogous to the LC50. The critical thermal maximum is a temperature that is lethal to all fish allowed to remain at or above that temperature. This temperature reflects the true lethal limit. The upper avoidance temperature is an avoidance threshold, above which fish tend to move to adjacent, cooler-temperature control areas. The upper avoidance temperature is relevant to chlorine toxicity, because if a fish avoids this temperature in a discharge canal they would also reduce their exposure to chlorine, which is relevant with heated effluents. The final preferendum is a temperature range under which fish will commonly congregate in an infinite temperature gradient. These definitions are discussed further in Wismer and Christie (1987).

III. GENERALIZED BEHAVIORAL RESPONSE OF FISH TO CHLORINE

Studies of behavioral responses of fish to chlorine are important in understanding if and how fish react to this toxicant (Cherry and Cairns, 1982; Gray, 1983). In general, fish respond to chlorine exposure in a series of phases, each of which has more or less distinct characteristics. In the first phase, fish become restless and frequently gulp air at the surface. Basch and Truchan (1976) observed gulls feeding on small fish floundering on the surface during a chlorination event at a Lake Michigan power plant. A general lethargy usually follows and can last throughout the exposure period (Zeitoun, 1978b; Seegert et al., 1979). When fish become lethargic, they may appear dead; they have shallow respiration rates and often are immobile. Fish that succumb to chlorine sink to the bottom without convulsive movements. Their respiration slows and finally stops. Fish killed due to chlorine exposure usually assume a natural posture. However, some fish, such as sauger (Stizostedion canadense), die with the mouth gaping and the gills flared (Seegert et al., 1979). High chlorine concentrations also may result in the loss of large amounts of mucous, particularly for common carp (*Cyprinus carpio*) (Seegert et al., 1979).

The equilibrium loss response (EL50) seems particularly important for minimizing sublethal effects of chlorine exposure and reducing losses by predation. Chinook salmon (*Oncorhynchus tshawytscha*) exposed to elevated temperature and chlorine required a 55% reduction in the lethal time (LT50) to ensure that equilibrium loss did not occur (Stober and Hanson, 1974). Salmon generally did not recover from chlorine exposure once equilibrium loss occurred.

IV. GENERALIZED PHYSIOLOGICAL EFFECTS OF CHLORINE

Zeitoun (1977, 1978a,b) provided insights into the biochemical responses of rainbow trout (*Oncorbynchus mykiss*) to chlorine. Hematological analyses suggested that chlorine was not the primary cause of observed stress. Of parameters measured, only hematocrit and hemoglobin concentrations differed between caged and control fish. The generalized response of increases in the proportion of red blood cells in the blood of test fish, and the subsequent increase in hemoglobin could be the result of various stress factors and are not specifically due to chlorine. Sprague (1971) reported the same problem with nonspecificity. Rainbow trout exposed to sublethal concentrations of total residual chlorine had less hepatic ribosomal translational ability (Orvos et al., 1986). These results may provide an explanation for the altered plasma proteins noted in other studies.

The ventilation rates of bluegill (*Lepomis macrochirus*) exposed to chlorine decrease initially, at low concentrations of chlorine, but increase at higher levels of chlorine (Miller et al., 1979). Some investigators also have reported that the coughing rate of fish increases 15-fold in response to intermittent chlorine concentrations of 0.4 to 0.5 mg/l (Bass and Heath, 1977). These authors also reported that the arterial PO₂ declined by about 60% during the first pulse peak. Work by Block (1977) has shown that the gill is the chief target organ for chlorine toxicity. The authors surmised that chlorine kills fish by internal hypoxia induced by gill damage. Histopathological studies (Bass et al., 1977) have indicated that sublethal concentrations of chlorine cause moderate gill hyperplasia and swelling of the lamellar epithelial cells. These studies highlight the numerous sublethal effects of chlorine that may alter the fitness of the fish. Studies investigating the long-term effects of chlorine exposure are nonexistent.

Wedemeyer and Ross (1973) suggest that the stress response and susceptibility of fish to chlorine may be related to diet. However, fish living in thermal discharge canals usually have abundant food resources (Coutant, 1970), which may negate diet as a major cofactor in the ecological effects of chlorine. Additional evidence presented by Marking et al. (1984) suggests that diet appears to have little influence on the sensitivity of young rainbow trout to chlorine. Hettrick et al. (1984) reported that chlorine did not influence the susceptibility of striped bass (*Morone saxatilis*) to *Vibrio anguillarum*, a pathogenic bacterium.

V. REPEATED EXPOSURES AND ACCLIMATION TO ELEVATED CONCENTRATIONS OF CHLORINE

Fish can acclimate to a variety of conditions, including temperature and some toxicants. In some situations, repeated or continuous exposures can alter the sensitivity of fish to subsequent exposures. Several researchers have noted that fish can live in areas where the TRC concentrations have been deemed lethal to fish (Stewart et al., 1996; Lotts and Stewart, 1995). Only one study has used a combination of field and laboratory studies to provide experimental insight into this phenomenon. Lotts and Stewart (1995) assessed the acclimation of fish to chlorine using time-todeath for fish exposed to a single lethal regime of TRC. These in situ experiments involved capturing striped shiners (Luxilus chrysocephalus) and central stonerollers (Campostoma anomalum) in areas where TRC was high and in other areas where it was undetectable. Fish from both sites were caged at each of the two sites. At the site where TRC concentrations were high, fish captured from near the site survived 2 to 4 times longer than fish captured from a site further downstream where exposure to TRC was negligible. In laboratory experiments, the authors exposed golden shiner (Notemigonus crysoleucas) to TRC over weekly stepwise increments, then challenge tested the minnows with a lethal dose of TRC. Fish previously exposed to TRC survived longer than nonexposed fish. The magnitude of the mean time to death of the fish increased with the previous acclimation TRC concentration and duration of exposure. This study suggests that several minnow species are capable of acclimating fairly rapidly to TRC. To date, however, there is no evidence that any of the fish species that are more thoroughly reviewed herein can acclimate to TRC.

In another study, goldfish (*Carassius auratus*) responses to TRC indicated that a chlorination procedure should not exceed two to three

exposures per day (exposure episodes were 15 to 30 min), with TRC concentrations being 0.5 to 0.75 mg/l (Dickson et al., 1977). These authors surmise that acute mortality would not occur for goldfish or other species with tolerances similar to goldfish. Goldfish, however, are generally considered to be tolerant to many toxic conditions. Thus, these concentrations are likely greater than what would be expected for most Great Lakes fishes. Temperatures during this study ranged between 12 and 22°C. Currently, it is unclear whether the effects of repeated exposures to TRC increase the sensitivity of fish to subsequent chlorine exposures in all cases.

VI. INTERPLAY OF TEMPERATURE AND CHLORINE

Many studies have investigated the interactive effects of chlorine and water temperature on fish. Some of the more common Great Lakes fishes are discussed below. Here we present what is known about some other fish species, and the generalized effects of temperature on fish as it relates to chlorine toxicity.

