

Sea Lamprey (*Petromyzon marinus*) Parasite-host Interactions in the Great Lakes

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ABSTRACT. Prediction of how host mortality responds to efforts to control sea lampreys (*Petromyzon marinus*) is central to the integrated management strategy for sea lamprey (IMSL) in the Great Lakes. A parasite-host submodel is used as part of this strategy, and this includes a type-2 multi-species functional response, a developmental response, but no numerical response. General patterns of host species and size selection are consistent with the model assumptions, but some observations appear to diverge. For example, some patterns in sea lamprey marking on hosts suggest increases in selectivity for less preferred hosts and lower host survival when preferred hosts are scarce. Nevertheless, many of the IMSL assumptions may be adequate under conditions targeted by fish community objectives. Of great concern is the possibility that the survival of young parasites (parasitic-phase sea lampreys) varies substantially among lakes or over time. Joint analysis of abundance estimates for parasites being produced in streams and returning spawners could address this. Data on sea lamprey marks is a critical source of information on sea lamprey activity and potential effects. Theory connecting observed marks to sea lamprey feeding activity and host mortality is reviewed. *Uncertainties regarding healing and attachment times, the proba-*

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bility of hosts surviving attacks, and problems in consistent classification of marks have led to widely divergent estimates of damages caused by sea lamprey. Laboratory and field studies are recommended to provide a firmer linkage between host blood loss, host mortality, and observed marks on surviving hosts, so as to improve estimates of damage.

INDEX WORDS: Sea lamprey, functional response, lake trout, predation, wound, scar.

INTRODUCTION

The intense interest in sea lamprey (*Petromyzon marinus*), especially among biologists who work on the Great Lakes, stems from damages caused by sea lamprey on their host populations during their parasitic juvenile phase. Despite this, there remain major gaps in understanding of parasite-host interactions, and the general ecology of the parasitic phase. This largely stems from the difficulty of observing either sea lamprey or sea lamprey feeding events within large water bodies. While important work has been done since the 1979 Sea Lamprey International Symposium (SLIS-I) (Smith 1980, Fetterolf 1980), study of the parasitic phase needs to be emphasized if the promise of the Integrated Pest Management (IPM) approach is to be realized.

In this paper a review and synthesis of the understanding of these parasite-host interactions, especially new developments since SLIS-I, is presented. Results and ideas presented at SLIS-I had a substantial influence on the subsequent direction of sea lamprey management and research. Of critical importance was the adoption the IPM approach to sea lamprey (Sawyer 1980). Central to this IPM approach is determination of an "economic injury level," which specifies the optimal level of control. Control efforts are generally directed toward spawning adults or larvae and measured in terms of changes in the number of parasitic juveniles that are produced. Because economic damages primarily result from the death of large host fish, such as lake trout (*Salvelinus namaycush*), this led to the development of models intended to predict host losses, given the number of metamorphosing juveniles and information on host abundance (Walters *et al.* 1980, Jones *et al.* 1993, Koonce *et al.* 1993). The development of predictive models also influenced laboratory and field research, with an increased emphasis on estimating parameters needed for the models, such as the probability of a host surviving a sea lamprey attack. Uncertainty in this area was emphasized by several papers from SLIS-I, which suggested that uncontrolled sea lamprey populations might do little damage to hosts such as large lake trout, at least under some conditions (Christie and Kolenosky 1980, Farmer 1980, Kitchell and Breck

1980, Youngs 1980), in contrast with observations of severe damages (Moore and Lychwick 1980, Pearce *et al.* 1980, Pycha 1980, Spangler and Collins 1980, Spangler *et al.* 1980, Swanson and Swedberg 1980, Walters *et al.* 1980).

The emphasis of this paper is on predicting and measuring host deaths (especially those of large hosts such as lake trout) caused by parasitic-phase juvenile sea lamprey. First the current parasitic-phase model used in the Integrated Management of Sea Lamprey (IMSL) model is briefly described (Koonce *et al.* 1993, Larson *et al.* 2003). The IMSL model forms a conceptual basis for control efforts throughout the Great Lakes, and has been used directly to establish an economic injury level for Lake Ontario (Koonce *et al.* 1993) and to estimate host deaths in that lake (Larson *et al.* 2003). By necessity (as is true for all models), the parasitic-phase IMSL model makes a number of assumptions and simplifications, and still contains parameters that are difficult to estimate. After describing the IMSL model, information on complexities involved in the ecology of the juvenile stage and parasite-host interactions that challenge the model, and on efforts to estimate critical parameters required by the model is presented.

The interpretation of how marks left on surviving hosts by sea lamprey can be interpreted, and how such data and other information can be used to estimate and predict the effects of sea lamprey, is considered in some detail. Attacks on hosts are rarely observed directly, and dead hosts with wounds are seen infrequently (but see Bergstedt and Schneider 1988, Schneider *et al.* 1996). Hence marks left by sea lamprey on surviving hosts are a critical source of information. These marks have been used both as a means to estimate host mortality caused by sea lamprey (Pycha 1980, Eshenroder *et al.* 1995, Sitar *et al.* 1999), and as a means to calibrate predictive models of sea lamprey feeding (Larson *et al.* 2003). The discussion of assumptions underlying the interpretation of marking data are widely distributed in a series of "gray literature" reports, and we bring this information together and expand upon it.

Finally, new laboratory (Cochran *et al.* 1999, Swink 2003 and references therein) and field data (Bergstedt and Schneider 1988, Bergstedt and

Swink 1995, Bergstedt *et al.* 2003a) are used as a basis for calculating “back of the envelope” estimates of possible lake trout deaths, and these are compared to other estimates of host losses across the Great Lakes as a check on the consistency of the underlying assumptions involved in these calculations. The review presented herein indicates a large amount of uncertainty regarding the number of host fish killed by sea lamprey and unknowns and future directions in attempting to better predict host deaths caused by sea lamprey are discussed. In particular, field and laboratory studies and data analysis to better link host blood loss, host mortality and observed field marking on surviving hosts are called for.

THE IMSL PARASITIC PHASE SUBMODEL

Predicting mortality of host fish caused by sea lamprey depends on models for the functional, numerical, and developmental responses. The functional response is the relationship between a predator’s (or parasite’s) attack rate and prey density (Solomon 1949, Holling 1959). The numerical response reflects how predator numbers change in response to their prey abundance (Holling 1959, 1961; Rosenzweig 1971). The developmental response describes how predators might grow faster and, as a result, consume more prey when their prey are abundant (Murdoch 1971). All explicit functional response models previously developed for sea lamprey (including IMSL) have been type 2 (Fig. 1), assuming that the rate at which individual sea lamprey attack prey will increase with host density, but reach a maximum at high prey densities, at which point they spend essentially all their time either attached to hosts or in a latent “satiated” condition with no time available for search and encounter with additional hosts.

