MASTER

THE EFFECT OF ARSENIC ON THE THERMAL
TOLERANCE AND SURVIVAL OF NEWLY HATCHED
MUSKELLUNGE FRY (ESOX MASQUINONGY)

THESIS

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A thesis submitted in partial fulfillment of the requirements for the degree of Master of Arts of the State University College at Buffalo.

By

Frank V. Paladino
Buffalo, New York

Director: Dr. James R. Spotila
Assistant Professor of Biology
Buffalo, New York

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ABSTRACT OF THESIS

THE EFFECT OF ARSENIC ON THE THERMAL TOLERANCE AND SURVIVAL OF NEWLY HATCHED MUSKELLUNGE FRY (ESOX MASQUINONGY)

Newly hatched muskellunge fry were raised in tanks containing 0.00, 0.05, 1.0 and 5.0 ppm arsenic as sodium arsenite (NaAsO_2) at a temperature of 15°C ($\pm 0.5^\circ\text{C}$) and a photoperiod of 12 hours of light alternating with 12 hours of dark (L. D. 12 : 12). Dissolved oxygen, alkalinity, pH, and ammonia ranged between 8.0 - 10.5 $\text{mg l}^{-1} \text{O}_2$, 149 - 190 $\text{mg l}^{-1} \text{CaCO}_3$, 7.2 - 7.7 and .05 $\text{mg l}^{-1} \text{NH}_4$ respectively.

Temperature tolerance of fry, Critical Thermal Maximum, (CTM), was significantly reduced by exposure to arsenic ($P = 0.05$). Fry raised at arsenic concentrations of 0.05 ppm or greater suffered 100% mortality during or shortly after swim-up. Control (0.00 ppm) fry had a sharp drop in CTM during swim-up, became active feeders and continued to develop normally.

Frank V. Paladino
Frank V. Paladino

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INTRODUCTION

Industrial waste disposal methods and pesticide application practices have introduced high concentrations of many heavy metals into aquatic ecosystems. Numerous publications have described the effects of heavy metals, such as arsenic, cadmium, copper, lead, selenium, tin, zinc and especially mercury on the physiology and survival of fish (Surber 1932, Hart et al. 1945, Douderoff et al. 1951, Lawrence 1958, 1962, 1966, Cairns and Schnierer 1957, 1959, 1964, Dupree 1960, McKee and Wolf 1963, Johnson 1968, Uthe and Bligh 1971, Wood 1974, Cairns et al. 1975).

A review of the available literature resulted in limited and conflicting information on the effects of heavy metals on immature fish. The majority of fish toxicity studies have been conducted using adult indicator organisms such as bluegill sunfish, (Lepomis macrochirus) and smallmouth bass, (Micropterus dolomieu). A few studies indirectly indicated that developing fish were more susceptible to heavy metal pollution than were adults (Gilderhaus 1966, Hiltebran 1967, Nimi and LaHam 1975).

Cairns and Schnierer (1957, 1959) described how pH, temperature, and alkalinity affected the toxicity of zinc to adult L. macrochirus. To my knowledge no research has been conducted to determine the effect of a heavy metal on the thermal tolerance of a developing fish. Some studies have indicated that developing immature fish have a lower temperature tolerance than their eggs and adults (Brett 1956, Banner and Van Arman 1973, Hokanson et al. 1973). Hassan and Spotila (1976), found that rearing temper-

ature affected the thermal tolerance of developing muskellunge fry (Esox masquinongy). They postulated that rearing temperatures and other parameters such as heavy metal contamination of the hatchery water might be contributing to the large scale mortalities of fry at the New York State muskellunge hatchery on Chautauqua Lake.

Chautauqua Lake is located in Chautauqua County at the far western part of New York State. It is finger like in shape, oriented in a north-south direction with a length of 27 km and a width that varies from 1 to 3 km (Fig. 1). The New York State Department of Environmental Conservation established a hatchery in 1964, at Pendergast Point, in order to raise muskellunge (Esox masquinongy) to fingerling size (12 - 20 cm) for stocking. Numerous die-offs have occurred at the hatchery, with 100% mortalities occurring in seven of the last ten years (Youmans 1974). The reason for these die-offs has not been determined.