Within limits, most biological process accelerate with increased temperatures (Schmidt-Nielsen, 1990). An increase in temperature typically results in an increase in metabolic rate, which generally causes a proportional increase in cardiac output (Mirkovic and Rombough, 1998). An increase in cardiac output with increasing temperature has been shown for several fish species and is due in part to an increase in heart rate caused by an increase in membrane permeability of pacemaker fibers (Randall, 1968, 1970; Farrell and Jones, 1992) and changes in neural and humoral regulation (Farrell, 1984). All species have an upper limit of metabolism, so an increase in resting cardiac output and heart rate with an increase in temperature would reduce the metabolic scope available to respond to a perturbation. The mechanism behind this relationship is due to in the mechanical limits of the heart. Cardiac ejection is much higher in fish than in mammals (Farrell, 1991). In rainbow trout, this value can be nearly 100% of ventricular volume. Consequently, stroke volume is determined almost solely by enddiastolic volume and therefore limited by cardiac filling time. As heart rate increases, cardiac filling time decreases, which causes stroke volume to decrease. Eventually, as temperature continues to increase, cardiac output, and consequently metabolic rate, levels off (Schreer et al., in press; Schreer and Cooke, in review). Therefore, when fish are exposed to chlorine at higher temperatures less of their metabolic scope is available for this added perturbation. Consequently, at higher temperatures, fish have a more limited capacity to adjust to this stressor and are more likely to succumb to the effects of a toxicant such as chlorine.

Bass and Heath (1977) investigated the effects of intermittent chlorination on bluegill and report on the strong interaction with temperature. At the lowest peak TRC concentration of 0.21 mg/l, no deaths occurred at any temperature. However, the fish did appear to be in distress during the chlorination, especially at 32°C. Fish were observed coughing at the surface and were less responsive to the observer's presence during the chlorine pulse than at lower temperatures. Mortality was less than 15% at 0.31 mg/l of peak TRC. Some of the bluegill exposed to 0.42 or 0.52 mg/l peak TRC became lighter in color after exposure, and died shortly thereafter. A notable effect of temperature occurred at a peak TRC concentration of 0.52 mg/l where the LT50 was 74 h at 6°C and decreased to about 20 h at 32°C. These LT50 values correspond to the free chlorine concentrations of 0.44 and 0.39 mg/l, respectively.

The synergistic effects of temperature and chlorine were also evident in the hatching, embryonic success, and ontogeny of mummichog (*Fundulus beteroclitus*) (Middaugh et al., 1978). Similar work by Burton et al. (1979) revealed a major interaction between TRC, change in temperature, and exposure time on the mortality of eggs and larvae of striped bass.

In another study, spotted bass (Micropterus punctatus) and rosyface shiner (Notropis rubellus) were exposed to successively doubled doses of TRC from 0.025 to 0.80 mg/l at five temperatures, ranging from 6 to 30°C (Cherry et al., 1977). Both species generally avoided higher concentrations of TRC, and both species avoided TRC at a concentration of 0.05 mg/l when acclimated and tested at 6°C. Avoidance at 24 and 30°C occurred at 0.20 mg/l. After initial avoidance response, the rosyface shiner continued to avoid the remaining higher residual levels. Spotted bass showed less avoidance, or none at all. Similar behavioral responses were observed when fish were exposed to a single concentration (0.05 or 0.10 mg/l) for three successive 10-min observation periods. Relative to TRC or combined residual chlorine at a variety of acclimation temperatures, the spotted bass and rosyface shiner appeared to avoid higher concentrations than other species. For example, rainbow trout (as reviewed below), one of the more sensitive species, avoided TRC concentrations as low as 0.001 mg/l (Sprague and Drury, 1969).

Cherry et al. (1977) report that the avoidance response of spotted bass and rosyface shiner was closely related to the calculated hypochlorous acid (HOCl) fraction of the TRC. The relative amount of hypochlorous acid declined as the pH and acclimation temperature increased. The authors concluded that hypochlorous acid may be the most important constituent within TRC to influence the avoidance response.

In a study of pink salmon (*Oncorhynchus gorbuscha*) and Chinook salmon in seawater, Stober and Hanson (1974) report that a decrease in the tolerance of both species to residual chlorine was demonstrated with increasing temperature and exposure time. The authors concluded that although the addition of heat increases the rate of salmon mortality, chlorine was the overriding lethal factor.

VII. ECOLOGICAL EFFECTS

Qualitative changes in fish community structure result under conditions of toxic stress. Localized species diversity is reduced and stress-tolerant species become more predominant (Dickson et al. 1977). Over time, this may happen on a scale larger than the discharge area. More often it will be limited to the area under which chlorine concentrations are detectable. The true ecological consequences of these changes have yet to be documented (Sarokin and Schulkin, 1992).

VIII. SELECTED GREAT LAKES FISHES

Here we provide an overview of chlorination experiments on several common Great Lakes fishes. We also provide information on the thermal tolerances and preferences of these species during the summer months. Specifically, we provide information on the upper incipient lethal temperature (UILT), critical thermal maximum (CTMax), upper avoidance temperature, and final preferred temperature. Taken together, these data can be used by fisheries managers, regulators, and the utility industry to help identify when chlorination may be injurious or lethal to fish.

A. SMALLMOUTH BASS

1. Chlorine (Table 1)

Data on chlorine effects of smallmouth bass (*Micropterus dolomieu*) are limited. A stream study by Tsai (1971) suggested that concentrations of 0.1 mg/l were enough to eliminate smallmouth bass from streams. Cherry et al. (1977) reported that smallmouth bass exhibited avoidance responses at temperatures between 6 and 30°C for concentrations of 0.05

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TABLE 1	Summary

emperature	Concentration	Chlorine	Duration	Effect	Reference
°C)	(mg/l)	Form	(min)		
	0.1	TRC		Absent in streams	Tsai 1971
	0.05 – 0.20 (6-30C)			Avoidance response	Cherry et al. 1977
	0.5	Residual (Free)	900	50% mortality	Pyle 1960
lank cells in	licate data not reported	(for all tables). Dat	a in tabular form ha	ve been condensed and sum	narized

from original sources. It is thus strongly recommended that readers requiring detailed values consult the original sources.

to 0.20 mg/l. Pyle (1960) reported 50% mortality of smallmouth bass after 15 h at a TRC of 0.5 mg/l.

2. Temperature (Table 2)

The final preferred summer temperatures of smallmouth bass range between 28 and 31°C (Coutant, 1977a). Smallmouth bass avoid temperatures above 33 to 35°C (Coutant, 1977a). Upper incipient lethal temperatures are approximately 35 to 37°C, and the critical thermal maxima for the species is 36°C (Reutter and Herdendorf, 1976; EPA, 1974; Ellis, 1984).