The parasitic-phase submodel of the IMSL operates in discrete time, with annual host mortality determined by a single set of host and sea lamprey abundances representing summer-autumn conditions when lethal attacks are most likely. The submodel centers on a functional response, with additional assumptions connecting attack rates to host mortality and observed marking data. There is no within-generation numerical response. That is, the number of parasites is assumed to be directly proportional to the number of metamorphosing juveniles, with a proportionality constant representing survival of parasites to the summer-autumn period when lethal attacks occur. While the survival of parasites to the late summer may not depend heavily upon the abundance of

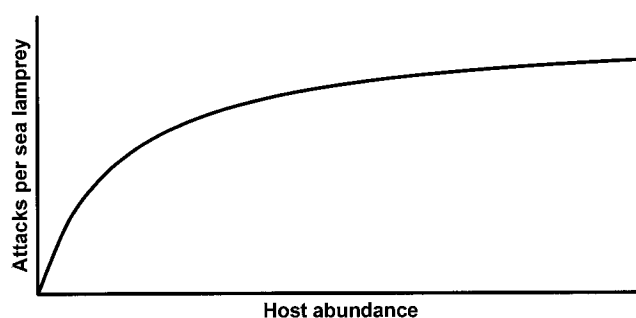


FIG. 1. Relationship between total attacks per sea lamprey in a season (a) and the abundance of hosts (N) assumed by equation 1. The graphed relationship is calculated with just one type of host present, but shows the general effect of increasing host density that is incorporated in the multi-species functional response equation.

larger hosts, below it is argued that survival to this time may vary in response to hosts available for young parasites (Stewart *et al.* 2003). Sea lamprey size is assumed to increase when hosts are more abundant, thereby incorporating a developmental response. This model has been applied at the whole-lake or major-basin spatial scale, assuming parasitic-phase sea lamprey are well mixed on this scale. This is partially supported by the observation that at the completion of the parasitic phase, spawning-phase sea lamprey do not home to their natal stream (Bergstedt and Seeleye 1995, Bergstedt *et al.* 2003b). As is discussed below, however, there are within-basin variations in sea lamprey feeding activity (between inshore and offshore or along shore), and this can pose a difficulty when applying and parameterizing this model.

The IMSL parasitic phase submodel is currently in use for Lake Ontario (Larson *et al.* 2003), a preliminary model has been developed for Lake Erie (Joseph F. Koonce, unpublished analysis), and an earlier version was evaluated for Lake Superior (Koonce 1986) but is no longer in use. The IMSL parasitic phase submodel was described by Koonce (1986, 1987), Koonce and Locci-Hernandez (1989), and Greig *et al.* (1992). Koonce *et al.* (1993) describe the IMSL model in a broader context. A related submodel for the parasitic phase was used in the SIMPLE fish community model (Jones *et al.* 1993). These submodels evolved from similar models developed in adaptive management workshops (Koonce 1982, Spangler and Jacobson 1985), which in turn built upon the initial modeling work by Walters *et al.* (1980) arising from SLIS-I. All these

models used a multispecies type 2 functional response (Murdoch 1973):

$$a_i = \frac{F\lambda_i N_i}{1 + \sum_j \lambda_j N_j h_j} \quad (1)$$

where a_i is the number of attacks on host type i by an individual parasite, F is the length of the feeding season, N_i is the density of host type i , λ_i is the effective search rate on host type i , and h_i is the attachment time for host type i (Fig. 1). As currently implemented, h is assumed the same for all host types. Although handling times will really vary among host types, this assumption is not as extreme as it first appears because it is applied only to the larger host species that form a substantial portion of the diet during the summer and autumn feeding season that is being modeled. The ratio of season length to attachment time (F/h) is a critical parameter as it is the maximum number of attacks a parasite can make during a feeding season. Current parameters set this maximum at 7.5 ($F = 0.41$ years, $h = 0.0548$ years (20 days), Koonce and Locci-Hernandez 1989). When hosts are abundant the attack rate approaches this maximum and no longer depends upon the effective search rates. The relative values of the effective search rates still determine how attacks are distributed among hosts. The effective search rates are assumed constant and thus are proportional to the relative rates at which individuals of different types of hosts are encountered by reactive (unattached) sea lamprey.

The effective search rates are determined as the product of four sub-components, the distance swum by a host during the attack season (S), the reactive area (R_a), the probability of attack once a host is encountered (P_a), and habitat overlap (H). Habitat overlap can be viewed as the fraction of the total search time the sea lamprey occurs in habitat occupied by the host type, and is intended as a simplified representation of the complex spatial relationships among host species and sea lamprey. These are set as host species or strain-specific constants. The other three components are functions of host length (Table 1). The formulation envisions sea lamprey as an ambush predator which encounters prey as they move through a plane defined as the reactive area. This reactive area is determined by a reactive distance, which is assumed to be directly proportional to host length up to some maximum. Because encounters occur via host movement through a reactive area, they depend upon the distance swum by hosts during an attack season, which is also a function of host

length. Once a host is encountered, the probability it is attacked is modeled as an asymptotically increasing function of host length. While the model formulation is framed mechanistically, as though parasite-host interactions occur in a homogenous environment, development of parameter estimates has reflected a more phenomenological approach in recognition of the spatial complexities of host and sea lamprey distributions.

The functional response model is used to generate predictions of instantaneous mortality due to sea lamprey (M_L) and the total number of attacks per host. The total number of attacks per host, together with the probability of surviving an attack and healing time, are used together to predict marking data (see "Measuring Sea Lamprey-Induced Mortality"). The mean total number of attacks per individual host of type i is given by $A_i = a_i L / N_i$, where L is the number of parasitic lamprey, and a_i is from equation 1. Then, approximately

$$M_{L,i} = (1 - P_{S,i}) A_i \quad (2)$$

where $P_{S,i}$ is the probability a host of type i will survive the attack.

This approximation treats the attacks as occurring evenly over the year, so that A_i is both the total number of attacks and the annual rate of attacks. With A_i defined as the attack rate, the derivation of equation 2 is outlined in the next section. This approximation will work reasonably well even when attack rates are strongly seasonal, unless the total mortality is quite high ($Z \geq \sim 1.0$). When Z is extremely high, within-season depletion of hosts is severe and equation 1 will not be an adequate predictor of total attacks because it is based on the number of hosts present at the beginning of the season. The probability of survival ($P_{S,i}$) is modeled as dropping from a maximum for small ratios of sea lamprey to host weight to near zero for large ratios (Table 1). Host weights vary with species and age, and for lake trout, growth is assumed to be density dependent. In particular, based on observed data from the Great Lakes, annual increments in lake trout weight are assumed to follow a Walford equation, with intercept and slope modeled as linear functions of lake trout population biomass (Koonce 1986, Table 1). There is some potential for simplifying this part of the model if incremental growth in length could be modeled by a Walford equation and only the slope depended upon population biomass (Walters and Post 1993). Average weight of parasitic-phase sea lamprey is assumed to be an asymp-

TABLE 1. *IMSL parasitic submodel functions referred to in the text.*

Function Description	Function	Parameters
Probability of survival given an attack	$P_S = P_{\max}(1 - PR^2/(PR^2 + c_1))$	P_S = probability of survival for specified host type. P_{\max} = maximum probability of survival. PR = ratio of sea lamprey to host weight. c_1 = specified constant.
Probability of attack given an encounter	$P_a = (A L_i^n)/(Bn + L_i^n)$	P_a = probability of attack given an encounter with specified host type i . L_i = Length of host type i (mm). A, B, n = function coefficients.
Reactive area	$R_a = \pi(\max\{R_c L_i, 0.006 \text{ km}\})^2$	R_a = Reactive area for specified host type (km^2). R_c = Reactive distance per length of prey (km/mm).
Distance swum by host in attack season	$S_i = 7.884 L_i$	S_i = distance swum (km). The constant 7.884 (km/mm attack season) assumes an average swimming speed of 0.61 body lengths/s.
Lake trout growth	$W_{a+1, t+1} = w_k + r W_{a, t}$ $w_k = a_w + b_w \text{Stock}$ $r = a_r + b_r \text{Stock}$ $a_w = 0.2, b_w = -0.1, a_r = 3.0,$ $b_r = -0.15$: for $a < 3$ $a_w = 1.3, b_w = -0.5, a_r = 0.9,$ $b_r = -0.15$: for $a \geq 3$	$W_{a, t}$ = weight (kg) at age a in year t . w_k, r = Walford parameters. a_w, b_w, a_r, b_r = intercept and slopes determining relationship between Walford parameters and Stock. Stock = lake trout biomass density (metric tons per km^2).
Sea lamprey weight dependence on host density.	$W_L = W_{\max} q / (c_2 + q)$ $q = \sum h \lambda_i N_i / (1 + \sum h \lambda_i N_i)$	W_L = average sea lamprey weight (kg). W_{\max} = constant that determines maximum possible average sea lamprey size. c_2 = specified constant. h = attachment time. λ_i = effective search rate on host type i . N_i = host density.

