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Alewife Mortality, Condition, and Immune Response to Prolonged Cold Temperatures

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ABSTRACT. *Alewife,* Alosa pseudoharengus, *have been recognized for several decades as one of the most important forage fish in the Laurentian Great Lakes. Although massive alewife die-offs have regularly been observed throughout the Great Lakes and other inland lakes, little substantive information is available regarding physiological mechanisms associated with adult alewife mortality. Long-term field surveys have shown a correlation between cold winter temperatures, poor condition and adult alewife mortality. In this study, adult alewife were raised in replicate pond systems and subjected to contrasting cold temperatures (4 and < 2°C) representing mild and severe winter conditions. We evaluated alewife mortality, condition and immune response to these temperatures. In contrast to our expectations, alewife exposed to mild and severe winter temperatures showed no difference in mortality or condition (measured as the ratio of dry to wet weight). Survival of alewife held in ponds with mild winter conditions (~ 4°C) was similar to that of alewife exposed to prolonged periods (more than six weeks) of temperatures < 2°C. This result contrasts with previous observations indicating that alewife cannot tolerate temperatures < 3°C. Circulating lymphocytes from alewife exposed to severe winter temperatures were significantly lower in number (~40%) compared to fish experiencing milder winter conditions, suggesting sub-lethal immunosuppression in response to the colder winter temperatures. Although colder winter temperatures did not directly induce alewife mortality, these results suggest that winter conditions that result in colder water temperatures can produce immunosuppression, thereby increasing alewife susceptibility to disease and mortality.*

INDEX WORDS: *Alewife, mortality, condition, immune response, temperature.*

INTRODUCTION

Alewife (*Alosa pseudoharengus*) have been recognized for more than three decades as one of the most important forage fish in the Laurentian Great Lakes (Smith 1970), as evidenced by the heavy reliance of salmonine predators on alewife as prey (Jude *et al.* 1987, Lantry 2001, Madenjian *et al.* 2002). Fluctuations in alewife abundance and condition can affect salmonine growth and survival (Stewart *et al.* 1981, Rand and Stewart 1998). As a result, salmonine communities in the Great Lakes are closely linked to the success of alewife populations upon which they rely as forage.

Massive alewife die-offs have been observed throughout the Great Lakes and in other inland lakes since the late 1800s (Goode 1884, Pritchard 1929, Colby 1973). Researchers have suggested that several factors (e.g., temperature induced spawning stress, low food availability resulting in poor condition) could cause mortality (Flath and Diana 1985, O'Gorman and Schneider 1986, Bergstedt and O'Gorman 1989), and cold temperatures are known to challenge the osmoregulatory capabilities of alewife (Stanley and Colby 1971, Snyder and Hennessey 2003). Temperature changes can alter the permeability of cell membranes in teleost fishes, leading to impaired ion transport and membrane-associated enzyme function (Hazel and Williams 1990, Hazel 1993). As such, severe winter temperatures were considered as a possible contributor to alewife mortality (Eck and Brown 1985, O'Gorman and Schneider 1986, Bergstedt and O'Gorman 1989). Fungal infections of *Saprolegnia sp*. were documented in field-collected and laboratory-reared alewife that had been exposed to cold winter temperatures (Graham 1956, Brown 1968,

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Colby 1973), but the causal nature of these infections has not been evaluated.

Although several studies have been conducted to investigate the effects of cold temperatures on the immune response of teleost fishes, factors influencing the immune response of alewife have not been previously evaluated. Exposure to cold temperatures has been suggested to produce immunosuppression, as measured by a decrease in lymphocyte function and number of circulating lymphocytes (lymphopenia) in bluegill (*Lepomis macrochirus*) (Cuchens and Clem 1977), channel catfish (*Ictalarus punctatus*) (Clem *et al.* 1984, Miller and Clem 1984), hybrid striped bass (*Morone chrysops* × *Morone saxatilis*) (Hrubec *et al.* 1997) and pinfish (*Lagodon rhomboids*) (Abruzzini *et al.* 1982). Lymphopenia has also been induced in brown trout (*Salmo trutta*) (Pickering 1984) and rainbow trout (*Oncorhyncus mykiss*) (Barton *et al.* 1987) by feeding cortisol, a stress hormone, directly to fish. Several cell types serve as indicators of an immune response to stimuli (Iwama and Nakanishi 1996): lymphocytes (comprised of T and B-cells, which are the primary cell types generally responsible for activating macrophages and producing antibodies respectively), neutrophils (short-lived, granulated phagocytic cells that combat microorganisms), monocytes (longer-lived phagocytic cells that combat microorganisms), and eosinophils and basophils (both involved in inflammatory and allergic responses associated with viral infections and parasites). Previous investigations have shown that immune system dysfunction in fish increases their susceptibility to antigens and subsequent mortality (Arkoosh *et al.* 1998b, Arkoosh and Collier 2002).