B. RAINBOW TROUT

1. Chlorine (Table 3)

Rainbow trout are perhaps the best studied fish with reference to sensitivity to chlorine. Zeitoun (1978a) conducted studies on rainbow trout survival during intermittently chlorinated heated effluents at a power generating station on Lake Michigan. Caged adult rainbow trout were held at three locations in the discharge canal. Controls were held in the forebay at ambient lake temperatures, and in a field trailer that was artificially heated to mimic the temperatures in the discharge canal. Monthly, fish in the discharge canal were exposed to a daily 30-min chlorination episode over a 4-d period. Results suggested that mortality appeared to decline as the location of the station was further downstream from the point of chlorine release. Mortalities appeared to increase with time of exposure and stabilized after the third day of study.

Eight covariates were deemed to be responsible for the mortality of caged fish, with the interaction of temperature and oxygen being the most significant. During colder months, gas bubble disease was prevalent and nearly 50% mortality was observed within 30 h. The heated control groups also had moderate mortality, which the investigators attributed to water temperature and dissolved oxygen limitations. The authors concluded that the mortalities of caged rainbow trout were not due simply to chlorine, but to complex additive functions and interactions of various stressors. Based on their studies, the authors recommend a conservative 0.25 to 0.3 mg/L TRC per 54 min limit for minimizing mortality of rainbow trout in the dynamic environmental conditions of a heated effluent.

The response of rainbow trout to intermittent chlorination also was studied by Brooks and Seegert (1977). Fish were tested at 5-degree temperature increments ranging from 10 to 20°C. Single exposure (30-

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TABLE 2 Summary of Su	ummer Thei	mal Toleranc	es and Prefer	ences for Sn	nallmouth Bas	S
Size or Age	UILT	CTMax	Upper Avoidance	Final Preferred	Comments	Reference
Juvenile	35					EPA 1974
Adult	37					Ellis 1984
		36.3				Reutter and Herdendorf 1976
	35					Cherry et al. 1977
Small				28		Coutant et al. 1977a
УОҮ				31		Coutant et al. 1977a
УОҮ			35			Coutant et al. 1977a
			33	31.3		Coutant et al. 1977a
				30-31		Wrenn 1980

Note: All temperatures in °C.

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TABLE 3 Summary of Toxici	Tomacontruc

TemperatureConcentrationChlorine $(^{\circ}C)$ (mg/l) FormForm $(^{\circ}C)$ (mg/l) Form 10 $(^{\circ}C)$ 0.08 All 10 0.001 Free 10 0.001 Free 222 0.02 0.05 Total 720 0.02 0.05 TRC 20 0.02 0.05 TRC 20 14.9 0.66 FAC, TRC 540 14.9 0.92 FAC, TRC 540 14.9 0.35 FAC, TRC 540 14.9 0.35 FAC, TRC 20 14.9 0.35 FAC, TRC 241 14.9 0.05 TRC 241 14.9 0.05 TRC 241 12 0.05 TRC 241 12 0.05 TRC 241 17 0.12 Free 481 17 0.12 Free 481 0.09 Free 481 0.09 Free 481 0.09 Free 481 0.09 Free 481					
(°C) (mg/l) Form 0.08 All Free 10 0.001 Free 10 720 0.02 0.02 Total 720 0.02 0.05-0.10 TRC 220 20 0.50 TRC 220 20 0.50 TRC 20 14.9 0.66 FAC,TRC 540 14.9 0.65 FAC,TRC 540 14.9 0.55 TRC 20 14.9 0.55 FAC,TRC 540 14.9 0.55 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.35 FAC,TRC 540 12 0.05 TRC 241 12 0.05 TRC 241 12 0.06 Free 481 17 0.10 TRC 241 17 0.12 Free 481 17 0.12	ture Concentration	Chlorine	Duration	Effect	Reference
0.08 All 0.001 Free 0.02 Total 0.02 Total 0.02 TRC 0.7 Free 0.7 Free 0.7 Free 0.7 Free 0.7 Free 14.9 0.6 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.92 14.9 0.93 14.9 0.10 12 0.05 13 0.10 14.9 0.10 17 0.12 17 0.12 17 0.12 17 0.12 17 0.12 17 0.12 17 0.12 17 0.12 17 <t< th=""><th>(mg/l)</th><th>Form</th><th>(min)</th><th></th><th></th></t<>	(mg/l)	Form	(min)		
0.001 Free 10 0.02 0.02 Total 720 0.7 Free 22 0.7 Free 22 0.7 Free 22 0.7 Free 22 20 0.05-0.10 TRC 20 20 0.50 TRC 20 14.9 0.6 FAC,TRC 540 14.9 0.92 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.35 FAC,TRC 540 12 0.05 TRC 24 12 0.05 TRC 540 12 0.05 TRC 48 17 0.10 TRC 48 17 0.12 Free 48 17 0.12 Free 48	0.08	All		7d LC50	Merkens 1958
0.02 Total 720 20 0.7 Free 222 20 0.50 TRC 220 20 0.50 TRC 220 20 0.50 TRC 220 20 0.50 TRC 20 14.9 0.66 FAC,TRC 540 14.9 0.35 FAC,TRC 540 12 0.05 TRC 24 12 0.06 Free 48 17 0.10 TRC 24 17 0.12 Free 48 20 87.165 786 30	0.001	Free	10	Slight avoidance	Sprague and Drury 1969
0.7 Free 222 20 0.05-0.10 TRC 20 20 0.50 0.50 TRC 20 14.9 0.6 FAC,TRC 540 14.9 0.6 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.05 TRC 24 17 0.06 Free 48 17 0.10 TRC 48 20 87.165 TRC 30	0.02	Total	7200	50% mortality	Basch et al. 1971
20 0.05-0.10 TRC 20 20 0.50 TRC 20 14.9 0.6 FAC,TRC 540 14.9 0.92 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.44 FAC,TRC 540 12 0.05 TRC 24 12 0.06 TRC 24 17 0.10 TRC 48 17 0.12 Free 48 20 0.82-1165 TRC 30	0.7	Free	2220	100% mortality	Ebeling 1931
20 0.50 TRC 20 14.9 0.6 FAC,TRC 540 14.9 0.92 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.05 TRC 24 12 0.05 TRC 24 12 0.00 Free 48 17 0.12 Free 48 20 082-165 TRC 30	0.05-0.10	TRC	20	1 st avoidance response	Schumacher and Ney 1980
14.9 0.6 FAC,TRC 540 14.9 0.92 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.44 FAC,TRC 540 12 0.05 TRC 24 12 0.05 TRC 24 12 0.05 TRC 48 12 0.09 Free 48 17 0.12 Free 48 20 0.82-165 TRC 30	0.50	TRC	20	total avoidance	Schumacher and Ney 1980
14.9 0.92 FAC,TRC 540 14.9 0.35 FAC,TRC 540 14.9 0.44 FAC,TRC 540 12 0.05 TRC 24 12 0.05 TRC 24 12 0.05 TRC 24 12 0.06 TRC 24 13 0.10 TRC 24 14 0.10 TRC 48 17 0.12 Free 48 20 0.82-165 TRC 30	0.6	FAC,TRC	540	100% mortality	Osborne et al. 1981
14.9 0.35 FAC,TRC 540 14.9 0.44 FAC,TRC 540 12 0.05 TRC 24 18 0.10 TRC 24 12 0.05 TRC 24 18 0.10 TRC 24 17 0.09 Free 48 17 0.12 Free 48 20 0.82-1.65 TRC 30	0.92	FAC,TRC	540	100% mortality	Osborne et al. 1981
14.9 0.44 FAC,TRC 24 12 0.05 TRC 24 18 0.10 TRC 48 12 0.09 Free 48 17 0.12 Free 48 20 0.82-165 TRC 30	0.35	FAC, TRC	540	40% mortality	Osborne et al. 1981
12 0.05 TRC 18 0.10 TRC 12 0.09 Free 17 0.12 Free 20 0.716 TRC	0.44	FAC,TRC	24 hr	50% mortality	Osborne et al. 1981
18 0.10 TRC 12 0.09 Free 48 17 0.12 Free 48 20 0.82-1.65 TRC 30	0.05	TRC		Avoidance	Cherry et al. 1982
12 0.09 Free 48 17 0.12 Free 48 20 0.7-1.65 TRC 30	0.10	TRC		Avoidance	Cherry et al. 1982
17 0.12 Free 48 20 0.82-1.65 TRC 30	0.09	Free	48 hr	50% mortality	Heath 1977, 1978
20 0.82-1.65 TRC 30	0.12	Free	48 hr	50% mortality	Heath 1977, 1978
	0.82-1.65	TRC	30	100% mortality	Brooks and Seegert 1977
12 0.06 Free 96	0.06	Free	96 hr	50% mortality	Heath 1977, 1978
17 0.09 Free 96	0.09	Free	96 hr	50% mortality	Heath 1977, 1978