totically increasing function of host density (Table 1). Thus, although the parameters of the sea lamprey functional response do not depend upon sea lamprey size, the observed developmental response seen in the Great Lakes (Heinrich *et al.* 1980, Johnson and Anderson 1980, Kitchell 1990, Houston and Kelso 1991) is incorporated by modeling sea lamprey size as host-density dependent together with a functional relationship between the probability of a host surviving an attack and parasite size.

MEASURING SEA LAMPREY-INDUCED HOST MORTALITY

Changes in total mortality of host fish have been linked to the frequency of sea lamprey wounds, the rate at which sea lamprey scars accumulate, or estimates of the abundance of sea lamprey (Budd *et al.* 1969, Pycha 1980, Spangler *et al.* 1980, Eshenroder

et al. 1995). Non-lethal injuries resulting from attempted predation have been used to make inferences regarding predation for a wide range of other organisms, including terrestrial and aquatic vertebrates (Schoener and Schoener 1982, Morin 1985, Mushinski and Miller 1993, Shargal *et al.* 1999), crustaceans (Murtaugh 1981, Juanes and Smith 1995), and other invertebrates (Oji 1996, Peddle and Larson 1999, Kowalewski and Flessa 2000).

The logical basis for the approaches applied to sea lamprey marking data are discussed here. Marks are referred to as "wounds" and "scars." Wounds are marks that heal to become scars, and scars are assumed to persist so they reflect the activities of several cohorts of sea lamprey. Following King's (1980) nomenclature, type A marks are those where the skin at the attachment site is broken, exposing the underlying musculature, and type B marks appear as abrasions or elongated scrapes with no visi-

ble break through the integument. Each type of mark can be in stages 1 to 4 of healing, where stage 1 indicates the most recent attack with the least healing. Here marks are referred to using this system, as for example A3, meaning a type A mark in stage 3. For the theory discussed below some subset of type A marks are classified as wounds, whereas later stages of type A marks are classified as scars.

Theory Underlying Interpretation of Wounding and Scarring Data

The theory underlying interpretation of sea lamprey marking on surviving hosts was developed in a series of Great Lakes Fishery Commission (GLFC) Completion and Progress Reports (Koonce and Pycha 1985, Koonce 1986, and Koonce *et al.* 1987), and by Eshenroder and Koonce (1984), Koonce and Locci-Hernandez (1989), and Greig *et al.* (1992). In Appendix 1, the results are extended to the case of temporal variation in both attack rates and the probability of surviving an attack. A closely related model for interpreting sea lamprey marks is also discussed (Robson 1970).

Models for interpreting non-lethal marks or wounds have been developed in the context of other systems also. Schoener (1979) considered the case where the mark is permanent (akin to a “scar”) and showed that if the injury producing agent was the only source of mortality, then mark frequency and predation intensity were unrelated and marks only provide information on the success rate of an attack. When other sources of mortality exist, he showed that mark frequency and predation intensity would generally be positively related, but only weakly. He advocated using models to correct raw marking data, but also emphasized the difficulties involved. Murtaugh (1981) considered the case where injuries heal quickly, in the form of a simple deterministic model, and applied the model to infer predation rates by Mysids from observed marking frequency in *Daphnia*. In this case, one can expect a close relationship between wounding frequency and predation intensity.

These earlier efforts at making inferences from marking data support the general approach to marking in the Great Lakes, which has emphasized using recent marks or increments in marks, rather than all marks that can be attributed to sea lamprey activity. Thus, the case addressed here is most closely related to that considered by Murtaugh (1981). The models and theory presented differ in that they have a probabilistic basis, allow for multiple attacks dur-

ing the healing period (or the time interval during which marks are assumed to accumulate), and can allow for the situation where the observation process interrupts an attack.

In the “Constant Rates” section it is shown that host mortality caused by sea lamprey can be expected to be proportional to observed wounds per host, with a proportionality that depends upon the probability of a host surviving an attack. This relationship becomes more complex when fish are collected while lethal attacks are ongoing. In the “Seasonally Varying Rates” section it is argued that the proportionality between mortality and wounding is retained when attack and survival rates vary, but that it might not be possible to infer the probability of surviving an attack from the relationship between total mortality and observed wounding rates. Methods suggested by Robson (1970), which can be used to evaluate seasonal patterns in the lethality of an attack, are also discussed and it is noted that these methods assume a class of fresh wounds that are observed before mortality occurs and require information on attachment time.

Constant Rates

First the fact that attack rates and the probability of surviving an attack vary seasonally and with host size (for a given season and associated size of sea lamprey) is ignored and these topics are returned to below. For simplicity in presentation subscripts denoting host types have been dropped.

Attacks are assumed to follow a Poisson process. That is, during a short period of time t to $t + \delta$, the probability that an individual host will be attacked is $A\delta$, the probabilities are independent among such short intervals, and the probability is low for multiple attacks within any short interval. The attack rate, A , and the probability of surviving an attack, P_S , are assumed constant, and it is further assumed that the probability of surviving a given attack does not depend upon the existence or timing of other attacks on the host. Therefore, the instantaneous mortality rate is the product of the attack rate and the probability of dying from an attack (equation 2) and marks accumulate on surviving fish at rate:

$$m = AP_S \quad (3)$$

Combining equations 2 and 3 gives the relationship between the rate of instantaneous mortality and marking rate:

$$M_L = m(1 - P_S) / P_S \quad (4)$$

If observed marks are wounds that accumulated over some defined time period of length T (say the amount of time it takes a “wound” to heal and become a scar), then the relationship between lamprey-induced mortality and “expected” wounds per fish $E(W)$ is given by

$$M_L = [E(W) / T][(1 - P_S) / P_S] \quad (5)$$

Although a complete test of the assumptions underlying this relationship has not been done, previous reports of frequency distributions of wounds have been consistent with the Poisson distribution that is predicted by this model (Eshenroder and Koonce 1984). These tests have been based on small to moderate samples because analysis was restricted to groups (e.g., size categories or sampling locations) expected to be attacked at similar rates. Rutter and Bence (2003) have partially circumvented this problem by fitting a generalized nonlinear model to the data on wounding (A1 to A3 type marks combined) observed on individual lake trout caught in spring surveys in Lake Huron. This model allowed different expected wounding rates for each fish (depending upon factors such as lake region, year, and fish length), but allowed a joint test of the assumed Poisson distribution. Although they discovered significant hyper-dispersion, suggesting that multiple wounds were more common than expected under the Poisson assumption, the magnitude of the departure from the assumed Poisson model was small. Their results suggest that a theory based on an assumed Poisson processes is still a credible approximation.