Based on previous observations, two lines of evidence prompted us to evaluate whether the immune response of alewife changed in response to contrasting winter temperature conditions representative of mild and severe winters: (1) the observed immune response to cold temperatures in other fishes, and (2) observations that colder temperatures increased the susceptibility of alewife to *Saprolegnia* sp. infection and subsequent mortality (Graham 1956, Brown 1968, Colby 1973). In this study we evaluated adult alewife mortality, condition and immune system response to different thermal conditions in replicated pond experiments. We hypothesized that alewife exposed to mild winter temperatures would experience reduced mortality, lower water content, and greater levels of immune system activity than alewife exposed to colder temperatures, and that alewife mortality would be near 100% in ponds with severe winter temperatures.

METHODS

This experiment was conducted in replicate 0.1 ha ponds (2.5 m maximum depth) at the Cornell Experimental Pond Facility. Each pond is lined with clay, limiting water exchange between ponds and other inputs such as groundwater that might provide thermal refuge during winter conditions. Anoxic conditions were not observed within the study ponds, and water conductivity was ≈ 300 µS/cm throughout the experiment.

Alewife were collected from Waneta Lake (Schuyler County, NY) on 26 Oct 04 from a single site with a water temperature of approximately 10°C. Based on previous experience, alewife were kept at approximately 10°C during transport and water temperature in the experimental ponds was also 10°C at the time of stocking. Thirty alewife were dried for water content analysis prior to stocking, and 160 alewife were stocked into each of four fishless ponds (640 alewife total) on 27 Oct 04. Only mature, adult alewife between 120 and 140 mm were used in this experiment. Contrasting winter temperatures were produced by maintaining ice-free conditions using aeration in two of the study ponds, beginning at the time of ice formation (early December). Aeration produces ice-free conditions which allows for heat exchange between surface waters and the air, producing colder temperatures in ponds without an insulating layer of ice and snow. Ponds were aerated using a small land-based air compressor with four hoses contained in individual, large (0.5 m diameter) weighted flower pots (two in each pond) from 11 Dec 04 to 5 Jan 05 to generate cold pond temperatures. Hoses were held in place through the bottom of the upright flower pots and were never in contact with pond sediment, thereby minimizing resuspension and circulation of substrate material but allowing thorough thermal mixing. Temperature loggers were deployed at a depth of 1 m in each of the four ponds from December 04 to April 05.

We attempted to collect alewife from the frozen ponds to evaluate alewife feeding activity during the winter months using 30-m long, 25.4 mm stretch mesh, monofilament gill nets during mid-January 05. Weighted ropes were placed on the bottom of each pond prior to ice formation and then used to pull gillnets across the ponds.

On 21 Apr 05 water was pumped from each pond

to achieve a depth of approximately 1.2 m. A bag seine approximately 40-m long and 1.2-m tall was used to collect 25 live alewife from each pond. Alewife were sacrificed using MS-222 (125 mg/L) and blood samples were drawn using a 28-gauge needle and 0.5 mL non-heparinized syringe from the caudal vessels in the hemal arch of ten individual alewife from each of the four ponds. For white cell differential counts (the process of identifying and counting different types of white blood cells), blood smears were prepared immediately on glass slides that were dried and subsequently fixed and stained using a three step Diff-Quik kit (Sigma-Aldrich Chemical; St. Louis, MO).

We performed white cell differential counts by locating monolayered cell regions on each slide (using 1,000× magnification), then identifying the first 200 white cells that were observed within each blood smear. These analyses were conducted by a single, trained individual for standardization. The resulting data were used to generate a ratio of lymphocytes, neutrophils, monocytes, and other granulocytes to total white blood cells that was subsequently used to estimate differential white cell counts. Due to their relative scarcity, eosinophils and basophils were combined into a single category, called "other granulocytes." A total white blood cell count was determined by averaging the number of white blood cells within ten high-power fields (400× magnification) in a monolayer portion of each slide, then multiplying this value by 2,000 to arrive at a cell count quantified as number per microliter (Campbell 1994a, Campbell 1994b). The differential ratios and leukocyte count data were combined to obtain a total count for each cell type. These estimated values for each cell type assumed that each slide was prepared in a similar fashion (i.e., similar pressure was used for each smear) and that the blood was of similar viscosity. Cells were not counted at slide locations that included clumped regions of cells. In order to maintain consistency in the white cell count estimates, these counts were conducted by the same trained individual who processed the differential counts.

Fifteen alewife were sacrificed (see Methods above) from each of the four ponds at the end of the experiment to compare the ratio of dry/wet weight in fish from each experimental treatment. Alewife were kept on ice until they could be accurately weighed (to the nearest 0.5 g) and measured in the lab, then were subsequently reweighed after being placed in a drying oven at 60°C for 168 hours. The proportion of water in each individual was used as an indicator of condition, following the approach described by Flath and Diana (1985). After alewife were collected for these analyses, the ponds were drained completely and all remaining alewife were collected and counted.