min) LC50 values were 0.99 mg/l at 10°C and 0.94 mg/l at 15°C. Two groups of trout tested at 20°C had lower (30-min) LC50 values: 0.60, and 0.43 mg/l. No mortality occurred at concentrations <0.65 mg/l at 10 °C, or at <0.54 mg/l at 15°C. At 20°C, no mortality was observed at 0.30 and 0.45 mg/l. Three successive 5-min exposures to TRC at the LC50 values were 2.87 and 1.65 mg/l at 10 and 20°C, respectively. Rainbow trout exhibited rapid mortality in all tests except the 10°C triple exposure series: in that treatment, mortality occurred at 12 to 24 h.

When exposed to lethal or near lethal concentrations of chlorine, rainbow trout initially swam near the surface of the water. The fish also were described as being lethargic. In this study, the authors recommended a mean maximal TRC value of 0.668 mg/l. It should be noted that this concentration is based on the short-term exposures. The maximal TRC value would likely decrease at longer exposure durations.

Brooks and Bartos (1984) studied the effects of exposure duration to free and combined chlorine on rainbow trout at 25°C. The experiments consisted of single 15-, 30-, and 120-min exposures, and four successive 30-min exposures, spaced at 2-h intervals. Monochloramine was the least toxic and hypochlorous acid was the most toxic, based on LC50 values. The authors noted a significant difference in toxicity between each of the time exposures, with the 120-min exposure being almost three times more toxic than the 15-min tests.

A field study by Osborne et al. (1981) suggested that rainbow trout exhibited 100% mortality when exposed to chlorine at concentrations greater than 0.6 mg/l. A 1 d LC50 concentration of 0.44 mg/l was calculated for 14.9°C water.

Heath (1977) exposed fingerling size rainbow trout three times daily for up to 7 d to pulses of either free chlorine or monochloramine at 5, 12, and 24°C. At 12°C, the trout were more sensitive than at 5 or 24 °C to free chlorine pulses. Free chlorine was 8 to 14 times more toxic than the combined form. Data indicate that trout are highly sensitive to free chlorine. During a week of exposure to 0.04 mg/l pulses, approximately 10% of the trout would be killed. Pulses of 0.15 mg/l killed all of the fish. It should also be noted, however, that when no free chlorine was present, no mortality occurred up to total concentrations of 0.4 mg/l.

Studies by Schumacher and Ney (1980) examined the avoidance response of rainbow trout to 20-min single dose chlorination at 20°C. Fish began to move downstream at total residual chlorine values of 0.05 mg/l. Approximately 95% of the fish had moved downstream when total residual chlorine values reached 0.5 mg/l. Percentage of fish in the discharge declined linearly as total residual chlorine concentrations rose.

2. Temperature (Table 4)

The final preferred summer temperatures of rainbow trout range between 15 and 19°C (Coutant, 1977a; Spigarelli and Thommes, 1979). Rainbow trout avoid water that exceeds a temperature of about 20°C (Spigarelli and Thommes, 1979; Coutant, 1977a). The upper incipient lethal temperature is about 23°C, and the critical thermal maximum is 26.2°C (Brown, 1974; Houston, 1982).

C. FRESHWATER DRUM

1. Chlorine (Table 5)

Seegert et al. (1979) tested the effects of monochloramine on freshwater drum (Aplodinotus grunniens) at 10 and 20°C. The exposure regime consisted of four 40-min exposures administered at 0.5-h intervals over 24 h. In general, there was an inverse relationship between temperature and the LC50 concentrations. The LC50 values were 2.45 and 1.75 mg/ l for 10 and 20°C, respectively. The maximum concentrations causing no mortality were 1.73 and 1.48 mg/l for 10 and 20°C, respectively. Complete mortality (100%) occurred at concentrations of 2.84 and 1.94 mg/ l at 10 and 20 °C, respectively. Only 1% of the freshwater drum that lost equilibrium recovered. Mortalities were considerably more rapid at 20°C than at 10°C. Insufficient numbers of fish were obtained to complete full replicates and determine the values for the 30°C test group. However, at three concentrations tested (between 0.5 and 0.9 mg/l), no mortality occurred. A concentration of 1.05 mg/l caused 25% mortality, suggesting a LC50 concentration >1.05 mg/l. No mortality would likely be observed below 0.9 mg/l.

Although freshwater drum appeared moderately tolerant to chlorine in the study by Seegert et al. (1979), this species is still regarded as being generally sensitive to handling. For example, Harman et al. (1980) studied the physiological responses of freshwater drum to capture by commercial seine. These fish exhibited extensive hematological disturbance similar to fish that had been exercised for 40 min. The fish were difficult to maintain in the laboratory, and half of the fish expired from the cannulation procedure.