If sea lamprey-induced mortality and other mortality sources operate independently (so called “competitive mortality”), then **total instantaneous mortality** is linearly related to expected wounding:

$$Z = c + bE(W) \quad (6)$$

$$b = \frac{1 - P_S}{P_S T}$$

Here c represents the instantaneous rate of “other” mortality. If this other mortality is constant over time, and estimates of Z (\hat{Z}) and mean wounding (\bar{W}) are available, the linear relationship suggests that linear regression of \hat{Z} on \bar{W} could be used to estimate other mortality (c) and the proportionality between lamprey induced mortality and mean wounds (b). If the time period over which wounds accumulate were known, the probability of surviving an attack (P_S) could be estimated from the re-

gression results. If fishing mortality is negligible, or its magnitude is well estimated, the rate of natural mortality (excluding sea lamprey effects) can be deduced from the estimated intercept. Strictly speaking, such a regression assumes that the mean wounding values are measured without error. Practically, the measurement error simply needs to be small relative to the observed range of mean wounding to avoid substantial bias.

In practice, observed wounding will be composed of a mixture of (a) wounds from attacks where lamprey had finished feeding on their host prior to sampling and (b) wounds from attacks where lamprey feeding was interrupted by the sampling. The interpretation of the regression results described above assumes that the observed wounds are of the first type (a), because interrupted attacks will include some that the host would not have survived. This seems most likely to be the case for spring surveys in contrast with autumn surveys, since sea lamprey are feeding much more actively in the autumn. When interrupted attacks are common, observed wounding will be greater and the apparent probability of surviving attacks greater, if this is not taken into account. The expected number of wounds resulting from completed attacks is ATP_S , and the additional expected wounds due to interrupted ongoing attachments is Ah , where h is the attachment time. Thus, the proportion of wounds that are from completed attacks is $P_C = TP_S / (TP_S + h)$. This changes the relationship between sea lamprey-induced mortality and the mean or expected wounds per fish to:

$$M_L = [P_C E(W) / T][(1 - P_S) / P_S] \quad (7)$$

For a given level of wounding the sea lamprey-induced mortality will be lower, and the decrease will be more substantial when attachment time is large relative to the product of healing time and the probability of surviving an attack. This result can lead to large quantitative effects. For example, if wounds heal in 20 days (Ebener *et al.* 2003), the probability of surviving an attack is 0.5 (Swink 2003), and attachment time is 10 days then $P_C = 0.5$. Note, however, that sea lamprey mortality remains directly proportional to mean wounds per fish.

Seasonally Varying Rates

To this point attack rates and the probability of surviving an attack have been treated as constant over the observation period. The evidence for seasonality in attack rates and survival, with higher at-

tack rates and lower survival in the late summer and autumn, is convincing (see below). This poses two potential problems. First the theory given above was based upon a Poisson process, assuming constant rates and survival during the period that wounds accumulated. Second, the interpretation of regression results to estimate the probability of surviving an attack requires that the sea lamprey mortality rate during the period wounds have accumulated to be representative of rates over the entire year. Both of these issues complicate parameter estimation and interpretation, but neither seriously compromises the expected direct proportionality between the sea lamprey component of mortality and mean wounds.

The assumptions of a Poisson process can be relaxed to allow time varying rates of attack and survival from attacks (Appendix 1). The resulting non-homogeneous Poisson processes still lead to an expected Poisson distribution for the observed wounding frequency. In addition, for appropriately defined time averages of $A(t)$ and $P_S(t)$, the relationships between sea lamprey-induced mortality and attack rates or, average number of wounds per fish given by equations 2, 5, 6, and 7, still hold approximately, provided total mortality rates are not very high. There are, however, significant difficulties in estimating these time averages (Appendix 1). Clearly, seasonally varying sea lamprey mortality clouds the interpretation of the relationship between total annual mortality and mean sea lamprey wounds per fish, because the wounding statistic may not be representative of the entire year. However, if the rate of lethal attacks per host during the period the observed wounds are accumulated is proportional to the rate of lethal attacks in the rest of the year, the direct relationship between the sea lamprey component of mortality and mean wounds per fish is preserved. This suggests that the rate of sea lamprey mortality for a given observed number of wounds per fish could be estimated by the regression of total mortality on wounds per fish, but estimating the actual average probability of surviving an attack from the regression may not be possible.

Robson (1970) also proposed a nonhomogeneous Poisson process model for sea lamprey attacks and derived associated estimators of total and non-fatal attack rates. His underlying model of the attack process was identical to that described in Appendix 1. However, his approach assumes a class of "fresh" wounds that can be observed before any mortality or healing occurs. Robson (1970) viewed these wounds as resulting from attachments that were interrupted

by the observation process. While it might be possible to identify such very recent wounds (Mark P. Ebener, personal observation), and the approach is reasonable if such wounds are identified, such mark classification is currently not standard protocol in the Great Lakes (Ebener *et al.* 2003). The proposed estimator for the total attack rate (A) was:

$$l_t = \frac{w_t}{v_t} \quad (8)$$

$$\hat{A}(t) = \frac{l_t}{\hat{h}_t}$$

where w is the number of potential hosts collected at time t with one fresh wound and no other recent wounds, v is the number of potential hosts with no fresh or recent wounds, and h is the attachment time (or other period during which wounds can accumulate but mortality and healing of fresh wounds has not begun). The restriction to potential hosts without other recent wounds stems from a concern that fish with other recent wounds would be more subject to stress and thus attachment times to them would be less than seen in laboratory observations on which estimates of h would be based. If the non-homogeneous process can be approximated by a Poisson process with constant rates during the period the fresh wounds accumulated, l_t is an unbiased estimator of the Poisson parameter (expected number of fresh wounds per fish). If concerns about changing attachment times for previously- or multiply-wounded hosts are discounted, then the mean number of fresh wounds per host is another valid estimate of this quantity.

Robson (1970) viewed total attacks as consisting of lethal and non-lethal attacks. It was proposed that the rate of non-lethal attacks could be estimated by the rate of accumulation of new scars on tagged fish, and specific estimators are described by Spangler *et al.* (1980) and Robson (1970). Spangler *et al.* estimated the rate of lethal attacks as the difference between the total and non-lethal attack rates, and applied this method to estimate attack rates on lake whitefish (*Coregonus clupeaformis*) in Lake Huron (see "Observed Relationships Between Mortality and Marking").

Predicting Marking from Estimates of Sea Lamprey and Host Abundance

The IMSL Parasitic Phase Submodel has been used to predict marking rates observed in surveys of lake trout, and model parameters (mainly the

maximum probability of survival when the ratio of sea lamprey size to lake trout size was small) have been adjusted so that the observed and predicted marking rates match. In recent years this approach has only been followed for late-summer and autumn survey data. For such surveys, combining equations 2 and 7 (dropping subscripts for host type) yields:

$$E(W) = A(TP_s + H) \quad (9)$$

where A can be calculated from equation 1, and it is assumed that A and P_s are roughly constant over the period observed wounds have accumulated (a period before the time of the survey equal to the sum of attachment time plus healing time). The expected or predicted mean number of wounds and scars (healed wounds) accumulated in a year by a surviving host is AP_s . Average number of scars on a given year class at the end of the parasitic season is determined by adding this number of new marks to the number of scars retained from attacks in previous years. The number of retained scars is simply the number of scars present the previous year multiplied by an assumed proportion of scars that are retained (currently 0.9, Koonce and Locci-Hernandez 1989).