Chautauqua Lake waters have a past history of high arsenic levels (Ullman et al. 1961). In a recent study Lis and Hopke (1973) found arsenic in the lake waters ranging up to 43.3 ppm. In a later study Hopke et al. (1974) found that arsenic concentrations in lake sediments ranged from 0.5 to 300 ppm, with an overall average of 22.1 ppm. They stated that the sediments of the lake have higher arsenic values than the natural sources in the surrounding area. They attributed these higher concentrations of arsenic to the use of sodium arsenite as an aquatic herbicide during the period 1955 to 1964. During the 1960's arsenic was found in high concentrations in Chautauqua Lake waters. Since the lake is a

public drinking water source, the New York State Department of Health banned the further use of the aquatic herbicide, sodium arsenite, in the lake (Nicholson 1974).

I conducted a preliminary atomic absorption spectrophotometric analysis of the well water from the Pendergast Point Hatchery and found the concentration of arsenic was 3.52 times greater than the New York State Department of Health recommends for drinking water. The concentrations of all other metals tested were within the normal recommended ranges. Although the amount of arsenic found (0.035 ppm) was above the recommended concentration (0.01 ppm) it was below the calculated lethal concentration for most adult fish which is 12 - 20 ppm (Lawrence 1958).

Wood (1974) classified environmental toxic pollutants as (1) non critical, (2) toxic and relatively accessible or (3) toxic but very insoluble or very rare (see Table 1). The majority of the toxic and relatively accessible elements are of the heavy metal and metalloid groups. Arsenic is one of the very toxic and relatively accessible elements.

Arsenic is a known poison and its toxicity to animals is well documented. In drinking water taken over a long period of time a concentration of 0.21 ppm of arsenic has been reported poisonous to humans (Fairhall 1941). Arsenicals form strong bonds with sulfhydryl groups (-SH-), largely in the liver and skin, causing toxicity and accumulation in those tissues. Arsenite, a heavy metal anion, strongly inhibits essential sulfhydryl enzymes such as pyruvate and alpha-keto-glutarate dehydrogenase. Disulfide bonds appear to play an essential role in the polymerization of

proteins in the mitotic apparatus that form spindle fibers. Reagents that form mercaptides, like arsenic are spindle poisons and also dissolve the isolated mitotic apparatus (Dustin 1963). When a lethal dose of sodium arsenite was given to albino rats, intraperitoneally, death was preceded by a profound fall in body temperature. A smaller dose produced transient hypothermia, with a decrease in response and eventual tolerance. Although the site of action was not studied it seemed likely to have been the hypothalamic temperature regulating center (Norris and Elliot 1945).

Arsenic is made available for organismal uptake by natural geochemical cycles and microbial action. Arima and Beppu (1964) found an arsenite degradative enzyme induction system in Pseudomonas pseudomallei. A diagram of the biological cycle for arsenic is presented in Figure 2. The toxicity of arsenic depends on the form in which it is found. Almost all naturally occurring forms of arsenic, salts (arsenate, arsenite), oxides (arsenic trioxide), and acids (methylarsenic acid) are very toxic (McKee and Wolf 1963, Wood 1974, Crecelius 1975).

Lawrence (1958) stated that applications of sodium arsenite up to 10 ppm as arsenic trioxide (7.6 ppm As) were not harmful to fish. However, other studies (Gilderhaus 1966, Hildebran 1967) indicated that the threshold level was lower than expected. Lawrence (1962, 1966) later reported that two applications of 4.0 ppm sodium arsenite applied one month apart in experimental ponds, reduced the number of bottom organisms an average of 34% and reduced bluegill (L. macrochirus) production an average of 42% as compared with control ponds. Two applications of 8.0 ppm of sodium arsenite applied

one month apart reduced the number of bottom organisms an average of 45% and bluegill production 65%. An application of 4.0 ppm killed all microcrustacea and greatly reduced the population of rotifers.