Reutter and Herdendorf (1976) noted that freshwater drum were about as difficult to maintain in laboratory conditions as alewife and gizzard shad, two other sensitive species. Other researchers have systematically studied the sensitivity of this species in an attempt to reduce transport mortality. Johnson and Metcalf (1982) report delayed mortality rates ranging from 80 to 100%. Late-summer water temperatures resulted

Final Comments Reference	eferred	Point Beach, Lake Michigan Spigarelli and Thommes 1979	Point Beach, Lake Michigan Spigarelli and Thommes 1979	5 Lake Michigan Coutant 1977a	19 Lab Coutant 1977a	Brown 1974	Great Lakes Brown 1974	Houston 1982	
CTMax Up	Avoid	22	20-21		22	26.7		26.2	r :
UILT						23.3-25.6	25-26		eratures are in °C
Size or Age		Adult	Large adult	Adult	Fingerlings	Young		Fingerling	Note: All temp

 TABLE 4

 Summary of Summer Thermal Tolerances and Preferences for Rainbow Trout

 Summary of Summer Thermal Tolerances and Preferences for Rainbow Trout

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TABLE 5 Summary of Toxic	sity of Chlorine to F	-reshwater Drum			
Temperature	Concentration (mo/l)	Chlorine Form	Duration (min)	Effect	Reference
10	1.73	Monochloramine	4 X 40 min	No mortality	Seegert et al. 1979
10	2.45	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
10	2.84	Monochloramine	4 X 40 min	100% mortality	Seegert et al. 1979
20	1.48	Monochloramine	4 X 40 min	No mortality	Seegert et al. 1979
20	1.75	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
20	1.94	Monochloramine	4 X 40 min	100% mortality	Seegert et al. 1979
30	0.0	Monochloramine	4 X 40 min	No mortality	Seegert et al. 1979
30	1.05	Monochloramine	4 X 40 min	25% mortality	Seegert et al. 1979

in lower mortality rates. These authors also noted that fish tagged with jaw tags had significantly more mortality than nontagged controls, again highlighting the sensitivity of this species. In general, the capture and handling mortality of freshwater drum seems to be an overriding factor in field or laboratory studies.

2. Temperature (Table 6)

The final preferred summer temperatures of freshwater drum range between 22.2 and 30.3°C (Coutant, 1977a). Freshwater drum attempt to avoid temperatures above 30°C (Coutant, 1977a). The upper incipient lethal temperature is approximately 32.8°C, and the critical thermal maximum is 34 °C.

D. CHANNEL CATFISH

1. Chlorine (Table 7)

Brooks and Bartos (1984) studied the effects of exposure duration to free and combined chlorine on channel catfish (*Ictalurus punctatus*) at 30°C. The experiments consisted of single 15-, 30-, and 120-min exposures, and four repeated 30-min exposures at 2-h intervals. Responses of channel catfish to the four exposure conditions revealed significant differences among treatments for each chlorine compounded test. The 120-min and 4×30 -min exposures were twice as toxic as the 15-min exposure, except for the hypochlorite ion experiments that showed a 5.5-fold increase in toxicity between the short- and long-duration exposures. Monochloramine was the least toxic form of chlorine tested, and hypochlorous acid and dichloramine were equally toxic in channel catfish.

Heath (1977) exposed fingerling channel catfish three times daily for up to 7 days to pulses of either free chlorine or monochloramine at 5 and 24°C. At 24°C, the catfish may have been slightly more sensitive than at 5°C to pulses of free chlorine. However, the LC50s for free chlorine and monochloramine did not differ much. At higher concentrations of free chlorine, the median lethal times were statistically shorter at 24°C than at 5°C, indicating a more rapid toxicological action. The concentrations resulting in no mortality were very low. Even a trace of free chlorine in the chlorination pulses may cause some mortality of this species. The concentration resulting in no mortality after 100 h was approximately 0.12 mg/l; the 120-h LC50 was 0.25 mg/l.

Seegert et al. (1979) tested the effects of monochloramine on channel catfish at 10, 20, and 30°C. The exposure regime consisted of four 40-min exposures administered at 0.5-h intervals over 24 h. These authors

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e or Age UI t 32.8 32.8	LT CTMa 34	x Upper Avoidance	Final Preferred	Comments Lab	Reference Reutter and Herdendorf 1976 Houston 1982 Jinks et al. 1981
	۲ : :	30	22.2 26.5 27.5-30.3 26.5	Wabash River, IN Norris Reservoir, TN Lab Lake Monona, WI Lab	Coutant 1977a Coutant 1977a Coutant 1977a Coutant 1977a Coutant 1977a

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	annel Catfish
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TABLE 7	Summary of

Temperature	Concentration	Chlorine	Duration	Effect	Reference
(D°)	(mg/l)	Form	(min)		
10	0.78	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
20	0.49	Monochloramine	4 X 40 min	No mortality	Seegert et al. 1979
20	0.65	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
30	0.53	Monochloramine	4 X 40 min	No mortality	Seegert et al. 1979
30	0.67	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
30	0.09	Residual	Continuous	50% mortality	Roseboom and Richey 1977
Variable	0.05 - 0.18	TRC	100+ days	Reduced Growth	Hermanutz et al. 1987

found an inverse relationship between temperature and LC50. The LC50 values were 0.78, 0.65, and 0.67 mg/l for 10, 20, and 30°C, respectively. The maximum concentrations that resulted in no mortality were 0.49 and 0.53 mg/l for 20°C and 30°C, respectively. All channel catfish that lost equilibrium died. Mortality patterns were similar at 10 and 20°C, about one-third of the fish died before the fourth exposure. However, mortalities occurred more quickly at 30°C than at 20°C, and 53% of the mortalities occurred after the first exposure. Hermanutz et al. (1987) reported a consistent pattern of reduced channel catfish growth in relation to increasing concentrations of TRC.

2. Temperature (Table 8)

The final preferred summer temperatures of channel catfish range between 29.5 and 36°C (Yoder and Gammon, 1976; Spotila et al., 1979). Channel catfish avoid temperatures above 32°C (Coutant, 1977a). The upper incipient lethal temperature is approximately 30 to 33.5°C and the critical thermal maximum is 30 to 42°C (Bennett et al., 1998).

E. COMMON CARP

1. Chlorine (Table 9)

Heath (1977) exposed fingerling common carp three times daily for up to 7 d to pulses of either free chlorine or monochloramine at 6 and 24°C. The 166-h LC50s for free chlorine at 6°C was 0.245 mg/l and for chloramine 1.19 mg/l. This represents a fivefold difference between the two chlorine forms. No deaths were seen in the 0.15 mg/l peak free chlorine. At 24°C, some of the carp were anemic, which may account for their increased sensitivity. The 166-h LC50 for free chlorine was 0.219 mg/l.