Mean total mortality for alewife in each pond as well as alewife initial (Waneta Lake) versus final (post-pond experiment) water content were compared with an analysis of variance (ANOVA) using SPlus (Insightful Corporation, Seattle, Washington). Mixed-model analyses (pond as a random effect and treatment as a fixed effect using the PROC_MIX procedure) were conducted using SAS (SAS Institute Inc.) to test for treatment effects on alewife water content and white blood cell counts.

RESULTS

Temperatures (Fig. 1) in each of the two non-aerated (mild winter temperature) ponds remained below 3°C for 12 and 11 days, respectively, and dropped below 2°C for 9 and 4 days just prior to the onset of ice formation. Temperatures in each of the two aerated (severe winter temperature) ponds were lower than 3°C for 56 and 57 days, respectively, were < 2°C for 46 and 54 days, and approached 0°C on several occasions.

Survival in both temperature treatments (mild temperature ponds: 28% and 27.5%; severe temperature ponds: 30% and 23%) was not significantly different (one-way ANOVA; $F = 0.13$, $p = 0.75$, n's = 2). Eleven alewife were caught in gill nets set in a mild temperature pond (#231) on 11 January, however, additional attempts to capture alewife in the other ponds were unsuccessful. Diet items found within stomachs of the 11 alewives included chironomid larvae, cladocerans, and immature ephemeropterans, along with sediment and small pieces of macrophytes. A total of 36 individual diet items weighing approximately 1 g (total wet weight) were present in all stomachs analyzed yielding an average ration of < 0.01 g/g body weight. The alewife that were removed in mid-January were excluded from the survival analysis; however, their inclusion did not change the significance of the comparison (results not shown).

No difference in the dry/wet weight ratio was observed between alewife maintained in ponds with mild and severe winter temperatures, (Mixed-model; $F < 0.01$, $p < 0.99$, n's = 30) suggesting alewife condition was similar (*cf* Hartman and Brandt 1995). The mean percent water (± 1 SD) of alewife collected prior to stocking (69.2 ±

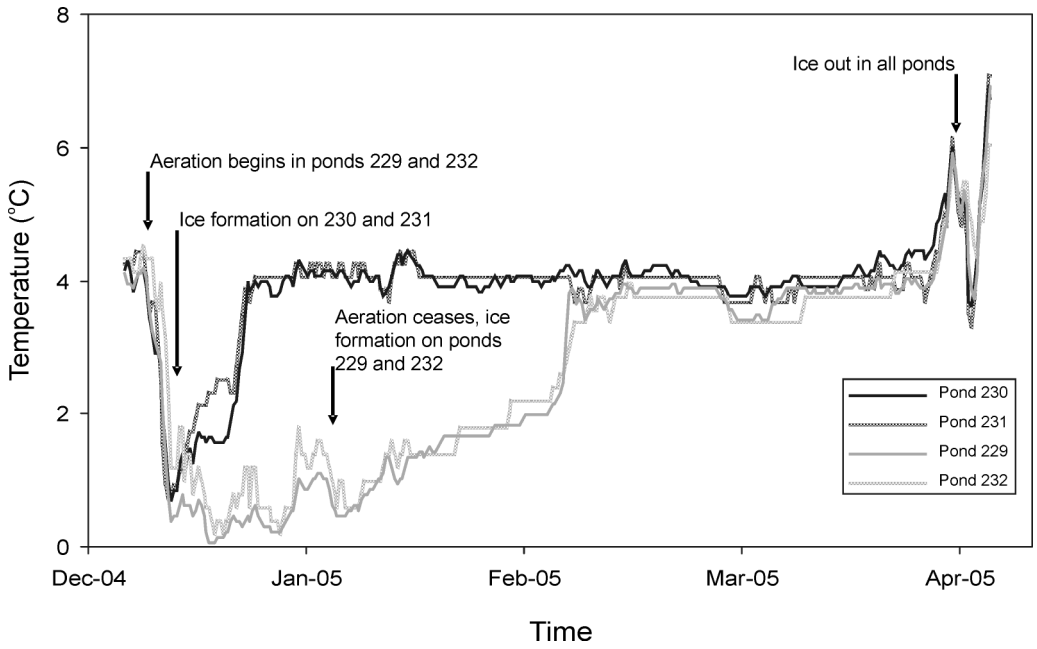

FIG. 1. Temperatures throughout the experimental period for each experimental pond. Status of the aeration system and ice conditions are included in the figure. Ponds #230 and 231 experienced mild winter temperatures while ponds #229 and 232 experienced more severe winter temperatures.

2.9%).was slightly lower than for fish at the end of the experiment (70.8 ± 2.3%) (one-way ANOVA; $F = 3.81$, $p = 0.03$, n's = 30). A Tukey's pairwise comparison confirmed that the percent water content of alewife at the time of stocking was lower than that of fish removed from ponds at the end of the experiment.