Mark Classification, Healing Time, and Interpretation

Theory presented above suggests that there should be an observable relationship between mortality and mean wounds per fish. This presumes, however, that “wounds” can be consistently classified and that observed “wounds” can be associated with a specific year for which the mean wounds per fish will be roughly proportional to mortality caused by sea lamprey. For example, for spring data A1 through A3 marks have generally been considered wounds that represent attacks from the previous year.

Ebener *et al.* (2003) present striking and disturbing information on both mark classification and healing. They show that there is a large amount of variability among readers in mark classification and consistent differences among agencies. Although Great Lakes agencies had on paper adopted the classification system of King (1980) and the protocol of Eshenroder and Koonce (1984), practice during the past 15 years has not necessarily followed the protocols. A substantial part of the variation was due to differences in interpretation of classification criteria, especially in how multiple marks ap-

parently made by the same sea lamprey were counted. They found that some of the variability could be reduced through training, but that even after training substantial among-reader variation remained. This was especially true for type A3 wounds, an important component of spring marking data. This latter result was also reported by Eshenroder and Koonce (1984).

There is surprising little information in the literature on healing times for marks on lake trout or other hosts. Consequently, Ebener *et al.* (2003) analyzed data on healing times collected as part of King's (1980) study. They found that healing times varied greatly among individual lake trout, and that there was a strong negative relationship between healing time and the temperature lake trout experience. The results suggest that at temperatures lake trout typically experience in the Great Lakes (Bergstedt *et al.* 2003a), some attacks in the late summer and autumn would still be classified as stage A3 or earlier at the time of a spring survey the next year, but they do not rule out the possibility that a substantial proportion would heal to the A4 stage during this period. The results also suggest that some attacks made by the cohort of sea lamprey returning to spawn in the spring of a year could lead to marks classified as A3 in the late summer. This result agrees with Schneider *et al.*'s (1996) observation that the number of A3 wounds in late summer were positively correlated with A1 wounds from the late summer of the year before.

Observed Relationships Between Mortality and Marking

In the previous sections it has been argued that a positive relationship between mortality caused by sea lamprey and wounds per fish is to be expected. This provides a theoretical framework supporting the general approach of Budd *et al.* (1969) and Pycha (1980), who analyzed increments in scars and spring wounding data, respectively. Pycha (1980) regressed estimates of total instantaneous mortality against the percentage of lake trout that were wounded and found a strong positive relationship, and Budd *et al.* (1969) found a positive relationship between mortality estimates and the percentage increase in scarring frequency of a year class from one year to the next. Greig *et al.* (1992), building on Eshenroder and Koonce (1984), Koonce and Pycha (1985), and Koonce *et al.* (1987), describe a protocol for this sort of analysis that differs somewhat from these approaches. First, on the basis

of theory like that described above, mean wounding is preferred over the percentage of fish that are wounded because the former leads to an expected linear relationship. Second, Greig *et al.* (1992) suggest calculating the mean wounds per fish as a weighted average of wounds per fish in different size classes, with weights equal to the catch per effort in assessment netting. This is equivalent to simply calculating the average wounds per fish over all fish in assessment netting. Pycha used as his percentage wounded the unweighted average of percentage wounded in three size categories (53.3–63.3 cm, 63.4–73.3 cm, > 73.3 cm). Budd *et al.* (1969) circumvented this issue by calculating separate scar increments for each age and year class, but as a result, assumed equal probability of surviving an attack down to quite small sizes. These contrasting approaches to weighting reflect concerns with different issues. Pycha used the unweighted average so that changes in the percentage wounding would not simply reflect changes in the size composition of the host population because of the higher wounding rates experienced by larger hosts. The protocol of Greig *et al.* is based on the concern that uncertain wounding rates stemming from small sample sizes associated with the largest lake trout size classes would overly influence results. These alternatives reflect a trade-off between bias and variance with no clearly defined “right” answer. Methods recently developed for estimating the relationship between mean wounds per fish and fish size (Rutter and Bence 2003) hold some promise for allowing reasonably accurate estimates of wounds per fish for specific sizes and ages of hosts. In spite of the substantial differences, the protocol given in Greig *et al.* (1992) produces results qualitatively similar to those of Pycha (1980) when applied to data from the same study (Koonce and Pycha 1985).

A more fundamental concern with the approach used by Pycha, however, is that the estimates of total instantaneous mortality came from catch curves applied to year-specific age compositions, which largely reflect mortality in the past rather than mortality contemporaneous with the lamprey wounding data (Ricker 1975). Koonce (1986) used simple simulations to show that the pattern of temporal variation in total mortality can influence the observed relationship between catch curve estimates of total mortality and wounding, and real relationships can be obscured. Apparently Pycha was only able to detect a relationship between total mortality and wounding because fishing mortality was

roughly constant and the change in mortality due to sea lamprey was sustained long enough. Pycha decided not to use cohort methods because there appeared to be strong variations in catchability from year to year. An analysis using cohort methods suggested similar levels of sea lamprey mortality to that of the original analysis (and suggested that the probability of surviving an attack did not vary much over the ages considered), but apparently high variability in catchability from year to year caused all the estimated effects to be nonsignificant (Koonce 1986). Advances in methods for estimating mortality from age-structured models in the last 20 years (Quinn and Deriso 1999) should allow some of the variation associated with year-to-year changes in catchability to be extracted. Such models are now relatively easy to implement, and experience with such models has grown rapidly in the Great Lakes fishery science community during the past several years. The major remaining impediment is the large amount of effort that is required to organize the necessary data. It is recommended that effort be devoted to applying such age structured assessment models in future evaluations attempting to relate total mortality to observed wounding.

The results of Youngs' (1980) study of sea lamprey effects on lake trout in Cayuga Lake provide a striking contrast to those of Pycha (1980) and Budd *et al.* (1969). The data examined by Youngs included Robson-Seber estimates of total mortality and fishing mortality, mean wounds per fish, and mean annual increment in scars per fish (based on autumn samples). Their approach was to construct regression models relating total mortality to fishing mortality and either wounds per fish or the increment in scars per fish. They found no significant effect of either wounds or increments in scars and a strong significant effect of fishing mortality. In their analysis, they treated the Robson-Seber estimate of fishing mortality as an index because of the likelihood of significant non-reporting of tag recoveries. The non-significant relationship between total mortality and wounds per fish was actually negative, and the direction of this relationship did not change when nominal fishing mortality was included as an additional explanatory variable in the regression.

Their failure to find a significant relationship between total mortality and wounds per fish is not surprising, given the lack of contrast in the observed wounds per fish (range from 0.42 to 0.60, Koonce *et al.* 1987). It is likely that measurement error in wounds per fish was large relative to this

range (Ebener *et al.* 2003), decreasing the power for detecting a significant effect and biasing downward the slope parameter for this variable. In addition, in Youngs' (1980) study, age-specific variations in mortality combined with variations in the number of tags recovered in different years and time varying mortality could have distorted the total mortality estimates. These kinds of problems led Ricker (1975) to recommend against use of the Robson-Seber estimator when age-dependent mortality seemed likely. Because fishery tag returns are used in estimates of total and fishing mortality, the combination of age-specific mortality and temporal variations in tag return rates introduce correlations between estimates of total and fishing mortality (James R. Bence, unpublished results). Although this could have led to some temporal variation in mortality being explained erroneously by fishing instead of sea lamprey, it would not cause estimated mortality rates to be grossly underestimated.