Gilderhaus (1966) evaluated the effect of sodium arsenite on adult and immature L. macrochirus in small fish rearing ponds. In general his experiment demonstrated that applications totaling 4.0 ppm or more of sodium arsenite in one year inhibited the growth of both adults and immatures, and adversely affected the survival of immatures. This 4.0 ppm concentration was well below the accepted dose of sodium arsenite ($2 - 10 \text{ ppm NaAsO}_2 = 2.5 - 12.5 \text{ ppm As}_2\text{O}_3$), which was listed by Lawrence (1958) as an acceptable dose for weed control and which was not harmful to aquatic animals.

Hiltebran (1967) found that fry of L. macrochirus, M. dolmieu, Erimyzon sucetta, and Compostoma anomalum, were more susceptible to the toxic action of sodium arsenite than were the fertilized eggs or the adults. Other experiments have demonstrated that fishfood organisms are susceptible to concentrations of arsenic as low as 1.0 ppm (Springer 1959, Gilderhaus 1966).

It is apparent from these studies that there are lethal and sublethal effects of arsenic upon adult and immature fish as well as the benthos and microcrustacea (Springer 1959, Gilderhaus 1966, Hiltebran 1967). There have been very few studies in which the effect of sublethal doses of arsenic on newly hatched fish fry have been investigated.

The presence of high concentrations of arsenic in the Pendergast Point hatchery water and the report of Hassan and Spotila

(1975), on the temperature tolerance of muskellunge fry, suggests that the mortalities at the hatchery may be due to an interactive effect between rearing temperatures and arsenic contamination.

The purpose of this study was to determine the effect of arsenic on the temperature tolerance and survival of newly hatched muskellunge fry. The results reported here may supply information which could provide insight on the hatchery mortality problem.

MATERIALS AND METHODS

Newly hatched muskellunge fry (Esox masquinongy), were obtained from the Pendergast Point hatchery on May 23, 1975. Approximately 3,000 fry were transported in a sealed plastic bag at a water temperature of $15^{\circ}\text{C} \pm 0.5^{\circ}\text{C}$. Upon arrival at the laboratory in Buffalo, New York, about 350 fry were immediately transferred to each of eight, 20 liter glass tanks. These tanks contained 17 liters of aerated, dechlorinated tap water.

Six tanks were previously seeded with arsenic, two each at 0.05, 1.0, and 5.0 ppm arsenic. Two tanks served as controls and received no arsenic treatment. Arsenic (As) was used in the form of sodium arsenite (NaAsO_2), a salt, which has been commonly used as an aquatic herbicide. Arsenic concentrations were checked initially and subsequently analyzed bi-weekly throughout the course of the experiment using flameless atomic absorption spectrophotometry. Analysis was conducted using a Varian Techtron atomic absorption spectrophotometer, by the method of Tam (1974).

Water temperatures were maintained at $15^{\circ}\text{C} (\pm 0.5^{\circ}\text{C})$ by placing the experimental tanks in large water baths supplied with heaters, refrigeration coils and thermoregulators. 15°C was chosen as the fry acclimation temperature because it approximates the rearing temperature normally used in the past at the Pendergast Point hatchery. Temperatures were measured daily with a mercury thermometer calibrated to $\pm 0.05^{\circ}\text{C}$.

Each glass aquarium was aerated and filtered by a plastic corner filter, filled with activated carbon and glass wool. A photoperiod of 12 hours of light alternating with 12 hours of

darkness (L : D 12 : 12) was centered at 1300 hours E.S.T. Illumination was provided by overhead fluorescent lamps. Light intensity was measured with an Epply pyranometer and ranged from 0.01 - 0.02 cal cm⁻² min⁻¹. Funk radiometer measurements ranged from 0.01 - 0.02 cal cm⁻² min⁻¹. Visible light as measured with a Weston light meter ranged from 68 - 180 foot candles, $\bar{X}$ = 121 foot candles (Hassan and Spotila 1976).