Circulating lymphocyte, neutrophil, monocyte and other granulocyte counts varied between alewife subjected to mild and severe temperatures (Fig. 2). The lymphocyte counts of the alewife subjected to mild temperatures was 60% higher than those for fish subjected to severe temperatures (see Table 1). Neutrophil, monocyte, and other granulocyte counts were not significantly different between the alewife from mild temperature ponds and severe temperature ponds respectively.

DISCUSSION

Our results indicate that 6 weeks of exposure to temperatures < 2°C was not sufficient to cause an increase in alewife mortality when compared with alewife exposed to less severe temperatures. Additionally, alewife condition—measured as the proportion of water in fish tissue—did not differ between fish subjected to mild and severe temperatures. Alewife water content was slightly greater when the fish were collected in the spring relative to when they were stocked, but this was likely due to lower feeding rates during the winter months

TABLE 1. Mean (± 1 std. dev.) white cell differential counts (No./µL) by treatment. The* F*-statistic and* p *value for Mixed-model analyses are listed. N = 20 for every treatment in each comparison.

Cell type	Mild temperature	Severe temperature	F	p
Lymphocyte	36,000 ± 10,000	22,000 ± 10,000	18.37	< 0.01
Neutrophil	720 ± 570	1,800 ± 1,900	5.24	0.15
Monocyte	3,200 ± 3,700	2,700 ± 3,000	0.21	0.69
Other Granulocyte	770 ± 1,000	480 ± 460	1.39	0.25

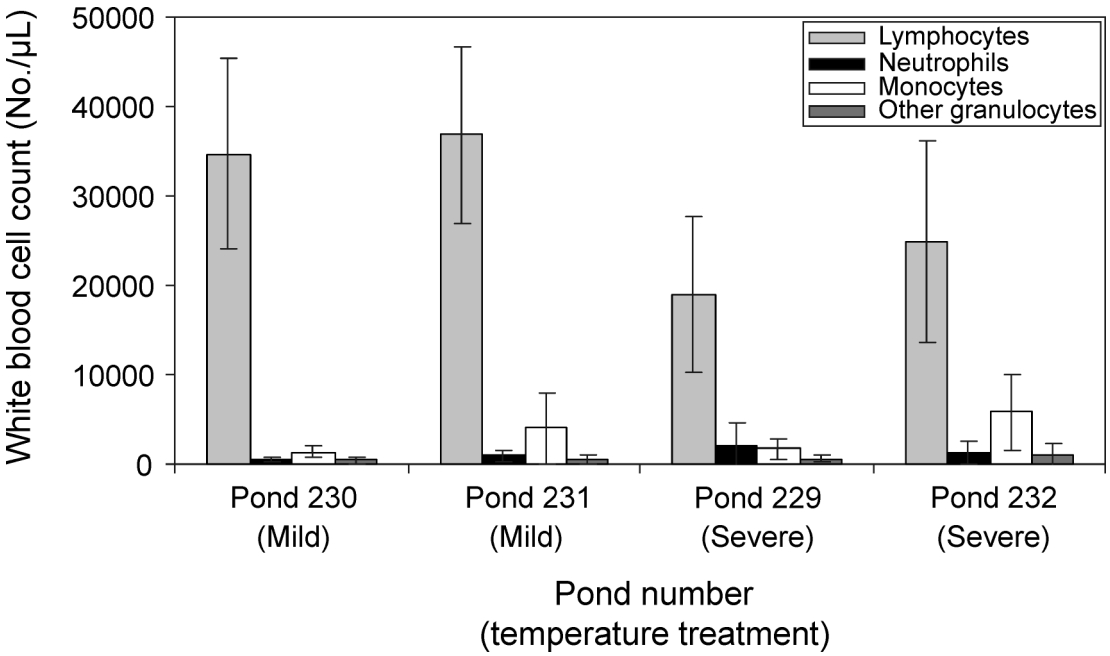

FIG. 2. Mean estimated cell counts from alewife collected from ponds in April 2005. Indices were defined as the mean white cell count per field at 1,000× magnification multiplied by the white cell differentials determined from the first 200 white cells identified. Error bars represent one standard deviation from the mean.

(Flath and Diana 1985). Diet analysis of alewife collected in January from a single mild temperature pond (#231) indicated that these fish were feeding at low levels, but we were unable to capture fish from the other ponds for comparison. The most distinct difference between alewife exposed to the mild and severe temperature treatments was the presence of lower circulating lymphocyte counts in alewife subjected to severe winter conditions.