Notwithstanding these problems, any positive proportionality between mortality and wounds per fish must have been much less in Cayuga Lake than has been observed by Pycha (1980). The average wounds per lake trout was 0.53 in Cayuga lake, and if the increment in mortality per wound was the same as estimated from Pycha's data by Koonce and Pycha (1985), this implies a instantaneous mortality rate due to sea lamprey of over 3.0, which is incompatible with total mortality rate estimates and age compositions reported by Youngs (1980). Although marking rates can vary greatly seasonally (Jacobson 1989) and by several fold among observers from different agencies even when observing the same fish (Ebener *et al.* 2003), it seems unlikely that methodological differences can explain away Youngs' observations. Similar high wounding rates on lake trout suffering moderate levels of mortality have more recently been observed in Lake Champlain (Marsden *et al.* 2003).

Although correlations between marking statistics and estimates of sea lamprey abundance have been reported (Smith *et al.* 1974), the most compelling evidence for the relationship between marking and host mortality comes from an innovative study on Lake Ontario, in which lake trout carcasses were surveyed by trawling (Bergstedt and Schneider 1988, Schneider *et al.* 1996). From 1982 through 1992 trawl samples collected during the 1 October to 15 November period from U.S. waters were assessed for dead lake trout. Additional trawl sampling was done during spring and summer and after 15 November in some years, but few dead lake

trout were recovered, even though substantial effort was exerted (Bergstedt and Schneider 1988). Live lake trout were sampled by gill net during September for the same years. Schneider *et al.* (1996) found a strong positive correlation between the density of dead lake trout with recent (A1 or A2) sea lamprey marks and total number of A1 marks observed on live lake trout (summed over all live lake trout collected with a standard amount of gillnet effort). Nearly all dead lake trout had recent sea lamprey marks, whereas a relatively small fraction of live lake trout had such marks. Through comparison of marking on live and dead fish, Schneider *et al.* also demonstrated that the probability of death from an attack was lower for Seneca strain (Finger Lakes origin) than for other strains, whereas there did not seem to be much effect of size on survival over the range of sizes attacked (most marks were on lake trout greater than 600 mm total length (TL) and almost no lake trout less than 400 mm, TL, were marked). Given the greater survival of Seneca strain lake trout, the lower marking rate observed on this strain clearly resulted from lower attack rates. Schneider *et al.* speculated that the lower attack rate on Seneca strain fish may have stemmed from the fact that they tended to occupy deeper and colder waters than other strains.

Estimates of Sea Lamprey Mortality Based on Assumed Attack Lethality and/or Healing and Attachment Times

For spring wounding data Greig *et al.* (1992) suggested estimating sea lamprey mortality from:

$$M_L = \bar{W}[(1 - P_S)/P_S] \quad (10)$$

(dropping subscripts denoting host type). They provided summary laboratory values (based on Swink and Hanson (1986, 1989) and Swink (1990)) for P_S as starting values, but recommended that P_S be estimated in situ using the regression approach described above, treating the slope as equal to $(1 - P_S)/P_S$. This estimator of sea lamprey mortality rates, using the default values for P_S , has been widely used in the upper Great Lakes (Ebener *et al.* 1989, Eshenroder *et al.* 1995, Sitar *et al.* 1999). It is usually applied to individual size classes of lake trout and is assumed to estimate sea lamprey-induced mortality in the previous year. The values for P_S were based on the laboratory estimates because in practice it has been difficult to obtain enough contrast in wounding rates to estimate P_S in the

field, and because variation in fishing mortality rates has made application of the regression approach problematic. As applied, the method implicitly assumes that the mean spring wounds per fish accurately reflects the total number of potentially lethal attacks experienced during the previous year by surviving lake trout (see "Theory Underlying Interpretation of Wounding and Scarring Data"). Thus, it relies on strong assumptions regarding the timing of lethal attacks and healing times (Sitar *et al.* 1999). If P_S were estimated using the regression approach (or a more elaborate cohort-based adaptations), much weaker assumptions apply.

Spangler *et al.* (1980) applied the methods of Robson (1970) to a tagging study of lake whitefish in Lake Huron to estimate the attack rates of sea lamprey and the probability of surviving an attack. In contrast to the other approaches described in this section, Spangler *et al.* (1980) did not assume the lethality of an attack. However their approach does implicitly assume the expected time sea lamprey remain attached to hosts. Their results clearly indicate strong seasonality in both attack rates and the probability of survival. The incidence of fresh wounds increased during the season from June through September. Although fresh wounds were as common in September as in August, accumulation of scars was greatest in August and near zero in September, suggesting that few of the fish attacked (and observed as freshly wounded) in September survived.

Spangler *et al.* (1980) estimated that during the June to November period, 75% of the attacks on age-3 to age-5 lake whitefish were fatal, i.e., $P_S = 0.25$. This quantitative estimate is based on several critical assumptions. First is the fundamental assumption that fresh wounds are a class of wounds observed prior to when mortality can occur. In addition, total attack rates were calculated as I_t , rather than as I_t/h_t , and thus attachments are implicitly assumed to last for the duration of a census period (approximately one month). Attachment times for lake whitefish during the peak feeding period of August to November could be substantially less than 1 month, which would act to overestimate total attack rate and P_S .

Attachment Time and the Probability of Surviving an Attack

Attachment time and the probability of surviving an attack are linked conceptually. All else equal, the longer a sea lamprey remains attached to a host the greater the reduction in host blood quality (Farmer

et al. 1975), and thus the more likely the host will die. In this section first attachment time and the probability of surviving an attack in relation to host size, sea lamprey size, and water temperature are considered. Then the possibility that attachment times and attack lethality might reflect a dynamic response by sea lamprey to variations in host abundance is turned to.

The Role of Sea Lamprey Size, Host Size, and Temperature

The length of an attachment required to kill a host varies substantially among individual attacks, at least partially because of large variations in the feeding rate among sea lamprey attacks (Farmer *et al.* 1975, Farmer 1980, Cochran and Kitchell 1989, Madenjian *et al.* 2003). Farmer *et al.* (1975) demonstrated a significant negative linear relationship between the log of days to death and the log of percent host blood removal per day. On the basis of this relationship Farmer *et al.* (1975) suggested that ratios of host weight to parasite weight in excess of 40 would ensure survival, and Farmer (1980) noted a corresponding threshold feeding rate of 10% of host blood volume per day, below which hosts would likely survive.

Kitchell (1990) used the 10% threshold to examine the seasonal potential for mortality of hosts of different sizes, given a seasonal temperature regime assumed to reflect that occupied by lake trout in Lake Michigan and a range of potential patterns for seasonal growth by sea lamprey. His calculations suggested that given contemporary patterns of sea lamprey growth in Lake Michigan, lake trout hosts larger than 3 kg would not suffer mortality. He calculated that smaller hosts would pass through a period of potentially lethal attacks centered in the autumn, which would increase in duration with decreasing host size. In these calculations, sea lamprey size in the autumn during the contemporary period was approximately 250 g, suggesting that a host to parasite weight ratio of 12 would be sufficient to protect a host from lethal attacks. This more optimistic threshold is a consequence of the assumption that the weight-specific feeding rate of sea lamprey would decline rapidly with increasing sea lamprey size, as embodied in a sea lamprey bioenergetics model (Kitchell and Breck 1980).