Seegert et al. (1979) tested the effects of monochloramine on emerald shiners at 10, 20, and 30°C. The exposure regime consisted of four 40min exposures, administered at 0.5-h intervals over 24 h. In general, there was an inverse relationship between temperature and the LC50 concentrations. The LC50 values were 2.37, 1.82, and 1.50 mg/l for 10, 20, and 30°C, respectively. The maximum concentrations causing no mortality were 1.85 and 1.25 mg/l for 10 and 30°C, respectively. All fish died at concentrations of 3.24, 2.38, and 1.96 mg/l at 10, 20, and 30°C, respectively. At 10°C, <3% of the carp that lost equilibrium recovered. However, at 20 and 30°C, the percentage of fish recovering increased to 19 and 11%, respectively.

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			Avoidance	Preferred		
	30-33.5					Hart 1952
		38				Reutter and Herdendorf 1976
		34.5-41				Cheetham et al. 1976
		30.9-42.1				Bennett et al. 1997
Adult	33.5					Brown 1974
large			32		Wabash River, IN	Coutant 1977a
large			34		Wabash River, IN	Coutant 1977a
			35		Lab	Coutant 1977a
				29.4-30.5		Spotila et al. 1979
				32-36	Ohio River, OH	Yoder and Gammon 1976
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	Common Carp	
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TABLE 9 Summary of Toxic	sity of Chlorine to C	common Carp			
Temperature (°C)	Concentration (mg/l)	Chlorine Form	Duration (min)	Effect	Reference
	0.72	TRC	65	Some mortality	Truchan and Basch 1971
	0.7	Free	6000	80% mortality	Ebeling 1931
10	1.85	Monochloramine	4 X 40 min	No mortality	Seegert et al. 1979
10	2.37	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
10	3.24	Monochloramine	4 X 40 min	100% mortality	Seegert et al. 1979
20	1.82	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
20	2.38	Monochloramine	4 X 40 min	100% mortality	Seegert et al. 1979
30	1.25	Monochloramine	4 X 40 min	No mortality	Seegert et al. 1979
30	1.50	Monochloramine	4 X 40 min	50% mortality	Seegert et al. 1979
30	1.96	Monochloramine	4 X 40 min	100% mortality	Seegert et al. 1979

2. Temperature (Table 10)

The final preferred summer temperatures of common carp range between 26 and 34°C (Yoder and Gammon, 1976; Spotila et al., 1979). Common carp avoid temperatures above 34.5°C (Coutant, 1977a). The upper incipient lethal temperatures are approximately 35 to 36°C, and the critical thermal maximum is 38 to 39°C (Spotila et al., 1979).

F. EMERALD SHINERS

1. Chlorine (Table 11)

Seegert et al. (1979) tested the effects of monochloramine on emerald shiners (*Nortropis atherinoides*) at 10, 20, and 30°C. The exposure regime consisted of four 40-min exposures, administered at 0.5-h intervals over 24 h. In general, there was an inverse relationship between temperature and the LC50 concentrations. The 24-h LC50 values were 0.63, 0.51, and 0.35 mg/l at 10, 20, and 30°C, respectively. The maximum concentrations causing no mortality were 0.46, 0.40, and 0.21 mg/l for 10, 20, and 30°C, respectively. Mortality (100%) occurred at concentrations of 0.97 and 0.59 at 10 and 30°C, respectively. Emerald shiners that lost equilibrium during exposure did not survive. Mortalities occurred more rapidly at warmer water temperatures. At 20°C, 55% of the mortality occurred after only one exposure.

A study of short-term exposure of total residual chlorine on emerald shiners was conducted by Fandrei and Collins (1979). The authors separately acclimated young-of-the-year, yearling, and adult fish at 10 and 25°C prior to testing. Fish were exposed to TRC at a constant concentration for 30 min, however, the TRC concentrations were noted for up to an additional 45 min. Results indicate that emerald shiners were approximately three times more sensitive at the higher water temperatures. Size or age-class of fish only slightly altered the effects of chlorine exposure: at the lower temperature, young-of-the-year fish were less sensitive. The 96-h LC50 for TRC concentrations ranged from 1.32 to 0.71 mg/l at 10°C and from 0.23 to 0.33 mg/l at 25°C.

Brooks and Bartos (1984) studied the effects of free and combined chlorine on emerald shiners at 25°C. The experiments consisted of single 15-, 30-, or 120-min exposures, or four successive 30-min exposures at 2-h intervals. Based on the LC50 concentrations for the four nominal chlorine compounds tested, dichloramine was the most toxic to emerald shiners and monochloramine the least.

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	Reference	Spotila et al. 1979	Coutant 1977a	Yoder and Gammon 1976					
mon Carp	Comments	Lab							Ohio River, IN
nces for Com	Final Preferred						29.7		26-34
s and Prefere	Upper Avoidance							34.5	
nal Tolerance	CTMax			38-39		39			
ummer Therr	UILT	35.7	35.7		35-36				
TABLE 10 Summary of S	Size or Age			Small	Large		Adult		

Note: All temperatures are in °C.

	d Shiner
	to Emeral
	Chlorine
	Toxicity of
TABLE 11	Summary of

Size or Age	UILT	CTMax	Upper	Final	Comments	Reference
			Avoidance	Preferred		
	30.7				Lab	Brown 1974
	35.2				Lab	Ellis 1984
	31				Lab	Carlander 1969
УОҮ	35.2	34.9				Talmage 1978
	32.6					Talmage 1978
				22-24		Reutter and Herdendorf 1976
			>28-30		Thermal Effluent	Brown 1974
			>31.1		Thermal Effluent	Brown 1974
			42		Field	Ellis 1984
			27.8			Jobling 1981
			25.1			Jobling 1981
Note: All tempe	ratures are in	°C.				

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2. Temperature (Table 12)

The final preferred summer temperatures of emerald shiner are 22 to 24°C (Reutter and Herdendorf, 1976). Emerald shiner avoid temperatures above 27.8 to 32°C (Jobling, 1981; Ellis, 1984). Upper incipient lethal temperatures are approximately 30.7 to 35.2°C, and the critical thermal maximum is 34.9°C (Ellis 1984; Talmage 1978). The high temperatures reported by Ellis (1984) arose from a field study that has somewhat disparate results compared with other studies.

F. YELLOW PERCH

1. Chlorine (Table 13)

The response of yellow perch (*Perca flavescens*) to intermittent chlorination was studied by Brooks and Seegert (1977). Fish were tested at 10, 15, 20, 25, and 30°C. Single-exposure 30-min LC50 values ranged from 0.70 mg/l at 30°C to 8.0 mg/l at 10°C. No mortality occurred at concentrations <5.1 mg/l at 10°C, or at a concentration of 0.48 mg/l at 30°C. Triple 5-min exposure LC50 values were 22.6 and 9.0 mg/l at 10 and 20°C, respectively. Mortality occurred immediately after exposure to chlorine in the 30-min single exposure at 10 and 15°C, but was delayed by 2 to 12 h at higher temperatures. This pattern was reversed in the 5-min triple exposure tests. Except at 10°C, 96% of the perch suffering loss of equilibrium eventually died. Therefore, the LC50 and EL50 values of yellow perch exposed to chlorine do not appear to differ.