Tanks were checked daily for dead fry, which were removed, rinsed once in distilled water and immediately frozen for future arsenic analysis. After swim-up live brine shrimp (Artemia nauplii) were fed ad. lib. to the fry. Ten fry were sampled bi-weekly from each treatment and also frozen for future analysis.

Water was analyzed bi-weekly for dissolved oxygen (D.O.), pH, alkalinity and ammonia. A Yellow Springs Instruments (Model #54) oxygen meter, calibrated using the Azide Modification of the Winkler method (Standards Methods 1971), was used to determine D.O.'s. PH was measured using a Fisher pH Meter (Model 140) which was standardized daily with stock buffer solutions. Ammonia concentrations were measured using the phenate method and alkalinities were determined using the potentiometric method (Standard Methods 1971). During the first three days of the experiment all tanks were treated with 250 mg of tetracycline to eliminate a Saprolegnia sp. infection, which was present in the control (0.00 ppm) and 0.05 ppm tanks only.

Random samples of six fry from each experimental treatment, were chosen every other day and tested for their thermal tolerance. The thermal tolerance of the fry was checked using the Critical

Thermal Maximum (CTM) method described by Hutchison (1961). A fry was placed into a test water bath at its acclimation temperature and water temperature was raised at a constant rate ($1.0^{\circ}\text{C min}^{-1} \pm 0.2^{\circ}\text{C}$), until the endpoint was reached (see results for description of endpoints used).

Data were statistically analyzed using a one way analysis of variance (one way ANOVA) and graphically using the method of Hutchison (1961). A one way ANOVA was performed on the individual values for the four treatments (0.00, 0.05, 1.0, 5.0 ppm Arsenic), to determine overall statistical differences between the treatments. Statistical differences between the treatments were also determined using the graphical analysis of Hutchison (1961). Differences in CTM through time in each treatment were analyzed using a one way ANOVA and graphically using the Hutchison method (1961). Calculated F values were considered significant at the 5% level of probability ($p = 0.05$) and highly significant at the 1% level ($p = 0.01$). Mortalities were analyzed graphically plotting the time vs. percent dead.

RESULTS

Water quality in the experimental tanks remained normal during the course of this experiment (Table 2). Dissolved oxygen ranged from a high of 10.5 mg l^{-1} to a low of 8.0 mg l^{-1} . Alkalinity ranged from a low of 149 mg l^{-1} of CaCO_3 on day one to a high of 190 mg l^{-1} on day 15. The pH fluctuated between 7.2 and 7.7. Tank temperatures remained stable at $15^\circ\text{C} \pm 0.5^\circ\text{C}$. Ammonia concentrations in all tanks remained below 0.05 mg l^{-1} .

The endpoint used for the CTM of pre-swim-up fry was the onset of dorsal lateral bending, as described by Hassan and Spotila (1976). This endpoint was clearly exhibited by these fry. More than 90% of the fry tested in this manner recovered if they were quickly transferred to a 15°C water bath. If the fry were taken 1.0°C past this endpoint less than 35% recovered. After fry began to swim-up, (day 8) I used the onset of spasms followed by a complete loss of equilibrium as the CTM endpoint. As fry approached their CTM they began to lose their equilibrium and initiated a shuddering movement, the gills became distended and the mouth opened in a gape. I used the rapid shuddering and mouth gaping as the CTM. This was the same endpoint described by Hassan and Spotila (1976). All fry recovered from this endpoint if they were placed in a 15°C water bath immediately after reaching their endpoint. Some fry underwent a temporary loss of righting response prior to the onset of spasms, but this was transitory and I did not use this as the endpoint. The different endpoints for pre- and post-swim-up fry were used because pre-swim-up fry were

sessile in nature and did not exhibit muscular control. The sessile nature of pre-swim-up fry warranted the use of a different endpoint.