By evaluating the alewife response to cold temperatures in replicated pond systems, this study expands upon what was learned about alewife thermal tolerance in laboratory studies conducted three decades ago (Colby 1973). Alewife collected for the experiments conducted by Colby (1973) were young-of-year alewife from Lake Michigan that never experienced winter conditions outside of the laboratory. When these fish were acclimated to low temperatures for prolonged periods of time in the laboratory they experienced mortality once temperatures were reduced to 4.0°C and lower. Colby (1973) also collected adult alewife that had experienced winter conditions in Lake Michigan and subjected them to decreased water temperatures. 100% of the alewife contracted "*Saprolegnia*-like" fungal infections prior to death when held at temperatures approximately 5.6°C and cooler (Colby 1973). Alewife in those experiments were reported to have appeared healthy prior to the initiation of cold temperature stress. Wells (1968), however, collected thousands of alewives in Lake Michigan at temperatures < 3.0°C in February and March, and smaller numbers of alewife were captured at shallow depths in even colder temperatures approaching 2.0°C throughout the sampled water column. With these observations in mind, it is worth noting that we observed similar high mortality in both ~ 4°C and < 2°C pond treatments.

Although considerable mortality was observed in our study, these types of field experiments remain challenging to conduct. Stress associated with transporting and maintaining alewife in relatively small ponds might have contributed to the observed mortality; however, these conditions were similar for all alewife used in the study (aside from the temperature manipulation). We also recognize that high overall levels of mortality in this study may have limited our ability to detect small differences in mortality resulting from severe winter temperatures. However, we expected differences in mortality to be large. Nevertheless, we have demonstrated that some alewife can survive extended periods of

time in water less than 2.0°C under certain conditions. These results were unexpected.

The lower counts of lymphocytes observed in alewife from ponds with severe winter temperature conditions was similar to that reported from other fish species exposed to altered thermal conditions. For example, striped bass were found to have significantly lower white cell counts (~20,000 total white cells versus ~60,000 per microliter respectively) after a 6 week exposure to cold (10°C) versus warm (18°C, 24°C, and 29°C) water temperatures in the laboratory (Hrubec *et al.* 1997). Differences in lymphocyte counts of alewife were also similar to those observed in induced stress trials using brown trout (Pickering 1984) and rainbow trout (Barton *et al.* 1987). Although we observed small, non-significant differences in the counts of other cell types in response to experimental temperature treatments, the response in lymphocyte counts (the dominant cell type) was larger, and significant differences between treatments were consistently observed. The lower lymphocyte counts likely play an important role in alewife health and susceptibility to antigens. Although the cell counts do not provide direct evidence of pathological challenges, previous studies have suggested that changes within fish immune systems similar to those observed in our study have increased the susceptibility of fish to disease (Barker *et al.* 1994, Arkoosh *et al.* 1998b, Arkoosh and Collier 2002). Limited blood work was conducted on alewife in the late 1970s in a study of a red cell infection called piscine erythrocytic necrosis (PEN) (Sherburne 1977). Since that time hematological characteristics of alewife have remained largely unexplored, and immunosuppression induced by cold temperatures has never been investigated. White cell data are not available for alewife from other ecosystems, thus comparisons between alewife white blood cell counts at the end of this experiment relative to alewife collected from natural settings remain speculative.

Substantial evidence linking massive alewife mortalities to disease has been found in the past. Several studies have shown that a range of factors—such as low temperatures, anthropogenic pollutants and general stress—can increase the incidence of disease and parasitism in a variety of fish species (Bly *et al.* 1997, Arkoosh *et al.* 1998a, Harris *et al.* 2000). These studies have confirmed that immunosuppression limits the ability of fishes to fend off disease and parasites and decreases their ability to survive when exposed to such challenges. The lower lymphocyte counts at colder temperatures indicates that alewife experiencing winter conditions of similar duration and intensity might be susceptible to disease and subsequent mortality resulting from a reduced white cell count. Graham (1956) suggested that alewife experiencing stress from rapid temperature changes were more susceptible to infection by *Saprolegnia* sp. than other alewife. Brown (1968) reported a 20% incidence of *Saprolegnia* sp. infection in alewife visually examined after the 1967 die-off in Lake Michigan. Colby (1973) observed that 100% of adult alewife exposed to cold temperatures contracted "*Saprolegnia*-like" fungal infections while control alewife experienced only 30% mortality. Despite these observations—and despite concerns about large alewife die-offs in the Great Lakes—little emphasis has been placed on studying the response of alewife to disease.

Age and maturity has been shown to affect the composition of white cell types in fish (Blaxhall 1972, McCarthy *et al.* 1975, Hrubec *et al.* 2001). The alewife used in this study were mature, adult fish, and only fish between 120 and 140 mm total length were evaluated for blood cell composition analyses. Thus, alewife age and maturity were not factors in the results of this study. Aeration was used to cool two of the four ponds, and the physical disturbance caused by aeration had the potential to alter other conditions such as dissolved oxygen, suspended solids, and water movement. An effort was made to minimize the aeration disturbance of pond substrates by using flower pots to minimize interactions between bubbles and pond substrate, yet at the same time water movement was sufficient to ensure that the cold ponds were well-mixed throughout the water column with respect to temperature and dissolved oxygen.