During exposure, yellow perch swim near the surface when exposed to lethal or near-lethal concentrations of chlorine. The perch also were described as being lethargic. Based on their findings, the authors recommended a mean maximal TRC value of 0.623 mg/l. It should be noted that this concentration is based on the short-term exposures, and so is probably underprotective. Arthur et al. (1975) reported that between 12 and 17°C, yellow perch had a 1-h LC50 value of 0.85 mg/l.

2. Temperature (Table 14)

The final preferred summer temperature of yellow perch is between 18 and 27°C (Coutant, 1977a; Clugston et al., 1978). Yellow perch avoid temperatures above 25 to 26°C (Coutant, 1977a). The upper incipient lethal temperature is about 31 to 32°C; the critical thermal maximum is 35°C (EPA, 1974; Hokanson, 1977).

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TABLE 12	Summary

Size or Age	UILT	CTMax	Upper	Final	Comments	Reference
			Avoidance	Preferred		
	30.7				Lab	Brown 1974
	35.2				Lab	Ellis 1984
	31				Lab	Carlander 1969
ХОҮ	35.2	34.9				Talmage 1978
	32.6					Talmage 1978
				22-24		Reutter and Herdendorf 1976
			>28-30		Thermal Effluent	Brown 1974
			>31.1		Thermal Effluent	Brown 1974
			42		Field	Ellis 1984
			27.8			Jobling 1981
			25.1			Jobling 1981
Note: All tempe	ratures are in	°C.				

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TABLE 13 Summary of Toxid	sity of Chlorine to)	ellow Perch			
Temperature	Concentration	Chlorine Form	Duration (min)	Effect	Reference
	0.72	TRC	65	Some mortality	Truchan and Basch 1971
	0.365	TRC	720	50% mortality	Arthur et al. 1975
	0.85	TRC	09	50% mortality	Arthur et al. 1975
10	15	TRC	30	100% mortality	Brooks and Seegert 1977
15	7.1	TRC	30	100% mortality	Brooks and Seegert 1977
20	2.1	TRC	30	100% mortality	Brooks and Seegert 1977
25	1.6	TRC	30	100% mortality	Brooks and Seegert 1977
30	0.95	TRC	30	100% mortality	Brooks and Seegert 1977
17-24	0.01-0.10	Monochloramine		Variable avoidance	Bogardus et al. 1977

Size or Age	UILT	CTMax	Upper	Final	Comments	Reference
			Avoidance	Preferred		
Adult	32.3				Summer	EPA 1974
Adult	30.9				Summer	Hokanson 1977
Adult		35			Summer	Reutter and Herdendorf 1976
ХОҮ				25-27	Summer	Coutant 1977a
Adult				27	Summer	Coutant 1977a
Juvenile				24	Summer	EPA 1974
ΥΟΥ			26.5	23.3	Summer (day)	Coutant 1977a
ХОҮ			25	22.5	Summer (night)	Coutant 1977a
Adult				18-21	Summer	Clugston et al. 1978

TABLE 14 Summarv of summer thermal tolerances and preferences for vellow perch

Adult Note: All temperatures are in °C.

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G. WHITE BASS

1. Chlorine (Table 15)

Grieve et al. (1977) studied the *in situ* response of white bass (*Morone chrysops*) to chlorination regimes in Lake Ontario using radiotelemetry. During periods when chlorine was not being released, white bass exhibited distinct preferences for several areas in the discharge canal. During chlorination, changes in activity and distribution of white bass were evident. Fish activity and distance traveled initially increased at the beginning of chlorination. However, by 45 min into chlorination, most fish movement had stabilized and the fish had taken up locations in areas where TRC concentrations were < 0.05 mg/l). The fish then became sedentary and occupied these areas until the release of chlorine stopped. Within 2 h, the fish had returned to their prechlorination locations. The authors concluded that white bass avoid chlorine exposure during short-term chlorination in excess of 0.035 mg/l.

Seegert et al. (1979) tested the effects of monochloramine on white bass at 10, 20, and 30°C. The exposure regime consisted of four 40-min exposures, administered at 0.5-h intervals over 24 h. In general, there was an inverse relationship between temperature and the LC50 concentrations. The LC50 values were 2.87, 1.80, and 1.15 for 10, 20, and 30°C, respectively. The maximum concentrations that resulted in no mortality were 1.45 and 0.78 for 20 and 30°C, respectively. Mortality was 100% at concentrations of 2.08 and 1.47 at 20 and 30°C, respectively. White bass that lost equilibrium during exposure did not survive. Mortalities accumulated more quickly at warmer temperatures. At 30°C, 65% of the mortality occurred after the first exposure.

2. Temperature (Table 16)

The final preferred summer temperature of white bass is from 26 to 30°C (Coutant 1977a; Yoder and Gammon, 1976). White bass avoid temperatures above 29 to 34.4°C (Spotila et al., 1979). The upper incipient lethal temperature is approximately 33.5°C, and the critical thermal maximum is 35.3°C.

IX. CONCLUSIONS AND RECOMMENDATIONS

Despite the apparent large number of studies that have been undertaken on the effects of chlorine on freshwater fish, interstudy comparisons are limited. Such comparisons are difficult due to differences in methodologies, nonreporting of important details such as chlorine species, water

Reference		ents Grieve et al. 1978	15 mg/l Grieve et al. 1978	Seegert et al. 1979						
Effect		Avoidance – altered movem	Occupied areas less than 0.0	50% mortality	No mortality	50% mortality	100% mortality	No mortality	50% mortality	100% mortality
Duration	(min)	45	120	4 X 40min	4 X 40 min	4 X 40min	4 X 40 min	4 X 40min	4 X 40 min	4 X 40min
Chlorine	Form	TRC	TRC	Monochloramine						
Concentration	(mg/l)	0.035	0.05	2.87	1.45	1.80	2.08	0.78	1.15	1.47
Temperature	(°C)			10	20	20	20	30	30	30

TABLE 15 Summary of Toxicity of Chlorine to White Bass

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Size or Age	UILT	CTMax	Upper	Final	Comments	Reference
			Avoidance	Preferred		
Adult			29	28-30	Summer	Coutant 1977a
			>29.8		Pickering ON	Ellis 1984
			>34		Colbert, AL	Ellis 1984
Young				26-29	Power Plant, OH	Yoder and Gammon 1976
1			29-34.4		Ohio River, IN	Spotila et al. 1979
		35.3			Lab	Reutter and Herdendorf 1976
үоү	33.5					Ellis 1984
	33.5					Houston 1982
	33.5					Spotila et al. 1979

quality parameters, and testing conditions. Such studies are further complicated by the varying sensitivities of different species, sizes, and life stages.