A one way ANOVA indicated that there was no significant difference in the CTM of fry taken from the two difference experimental tanks within a treatment, therefore the CTM data from these tanks was combined for further statistical analysis. There was a highly significant difference ($p = 0.01$) in the average CTM of muskellunge fry in different treatments (Table 3). CTM of the control fry ($\bar{X} = 32.42^{\circ}\text{C}$) were significantly higher (Fig. 3) than the 1.0 ppm ($\bar{X} = 30.68^{\circ}\text{C}$) and 5.0 ppm ($\bar{X} = 30.5^{\circ}\text{C}$) treatments. A graphical comparison of control and 0.05 ppm data indicated that the overall CTM were not significantly different (Fig. 3). This was because the standard error bars overlap slightly. However, a one way ANOVA (Table 4), for these two treatments indicated a significant difference ($p = 0.05$). Thus while the 0.05 ppm treatment did lower the CTM, the statistical significance of this effect was unclear when only the overall values were compared.

There was a highly significant difference ($p = 0.01$) in the CTM of muskellunge fry in all treatments with time (Tables 5, 6, 7 and 8). These results are presented graphically in Figures 4 - 7. There was a significant drop in the CTM of the fry in all treatments during swim-up, but this CTM decrease was more pronounced in the three arsenic treatments when compared to the control. The CTM of the control fry recovered from this sharp drop (32.26°C to 30.28°C), and began to slowly increase from 30.28°C on day 10 to

31.78°C on day 15 (Fig. 4). The CTM of all arsenic treated fry remained low and never recovered from the initial drop in CTM (Figs. 5 - 7). CTM of the 0.05 ppm fry dropped from 32.5°C on day 8 to 28.36°C on day 15 (Fig. 5). A similar pattern occurred for the 1.0 and 5.0 ppm tanks (Figs. 6, 7). The CTM for fry in all arsenic treatments continued to drop until day 15 when all fry in these treatments were dead. The swim-up period was prolonged in the tanks treated with arsenic.

Mortality curves (Figs. 8 - 11), show that all the arsenic treated fry died by day 15 of the experiment, which was seven days after the onset of swim-up. The control fry survived the experiment and had a low mortality until day 25, at which time the experiment was terminated due to a lack of sufficient live food. The mortalities for the fry in the arsenic treatments was low for the initial eight days but after the onset of swim-up there was a minimum of three times the average daily mortality increase in fry deaths. This increase in fry mortalities continued until day 15 when 100% of the arsenic treated fry were dead. The mortality curves indicate that the fry died more rapidly in the higher arsenic concentrations than the lower arsenic concentrations.

An attempt was made using atomic absorption analysis, to determine if there was any arsenic present in the eggs and fry prior to the experiment and to determine if any uptake or concentration of arsenic occurred in the fry during the course of this experiment. However all digestion processes attempted were ineffective due to high lipid concentrations in the eggs and fry. The lipids violently reacted with the zinc slurry and caused so much interference, that arsenic readings were not possible.

DISCUSSION

Arsenic has a significant effect on the temperature tolerance and survival of muskellunge fry. It has been demonstrated that a significant drop in CTM occurs in muskellunge fry (Hassan and Spotila 1976), during swim-up. In this experiment the control fry were the only group to survive the experiment and recover from the characteristic decrease in CTM that occurred during swim-up. Arsenic prolongs and pronounces this CTM decrease during swim-up. The thermal tolerance of the fry was altered by the presence of arsenic and a 100% mortality of the fry occurred in all arsenic treatments.

The higher arsenic concentration (5.0 ppm) caused a more pronounced decrease in CTM at swim-up and more rapid mortalities than the lower concentrations (1.0 and 0.05 ppm). The 5.0 ppm group had the lowest overall CTM (30.5°C) followed by 1.0 ppm (30.68°C), 0.05 ppm (31.54°C) and the control group (32.42°C). The death rate was most rapid in the 5.0 ppm group, followed by 1.0 and 0.05 ppm, all these groups eventually having 100% mortality by day 15. The effects of arsenic on the fry were increased with an elevation in the concentration of arsenic in the water.