Alewife white cell counts were significantly lower in fish exposed to cold temperatures, and though the procedure used for counting cells is limited to making comparisons between fish within the same study, white cell counts may present a useful and underutilized measure of the status of immune system integrity in other fishes captured in the wild. White cell counts have been used extensively in laboratory and clinical trials involving salmonid fishes, but they have not been used widely in natural systems (Barton and Iwama 1991, Anderson 1996). Basic measures of white cell differentials and counts, when combined with appropriate study design, have provided useful insights in natural systems (*e.g.*, Barker *et al.* 1994, Arkoosh *et al.* 1998b, Arkoosh and Collier 2002). White cell differentiation and enumeration are techniques that can be em-

ployed without harming individual fish, thereby facilitating the evaluation of fish immune system and stress responses to contrasting environmental conditions. We believe that the potential utility of these techniques has not been fully realized in natural aquatic systems.

Speculative explanations regarding the cause of massive alewife die-offs in the Great Lakes have focused on attempts to find correlations between environmental or biological factors—such as temperature, food availability and spawning stress—and long-term estimates of alewife abundance and condition (Flath and Diana 1985, O'Gorman and Schneider 1986, Bergstedt and O'Gorman 1989). Our experimental results suggested that poor alewife immune system integrity may also be related to severe winter temperatures. Severely cold temperatures could be responsible for producing sub-lethal effects causing alewife to be more susceptible to mortality from disease, parasitism, and bacterial infection. It is likely that under our study conditions—i.e., these were closed systems with low alewife density, the fish were relatively small, and the fish community was exposed to the same pathogens—all alewife were at a lower risk of disease and parasitism than under ambient Great Lakes conditions (Bagge and Valtonen 1999, Valtonen *et al.* 2003, Bagge *et al.* 2004). Although high mortality in this experiment may have limited our ability to detect slight differences in mortality if the alewife in the ponds had been exposed to high levels of pathogens, it is possible that alewife experiencing severe winter temperatures would have responded differently (e.g., higher mortality) relative to alewife in warmer ponds.

Alewife water content was not related to the cold temperature treatments in this experiment. This suggests that factors other than severe over-winter temperatures (e.g., alewife condition going into winter or spawning or some other stress) play a more important role in determining alewife mortality and condition in spring than severe temperatures themselves. Interactions between water temperature, winter duration, food availability, and alewife feeding efficiency could also affect alewife mortality and condition, but information regarding food availability, daily ration, and activity were not available to directly evaluate these factors. Nevertheless, because our experimental treatments did not influence fish condition, our results suggest that severe winter temperatures of the duration and intensity simulated in this study by themselves have little direct effect on alewife condition.

Surface and nearshore temperatures in the Laurentian Great Lakes approach 0°C during most winters. Although alewife often move to deeper, warmer (i.e., 3.0 to 4.0°C) water after fall turnover (Gross 1959, Wells 1968), Great Lakes alewife can be exposed to temperatures < 3.0°C for prolonged periods (Schroeder 1963). Recent climate circulation models project future changes in global climate, and it is likely that Great Lakes ice conditions and nearshore temperatures will be affected (Hodgkins *et al.* 2002). Our results suggest that severe winter temperatures have the potential to induce changes in alewife immune response which could lead to increased mortality. Our results expand upon previously available knowledge regarding the temperature tolerance and immune response of alewife in field conditions and will hopefully lead to further studies expanding our understanding of factors influencing winter alewife mortality.

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REFERENCES

Abruzzini, A.F., Ingram, L.O., and Clem, L.W. 1982. Temperature mediated processes in teleost immunity homeo viscous adaptation in teleost lymphocytes. *Proc. Soc. Exp. Biol. Med.* 169:12–18.

Anderson, D.P. 1996. Environmental factors in fish health: immunological aspects. In *Fish Physiology.* W.S. Hoar, D.J. Randall and A.P. Farrell, eds., pp. 289–310. San Diego, California: Academic Press.

Arkoosh, M.R., and Collier, T.K. 2002. Ecological risk assessment paradigm for salmon: analyzing immune function to evaluate risk. *Hum. Ecol. Risk Assess.* 8:265–276.

_____, Casillas, E., Clemons, E., Kagley, A.N., Olson, R., Reno, P., and Stein, J.E. 1998a. Effect of pollution

on fish diseases: potential impacts on salmonid populations. *J. Aquat. Anim. Health.* 10:182–190.

_____, Casillas, E., Huffman, P., Clemons, E., Varanasi, U., Evered, J., and Stein, J.E. 1998b. Increased susceptibility of juvenile chinook salmon from a contaminated estuary to *Vibrio anguillarum*. *Trans. Amer. Fish. Soc.* 127:360–374.

Bagge, A.M., and Valtonen, E.T. 1999. Development of monogenean communities on the gills of roach fry (*Rutilus rutilus*). *Parasitology* 118:479–487.