The large number of studies conducted in the 1970s and early 1980s that have highlighted the toxicity of chlorine has contributed to the development of other technologies for controlling biofilm. In some cases, closed loop systems incorporating generated ozone are used, however, ozone appears to have limited biocidal effectiveness (Kaur et al., 1992; Viera et al., 1999). In open loop systems the chlorine residuals have been held until levels are sufficiently low as to not affect the biota, or treated prior to release. Sodium sulfite shows the most promise as a dechlorinating agent and has been tested on bluegill and largemouth bass (*Micropterus salmoides*) (Wilde and Shealy, 1992). These strategies are effective for smaller generating stations; however, many of the Great Lakes generating facilities are large and the volume of water required to flush the condenser system would be difficult or costly to collect or treat. In addition, researchers have been experimenting with adding sodium bromide with chlorine to create hypobromous acid. The premise behind this approach is that at high pH the bromine oxidants that are produced are more effective at removing biofilm than hypochlorous acid. To date, however, the bromine oxidants appear to be more toxic, although they do have more rapid decay rates relative to chlorine (Fisher et al., 1999). Another promising biocide is dibromonitrilopropionamide (DBNPA) (Klaine et al., 1996). Obviously, we support and encourage the continued development and testing of alternative biocides and encourage readers to consult Characklis and Marshall (1990) for a comprehensive review of the range of alternative biocides and summaries of their mechanistic action and effectiveness.

We did not attempt to present or synthesize alternative biofouling control techniques that have been developed and are now used in some situations. In the Great Lakes region, chlorinated biocides are still predominant, and likely will be until alternative approaches have been shown to be effective and competitively priced. Obviously, alternative technologies will also require environmental assessments to determine their toxicity to fish and other biota.

In this review we have focused on fish; however, we recognize the importance of considering the wider range of possible effects on other biota, including humans, that are possible when using a toxicant such as chlorine. To this end, we recognize the importance of incorporating the concepts of ecological risk assessment methodology into the determination and application of an appropriate biocide. The Environmental Protection Agency (EPA) has been advocating the use of two approaches

for assessing environmental risk in effluents (Norton et al., 1992). The first is to focus on the effluent as a whole (e.g., Chapman, 2000), and the second is the application of chemical-specific water quality criteria. Both of these criteria focus on the magnitude, duration, and frequency of disturbances (Norton et al., 1992). Ecological risk analysis is an effective tool for organizing information used in environmental management decisions (Fairbrother and Bennett, 1999). This approach has been applied recently to the examination of the potential human health and ecological risks of TRC and chloroform discharges into receiving waters (Mills et al., 1998). The multiple stressors (Lowell et al., 2000) associated with chlorinated biocides in thermal effluents (e.g., toxicant, thermal stress) are particularly well suited to the use of ecological risk assessment.

The risks to the biota vary on a site-specific basis, but so do the biofilm prevalence and composition (Poulton et al., 1995). As suggested by Hall et al. (1981) and Bervoets et al. (1993), coordinated laboratory and field studies must be site specific to understand the environmental impacts. Only this approach will ensure that the complex series of interacting factors that contribute to fish distress or mortality are included in the design of chlorination regimes intended to reduce fisheries impacts. We also suggest that researchers include details of methods in order to facilitate the interpretation of work by other researchers.

At power plants, rapid temperature changes related to station generation load can occur and could impose a severe stress on fish (Beitinger and Magnuson, 1976; Schreer and Cooke, in review). The majority of the controlled chlorinations studies have used temperatures that are regarded as preferred, and thus often did not obtain measurements of fish closer to lethal temperatures. These lethal temperature conditions are particularly common in the summer at times when biofouling, and hence the need to chlorinate, are also at their peaks.

Based on the strong avoidance response of most species to low levels of chlorine, one possible strategy for reducing sublethal effects may be to slowly increase concentrations in a stepwise manner over a period of hours allowing time for fish with different sensitivities to detect the chlorine and move away from it. Because fish avoid lethal temperature and chemical conditions, fish kills in and around cooling discharges are rare (Gammon, 1971). Recent work has suggested that some individual fish reside in discharge canals in areas upstream of tempering pumps, where thermal extremes and fluctuations are greatest (Cooke et al., 2000; Schreer and Cooke, in review). It is these fish, in particular, that are at risk from chlorination. The concentrations and chlorination targets must be based on the most upstream areas that fish could occupy, and where concentrations potentially could be highest. Even if regulatory guidelines set targets at further points downstream, the targets must be based on where fish may be exposed. Chlorination also should be stopped if any fish are beginning to lose equilibrium. The majority of fish that have lost equilibrium often experience delayed mortality. Some researchers suggest that chlorination procedures should be developed that use chlorine only as frequently as necessary to control biofilm because higher frequency of exposure implies greater fish mortality (Dickson et al., 1977). The duration should also be as short as possible.

Another consideration in the laboratory studies conducted to date is that fish usually are acclimated to constant thermal conditions for several weeks before testing. At power plants, rapid temperature changes related to station generation load could impose a severe stress on fish due to physiological (Schreer and Cooke, in review), behavioral (Schreer and Cooke, in review), and biochemical (Beitinger and Magnuson, 1976) failures to adjust. Similarly, *in situ* studies of fish response to effluents does not control for the other environmental variables that may alter test results through the attribution of stress and mortalities to only one variable. Therefore, integrated studies that incorporate site-specific characteristics and include a controlled laboratory study are necessary (Mattice and Zittel, 1976; Cairns, 1981; Cherry and Cairns, 1982).

The sensitivity of different species to chlorine varies widely. As such, chlorination regimes should be evaluated on a daily basis to deal with differences in species composition and water temperatures. This requires the ability to document the presence and absence of different species in the vicinity of the chlorinated outfall. Ideally, these distributions would be assessed immediately prior to chlorination and would dictate the concentrations to be used. Realistically, this would be difficult, as numerous researchers have highlighted the problems of identifying fish distributions, relative abundance or even presence/absence in discharge canals. Videography is one method that may prove to be useful for assessing fish presence/absence in thermal discharge canals (Cooke and Schreer, 2001). If the true species complex present cannot be determined immediately prior to chlorination, historical data would be required. From this data, the most sensitive species to chlorine would be chosen based on available literature. In some cases, the species present may not have been studied previously and as such the relative sensitivity may be unknown (requiring further research). The chlorination regime that has been shown to induce no mortality or morbidity would then be chosen. Most of the chlorine exposures reported in the literature are for 50% mortality, but the highest concentration at which no mortality (Howells, 1983) or no sublethal effects, including loss of equilibrium, are likely more appropriate values. Obviously, a monitoring component becomes important, especially for the first several seasons, because the concentrations used are evaluated to determine whether the concentrations chosen were sufficiently conservative. When using literature values, it must be restated that the site-specific characteristics of each location are ignored, and this may provide unrealistic values. Only a series of *in situ* and laboratory studies for each situation will provide biologically meaningful data and the basis for relevant regulations.

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