The results indicate that the accepted arsenic herbicide treatment value, 2 - 10 ppm (Lawrence 1962), would affect the thermal tolerance and survival of immature muskellunge. In fact the levels of arsenic reported at Chautauqua Lake by Lis and Hopke (1973) may be lethal to muskellunge fry. Wood (1974) described how arsenic acts like mercury in the aquatic ecosystem in several

ways. He diagramed the biological cycle for arsenic (Fig. 2), which illustrates how arsenic remains in and recycles within the aquatic ecosystem. The immatures of subsequent generations could be affected by the continual release of arsenic from the sediments.

In light of the results of this study and other recent findings (Gilderhaus 1966, Hiltebran 1967, Cairns et al. 1975), it appears that present treatment values for all toxic chemicals should be re-evaluated. The existing standards were determined when accurate modern analytical equipment was not available. Many standard values were also determined using hardy adult indicator organisms such as bluegills (L. macrochirus), and bass (Micropterus sp.). The effect of toxins on immature fish could be very important to the stability of an aquatic ecosystem. Thus the tolerance of immature fish should be considered in the determination of new standards for heavy metals.

Since the swim-up period of development has been demonstrated to be stressful and critical in the morphogenesis of the muskellunge, any additional stress, such as that caused by arsenic pollution can be lethal. The results of this study lend support to the idea that immatures are less tolerant of toxicants and more susceptible to environmental perturbations than adults. These results are consistent with other fish studies which demonstrated that immature fish are less tolerant of toxic chemicals than adults (Gilderhaus 1966, Hiltebran 1967, Nimi and LaHam 1975).

The report of Hassan and Spotila (1976) and this study suggests that the mortalities at the Chautauqua Lake hatchery may be due to an interactive effect of rearing temperature and arsenic

contaminations. I suggest that the New York State Department of Environmental Conservation further investigate the possibility of arsenic contamination in the hatchery well water. The interactive effect of rearing temperatures and heavy metal contamination in the survival of muskellunge fry should also be investigated.

Table 1. Classification of elements according to their toxicity, after Wood (1974).

Noncritical			Very Toxic and relatively accessible			Toxic but very insoluble or very rare	
Na	C	F	Be	As	Au	Ti	Ga
K	P	Li	Co	Se	Hg	Hf	La
Mg	Fe	Rb	Ni	Te	Pb	Zr	Os
Ca	S	Sr	Cu	Pd	Sb	W	Rh
H	Cl	Al	Zn	Ag	Bi	Nb	Ir
O	Br	Si	Sn	Cd	Pt	Ta	Ru
N						Re	Ba

Table 2. Water quality for tanks used in the arsenic study.
Values are given as ranges.

Parameter	Treatment (ppm As)			
	0.00 (Control)	0.05	1.0	5.0
Dissolved Oxygen mg l ⁻¹ O ₂	8.2-10.5	8.6-10.5	8.0-9.8	8.6-10.5
Alkalinity mg l ⁻¹ CaCO ₃	157-175	162-189	149-190	152-184
pH	7.2-7.7	7.2-7.6	7.2-7.7	7.3-7.7
Temperature °C	14.6-15.3	14.4-15.6	14.8-15.4	14.5-14.8
Ammonia mg l ⁻¹ NH ₄	0.05	0.05	0.05	0.05

Table 3. One Way ANOVA for overall CTM of muskellunge fry reared in 0.00 (Control), 0.05, 1.0 and 5.0 ppm arsenic.

SOURCE	df	SS	MS	F
Between Means	3.0	104.77	34.9	
Within Means	176.0	846.6	4.8	7.26 **
Total	179.0	951.38	0.0	

Table 4. One Way ANOVA for CTM of muskellunge fry reared in 0.00 (Control) and 0.05 ppm arsenic.