Madenjian *et al.* (2003) built an individual based sea lamprey feeding model (IBM), where host mortality was based largely on the results of Farmer *et al.* (1975). In this model, sea lamprey feeding rate

varied among attacks with a mode close to 12% of body weight per day for sea lamprey less than 160 g (at 10°C). To avoid the growth of unrealistically large sea lamprey for the Great Lakes, mean feeding rate declined with increasing sea lamprey weight beyond this size. Madenjian *et al.* assumed that feeding rate, as a percentage of sea lamprey body weight, did not decline with increasing sea lamprey size until a size of 160 g had been reached because data collected by Farmer *et al.* (1975) did not suggest a decrease in feeding rate for sea lamprey up to this weight (Farmer *et al.* 1975, Farmer 1980, Cochran and Kitchell 1989, Madenjian *et al.* 2003). There are no direct measures on how sea lamprey feeding rate varies with body weight beyond 160 g. In Madenjian *et al.*'s model, attachment times varied among attacks, with an average length of approximately 10 days. An attack was lethal if it lasted long enough to reach the predicted time to death based on the rate of blood removal (Farmer *et al.* 1975). Although Madenjian *et al.* (2003) assume a higher feeding rate for most sizes of sea lamprey than Kitchell (1990), they impose a stricter requirement for lethality. In the IBM, 19% and 7% of attachments last long enough to lead to host death for 160-g and 250-g sea lamprey, respectively, feeding on a 3-kg host at 10°C. Over 90% and 60% of those attachments lead to a feeding rate of over 10% of host blood volume each day. Beyond 160 g, lethality of sea lamprey attack declines because of the modeled decline in feeding rate. This IBM lethality is less than has been observed in laboratory experiments (Swink 2003). Madenjian *et al.* (2003) note that their estimates of mortality only account for deaths that result directly from blood loss during an attachment and not for any latent effects that may lead to mortality after an attachment ends. During Swink's (2003) laboratory experiments some deaths attributed to sea lamprey occurred during a 2-week recovery period after the completion of an attack, but these made up only 35% of the total deaths attributed to sea lamprey, and thus do not fully explain the discrepancy.

In the laboratory, Farmer *et al.* (1977) explored the effects of sea lamprey size and water temperature on host mortality when groups of sea lamprey were feeding on groups of small (mean weight, 715 g) white suckers (*Catostomus commersoni*). Their results suggested that host mortality would increase with sea lamprey size and water temperature, at least over the range of temperatures typically occupied by salmonids in the Great Lakes.

Swink (2003) summarized information on host

mortality from a number of sea lamprey feeding experiments done at the U.S. Geological Survey Hammond Bay Biological Station during the 1980s and 1990s. He related the probability of death following a single sea lamprey attack to explanatory variables by applying stepwise logistic regression. He found that the strongest relationship was a positive association between mortality and temperature. Other explanatory variables included in his final model (in order of inclusion during the forward stepwise process) were sea lamprey weight change during the feeding trial (positive), initial sea lamprey weight (positive), host weight (negative), differences among host species (burbot (*Lota lota*) > lake trout > rainbow trout (*Onkoryhynchus mykiss*)), an interaction between temperature and sea lamprey weight change, an interaction between attachment time and sea lamprey weight change, and type of wound (A or B). The effects of sea lamprey weight gain, initial sea lamprey weight, host weight and temperature are illustrated in Figure. 2. Although the results of this synthesis were generally in agreement with results reported in individual studies, the dominant effect of temperature and the quite modest effect of wound type were somewhat unexpected, although the effect of temperature is consistent with bioenergetic considerations (Cochran *et al.* 1999). It seems likely that the importance of temperature became apparent because of substantial contrast in this variable across the studies. Swink (2003) noted that for some marks classified as type B the sea lamprey achieved substantial growth, whereas some marks classified as type A (at the termination of a trial) were initially type B and produced little weight gain, and this might explain why wound type did not have a large effect.

The potential importance of the relationships encapsulated by Swink's (2003) logistic regression model is demonstrated by calculating the probability of surviving an attack under conditions likely to be experienced in the field. In particular, for northern Lake Huron, information on the temperature occupied by lake trout (Bergstedt *et al.* 2003 a) and size of lamprey (Bergstedt and Swink 1995) is used to calculate seasonal patterns in the probability of survival given an attack (Fig. 3). The results suggest strong seasonal patterns in the probability of survival, given an attack driven by both temperature and sea lamprey size and somewhat higher probabilities of survival for Finger Lake origin strains of lake trout during the summer when they tended to occupy lower water temperatures than

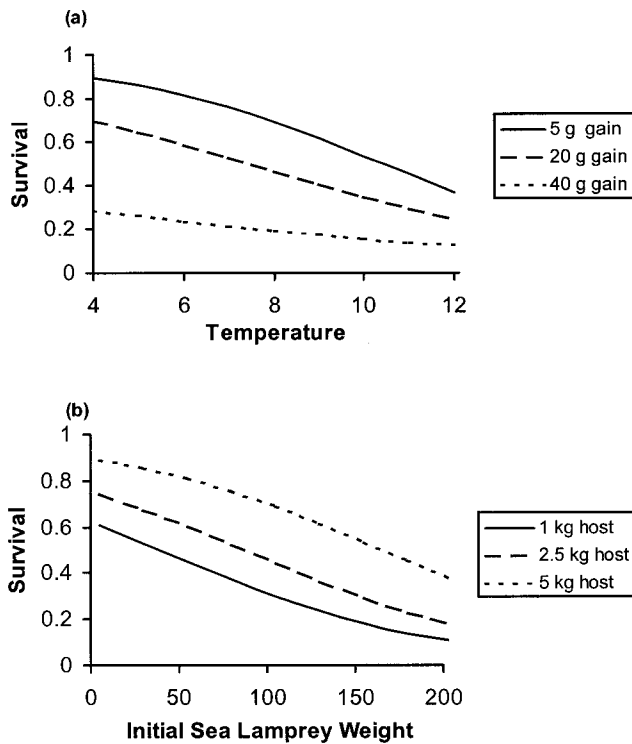


FIG. 2. Predicted survival from an attack by a sea lamprey for lake trout experiencing a type A wound, based on the logistic regression model fit to laboratory data (see Swink (2003)). (a) The relationship versus temperature for three amounts of weight gain. In this case initial sea lamprey weight was assumed to be 120 g and lake trout weight was assumed to be 2 kg. (b) The relationship between survival and sea lamprey weight at the beginning of an experiment, for three sizes of lake trout hosts. In this case a sea lamprey was assumed to gain 20 g in weight and feeding was assumed to occur at 10°C.

Great Lakes origin strains (Bergstedt *et al.* 2003a). They also illustrate a strong effect of host size. Although the directional effects of host and sea lamprey weight and water temperature seen by Swink (2003) are consistent with those proposed by Farmer *et al.* (1975, 1977), the probability of surviving an attack reported by Swink was substantially lower than what can be inferred from Farmer *et al.*'s results, especially for larger sea lamprey and larger host sizes.