_____, Poulin, R., and Valtonen, E.T. 2004. Fish population size, and not density, as the determining factor of parasitic infection: a case study. *Parasitology* 128:305–313.

Barker, E., Khan, R.A., and Hooper, R. 1994. Bioindicators of stress in winter flounder, *Pleuronectes americanus,* captured adjacent to a pulp and paper mill in St. George's Bay, Newfoundland. *Can J. Fish Aquat. Sci.* 51:2203–2209.

Barton, B.A., and Iwama, G.K. 1991. Physiological changes in fish from stress in aquaculture with emphasis on the response and effects of corticosteroids. *Annu. Rev. Fish Dis.* 1:3–26.

_____, Schreck, C.B., and Barton, L.D. 1987. Effects of chronic cortisol administration and daily acute stress on growth physiological conditions and stress responses in juvenile rainbow trout. *Dis. Aquat. Organ.* 2:173–186.

Bergstedt, R.A., and O'Gorman, R. 1989. Distribution of alewives in southeastern Lake-Ontario in autumn and winter—a clue to winter mortalities. *Trans. Amer. Fish. Soc.* 118:687–692.

Blaxhall, P.C. 1972. The hematological assessment of the health of fresh water fish a review of selected literature. *J. Fish Biol.* 4:593–604.

Bly, J.E., Quiniou, S.M.A., and Clem, L.W. 1997. Environmental effects on fish immune mechanisms. *Dev. Biol. Stand.* 90:33–43.

Brown, E.H., Jr. 1968. *Population characteristics and physical condition of alewives,* Alosa pseudoharengus, *in a massive dieoff in Lake Michigan, 1967.* Great Lakes Fisheries Commission Technical Report. 13.

Campbell, T.W. 1994a. *Avian hematology and cytology.* Ames, IA: Iowa State University Press.

_____. 1994b. Hematology. In *Avian Medicine: Principles and Application.* B. Ritchie, G.C. Harrison, and L.R. Harrison, eds., pp.176–198. Lake Worth, Florida: Wingers Publishing.

Clem, L.W., Faulmann, E., Miller, N.W., Ellsaesser, C., Lobb, C.J., and Cuchens, M.A. 1984. Temperature mediated processes in teleost immunity differential effects of in-vitro and in-vivo temperatures on mitogenic responses of channel catfish lymphocytes. *Dev. Comp. Immunol.* 8:313–322.

Colby, P.J. 1973. Response of the alewives (*Alosa-pseudoharengus*) to environmental change. In *Responses of fish to environmental changes*. W. Chavin, ed., pp. 163–198. Springfield, Illinois: C.C. Thomas.

Cuchens, M.A., and Clem, L.W. 1977. Phylogeny of lymphocyte heterogeneity. Part 2 Differential effects of temperature on fish thymus derived-like and bone marrow derived-like cells. *Cell. Immunol.* 34:219–230.

Eck, G.W., and Brown, E.H.J. 1985. Lake Michigan's capacity to support lake trout (*Salvelinus namaycush*) and other salmonines an estimate based on the status of prey populations in the 1970s. *Can J. Fish Aquat. Sci.* 42:449–454.

Flath, L.E., and Diana, J.S. 1985. Seasonal energy dynamics of the alewife (*Alosa pseudoharengus*) in southeastern Lake Michigan, U.S.A. *Trans. Amer. Fish. Soc.* 114:328–337.

Goode, G.B. 1884. *The fisheries and fishery industries of the United States.* United States Commission of Fish and Fisheries. Washington, D.C.: Government Printing Office.

Graham, J.J. 1956. Observations of the alewife, *Pomolobus pseudoharengus* (Wilson), in fresh water. *Univ. Toronto Biol. Ser.* 62:1–43.

Gross, R.W. 1959. A study of the alewife *Alosa pseudoharengus* (Wilson) in some New Jersey lakes, with special reference to Lake Hopatcong. Master's thesis. Rutgers State University, New Brunswick.

Hall, D.J., Cooper, W.E., and Werner, E.E. 1970. Experimental approach to production dynamics and structure of freshwater animal communities. *Limnol. Oceanogr.* 15:839–928.

Harris, P.D., Soleng, A., and Bakke, T.A. 2000. Increased susceptibility of salmonids to the monogenean (*Gyrodactylus salaris*) following administration of hydrocortisone acetate. *Parasitology* 120:57–64.

Hartman, K.J., and Brandt, S.B. 1995. Estimating energy density of fish. *Trans. Amer. Fish. Soc.* 124:347–355.

Hazel, J.R. 1993. Thermal biology. In *The Physiology of Fishes.* D.H. Evans, ed., pp 427–467. Boca Raton, Florida: CRC Press.

_____, and Williams, E.E. 1990. The role of alterations in membrane lipid composition in enabling physiological adaptation of organisms to their physical environment. *Prog. Lipid Res.* 29:167–227.