SOURCE	df	SS	MS	F
Between Means	1.0	18.03	18.03	
Within Means	86.0	280.20	3.26	5.54 **
Total	87.0	298.23	0.00	

*P = .05

**P = .01

Table 5. One Way ANOVA for CTM through time of muskellunge fry reared in 0.00 ppm arsenic (Control).

SOURCE	df	SS	MS	F
Between Means	7.0	217.63	31.09	
Within Means	38.0	41.35	1.08	7.61 **
Total	45.0	258.98	0.00	

Table 6. One Way ANOVA for CTM through time of muskellunge fry reared in 0.05 ppm arsenic.

SOURCE	df	SS	MS	F
Between Means	7.0	168.23	24.03	
Within Means	38.0	17.34	.46	52.66 **
Total	45.0	185.57	0.00	

*P = .05

**P = .01

Table 7. One Way ANOVA for CTM through time of muskellunge fry reared in 1.0 ppm arsenic.

SOURCE	df	SS	MS	F
Between Means	7.0	217.63	31.09	
Within Means	38.0	41.35	1.08	28.57**
Total	45.0	258.98	0.00	

Table 8. One Way ANOVA for CTM through time of muskellunge fry reared in 5.0 ppm arsenic.

SOURCE	df	SS	MS	F
Between Means	7.0	249.64	35.66	
Within Means	38.0	57.27	1.5	23.66 **
Total	45.0	306.91	0.00	

*P = .05

**P = .01

Figure 1. Outline map of Chautauqua Lake, New York.

MAYVILLE

HATCHERY

CHAUTAUQUA
LAKE

JAMESTOWN

Figure 2. The biological cycle for arsenic redrawn after
Wood (1974).

The biological cycle for arsenic

Figure 3. Graphical comparison of the overall CTM for muskellunge fry reared in 0.00 (control), 0.05, 1.0 and 5.0 ppm arsenic. Center vertical lines represent overall treatment CTM mean, open rectangles represent 2 standard errors to either side of the mean, Sample size was 45 for each treatment. If open rectangles of the different treatments overlap then they are non-significant $p = 0.05$. After method of Hutchison (1961).

Figure 4. CTM through time of muskellunge fry reared in 0.00 ppm arsenic (control). Verticle lines bounded by short horizontal lines represents the range, median horizontal line represents mean and black rectangles represent 2.54 standard errors to either side of the mean. Sample size was 6. If the black rectangles for the CTM of different days overlap then they are non-significant $p = 0.05$. After method of Hutchison (1961).

Figure 5. CTM through time of muskellunge fry reared in 0.05 ppm arsenic. See Figure 4 for explanation of graph.

Figure 6. CTM through time of muskellunge fry reared in 1.0 ppm arsenic. See Figure 4 for explanation.

Figure 7. CTM through time of muskellunge fry reared in 5.0 ppm arsenic. See Figure 4 for explanation.

Figure 8. Daily mortality (percent dead) through time of muskellunge fry reared in 0.00 ppm arsenic (Control).

Figure 9. Daily mortality (percent dead) through time of muskellunge fry reared in 0.05 ppm arsenic.

Figure 10. Daily mortality (percent dead) through time of muskellunge fry reared in 1.0 ppm arsenic.

Figure 11. Daily mortality (percent dead) through time of muskellunge fry reared in 5.0 ppm -rsenic.

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BIOGRAPHICAL SKETCH OF AUTHOR

Frank Vincent Paladino

completed his undergraduate education in June 1974 at the State University College at Plattsburgh, New York. receiving a Bachelor of Arts degree in Biology. As an undergraduate the author was a New York State Regents Scholarship recipient and a resident assistant in the residence halls.

The author entered the Master of Arts program in Biology at the State University College at Buffalo in September 1974. During his first semester he was a teaching assistant in the Department of Psychology. He was subsequently supported as a residence hall director by the Office of Residence Life. The author was married to Jacqueline Ann Corral on December 27, 1974. They are the parents of one son Michael Frank, born on December 7. 1975.

Commencing in August, 1976 the author will be a doctoral student in the Department of Zoophysiology at Washington State University in Pullman, Washington.