Hodgkins, G.A., James, I.C. III, and Huntington, T.G. 2002. Historical changes in lake ice-out dates as indicators of climate change in New England, 1850–2000. *Int. J. Climatol.* 22:1819–1827.

Hrubec, T.C., Robertson, J.L., and Smith, S.A. 1997. Effects of temperature on hematologic and serum biochemical profiles of hybrid striped bass (*Morone chrysops* x *Morone saxatilis*). *Am. J. Vet. Res.* 58:126–130.

_____, Smith, S.A., and Robertson, J.L. 2001. Age-related changes in hematology and plasma chemistry

values of hybrid striped bass (*Morone chrysops* x *Morone saxatilis*). *Vet. Clin. Path.* 30:8–15.

Iwama, G., and Nakanishi, T. 1996. The fish immune response: Organisms, pathogen and environment. In *Fish Physiology.* W.S. Hoar, D.J. Randall, and A.P. Farrell, eds.,Volume 15. San Diego, California: Academic Press.

Jude, D.J., Tesar, F.J., Deboe, S.F., and Miller, T.J. 1987. Diet and selection of major prey species by Lake-Michigan salmonines, 1973–1982. *Trans. Amer. Fish. Soc.* 116:677–691.

Lantry, J.R. 2001. Spatial and temporal dynamics of predation by Lake Ontario trout and salmon. M.S. thesis. State University of New York College of Environmental Science and Forestry, Syracuse, New York.

Madenjian, C.P., Fahnenstiel, G.L., Johengen, T.H., Nalepa, T.F., Vanderploeg, H.A., Fleischer, G.W., Schneeberger, P.J., Benjamin, D.M., Smith, E.B., Bence, J.R., Rutherford, E.S., Lavis, D.S., Robertson, D.M., Jude, D.J., and Ebener, M.P. 2002. Dynamics of the Lake Michigan food web, 1970–2000. *Can J. Fish. Aquat. Sci.* 59:736–753.

McCarthy, D.H., Stevenson, J.P., and Roberts, M.S. 1975. Some blood parameters of rainbow-trout (*Salmo-Gairdneri Richardson*).II. Shasta variety. *J. Fish Biol.* 7:215–219.

Miller, N.W., and Clem, L.W. 1984. Temperature-mediated processes in teleost immunity—Differential-effects of temperature on catfish in vitro antibody-responses to thymus-dependent and thymus-independent antigens. *J. Immunol.* 133:2356–2359.

O'Gorman, R., and Schneider, C.P. 1986. Dynamics of alewives in Lake Ontario following a mass mortality. *Trans. Amer. Fish. Soc.* 115:1–14.

Pickering, A.D. 1984. Cortisol-induced lymphocytopenia in brown trout, *Salmo trutta L. Gen. Comp. Endocrinol.* 53:252–259.

Pritchard, A.L. 1929. The alewife (*Pomolobus pseudoharengus*) in Lake Ontario. *Univ. Toronto Biol. Ser.* 33:39–54.

Rand, P.S., and Stewart, D.J. 1998. Prey fish exploitation, salmonine production, and pelagic food web efficiency in Lake Ontario. *Can J. Fish. Aquat. Sci.* 55:318–327.

Schroeder, E.H. 1963. *Serial atlas of the marine environment: Folio 2. North Atlantic temperatures at a depth of 200 meters.* New York, New York: The American Geographical Society.

Sherburne, S.W. 1977. Occurrence of piscine erythrocytic necrosis (Pen) in blood of anadromous alewife, *Alosa pseudoharengus*, from Maine coastal streams. *J. Fish. Res. Board Can.* 34:281–286.

Smith, S.H. 1970. Species interactions of alewife in Great Lakes. *Trans. Amer. Fish. Soc.* 99:754–765.

Snyder, R.J., and Hennessey, T.M. 2003. Cold tolerance and homeoviscous adaptation in freshwater alewives (*Alosa pseudoharengus*). *Fish Physiology Biochem.* 29:117–126.

Stanley, J.G., and Colby, P.J. 1971. Effects of temperature on electrolyte balance and osmoregulation in alewife (*Alosa pseudoharengus*) in fresh and sea water. *Trans. Amer. Fish. Soc.* 100:624–638.

Stewart, D.J., Kitchell, J.F., and Crowder, L.B. 1981. Forage fishes and their salmonid predators in Lake-Michigan. *Trans. Amer. Fish. Soc.* 110:751–763.

Valtonen, E.T., Pulkkinen, K., and Julkunen, M. 2003. Getting to the core of the parasite communities: revealing the exchange patterns among sympatric host species. In *Taxonomy, ecology and evolution of metazoan parasites,* C. Combes and J. Jourdane, eds., pp. 293–311. Perpigan, France: University of Perpigan Press.

Wells, L. 1968. *Seasonal depth distribution of fish in southeaster Lake Michigan.* U.S. Fish and Wildlife Service, Fisheries Bulletin. 67.

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