Other estimates of the probability of surviving a sea lamprey attack are based on field marking data. As described above, these estimates are based on

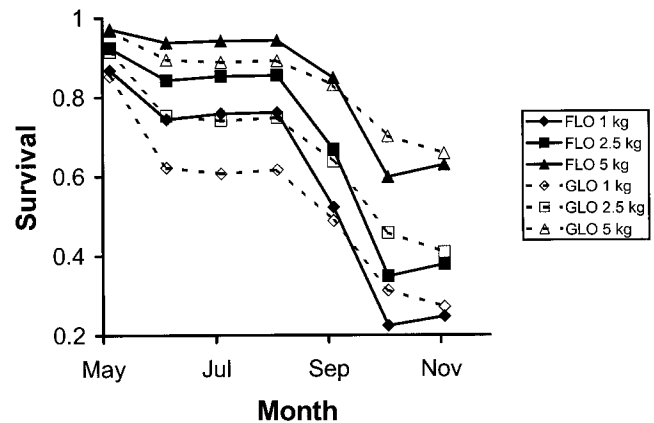


FIG. 3. Predicted seasonal variation in the probability of a lake trout surviving a sea lamprey attack. Predictions from logistic regression model of Swink (2003) using field data on seasonality in water temperature occupied by lake trout (Bergstedt *et al.* 2003a) and sea lamprey weight (Bergstedt and Swink 1995). In the legend numbers refer to the weight of lake trout in kg, "FLO" refers to lake trout strains of Finger Lakes origin (which includes Seneca and Ontario strains, see Bergstedt *et al.* 2003a), and GLO refers to strains of lake trout of Great Lakes origin. All attacks assumed to produce type A wounds. Weight gain was assumed to be 5 g at 5°C or less and to increase 3 g for each degree in increase in water temperature occupied, in rough accord with Swink's (2003) data.

strong assumptions regarding healing and attachment times. In addition, the possibility that attacks that leave type B wounds could frequently be lethal might compromise some estimates. Of particular interest is the estimated probability of surviving an attack currently used in the IMSL parasitic-phase submodel. This probability increases as the ratio of sea lamprey to host weight decreases, approaching a maximum value when the ratio is small, in general agreement with laboratory observations. However, the maximum probability of survival estimated for Lake Ontario (0.25, Koonce and Locci-Hernandez 1989) is less than that estimated by Swink (2003). This lower value was obtained by adjusting the probability of survival so that the IMSL parasitic submodel would match data on both marking and estimated numbers of dead lake trout from the "carcass study." It is worth emphasizing that the many uncertainties involved cannot rule out values more in line with Swink's (2003) summary

of laboratory observations. Some of these uncertainties are associated with the estimates of attachment times and healing times required by the submodel and the assumption that these and the effective search rate would be constant over the feeding season. Additional uncertainty stems from the need to expand the carcass survey estimates by a factor of approximately four to account for the entire lake (rather than just New York waters), and for the fact that the survey did not capture all lake trout that died during the feeding season (due to decay and other losses of carcasses). The IMSL calculations make use of the number of metamorphosing parasites, and uncertainty about this number also contributes to uncertainty regarding the probability of surviving an attack (Larson *et al.* 2003).

Dynamic Responses to Host Density

To this point the probability of surviving an attack was treated as a constant, given sea lamprey size, host size and temperature. Kitchell and Breck (1980), however, hypothesized that sea lamprey might decrease the time they remain attached to hosts in response to increases in host density. They suggested that this response might cause sea lamprey to switch from being a predator (with lethal attacks) to becoming a parasite (with nonlethal attacks) when prey are abundant. This idea stems from optimal foraging theory developed for predators feeding in patches. According to the basic models, a forager stays in a patch until the quality of the patch decreases to the point where it is more profitable to leave and search for a new patch. This quality threshold increases (and thus time in a patch decreases) as patch density increases and the expected time it takes to discover a new patch decreases. Theoretical predictions were examined empirically by Cook and Cockrell (1978) and Sih (1980) with sucking backswimming bugs (*Notonectidae*), treating their individual prey items as patches. They discovered that the energetic return rate from a prey did decrease as handling time increased, and that handling times decreased in response to increased prey abundance.

The idea of sea lamprey attachment time varying in response to host density was further explored by Cochran and Kitchell (1986, 1989), Cochran (1994), and Cochran *et al.* (1999). Cochran and Kitchell (1989) developed and parameterized a model describing how sea lamprey energetic return and host blood quality changed over time during a single attachment, and Cochran *et al.* (1999) extended the

model to incorporate the influence of temperature. Cochran *et al.* (2003) modified model predictions to account for information showing that sea lamprey energy density is higher for larger sea lamprey. This model combined with Kitchell and Breck's (1980) bioenergetics model for sea lamprey was used to successfully predict the growth of sea lamprey based on their attachment times.

Cochran and Kitchell (1989) addressed the potential for responsive attachment times experimentally by comparing attachment times of sea lamprey presented with one or two hosts. They were not able to detect significant differences in attachment times in response to the number of hosts available, although they noted that sea lamprey attachment times were highly variable and this could have obscured a real response to host density. In their experiment, each sea lamprey was presented with both prey density treatments, with half the lamprey presented two hosts first and half presented one host first. They found that the first attachment (in the first experimental trial) was significantly longer than the second attachment by the same sea lamprey (during the second experimental trial). Cochran and Kitchell (1989) discussed two potential explanations for this result. One possibility is that the sea lamprey responded to a recent feeding event as a proximate cue that hosts are abundant. An alternative explanation is that the sea lamprey grew and developed during their first attachment, and thus the larger sea lamprey more rapidly depleted their host's blood quality (and in some cases killed them) during the second attack.

Cochran and Kitchell (1986) showed that although the optimal attachment time decreases as the expected search time to find a host decreases, the increase in expected energetic return for the "optimal" attachment time would often not be large in comparison with a strategy of remaining attached until host blood quality had deteriorated to the point that the host died. They noted that if sea lamprey have a risk averse foraging strategy, variations in the sea lamprey's assessment of host density could cause many lamprey to remain attached until the host dies, even when the deterministic optimal foraging prediction is to detach while the host is alive. Under these conditions, substantial variation in the responses of individual sea lamprey is expected.

There is some evidence within the Great Lakes suggesting that the probability of surviving an attack can vary for reasons other than temperature or the relative sea lamprey and host sizes. The proportion of observed sea lamprey marks of different

TABLE 2. Proportion of sea lamprey marks classified into different categories of the King (1980) classification system, based on observations of lake trout sampled by Chippewa Ottawa Resource Agency biologists from commercial catches in Lakes Huron, Michigan and Superior during 1985 to 1999.

	Mark Category							
	A1	A2	A3	A4	B1	B2	B3	B4
Michigan	0.11	0.12	0.19	0.21	0.11	0.10	0.10	0.07
Superior	0.13	0.17	0.11	0.14	0.16	0.18	0.07	0.02
Huron	0.37	0.19	0.17	0.06	0.11	0.05	0.04	0.01

types and stages in commercial fishery samples appears to be quite different in northern Lake Huron than in either northern Lake Michigan or Lake Superior (Table 2), with a much higher proportion of A1 marks in northern Lake Huron. These data were collected by the same group of observers, thus issues having to do with mark interpretation (Ebener *et al.* 2003) are minimized. The qualitative pattern shown in Table 2 is retained for various seasonal and lake trout size subsets of the data. The relative frequency of type A1 marks on lake trout seen in Lake Ontario (Schneider *et al.* 1996) is similar to that seen in lakes Michigan and Superior and not like that seen in northern Lake Huron. One hypothesis that can explain the pattern is that lake trout with type A1 marks in northern Lake Huron have a lower probability of survival to a later time when the A1 mark would have healed. If so, this is consistent with the optimal-attachment-time hypothesis advanced by Kitchell and Breck (1980), as preferred hosts such as large lake trout are scarce in that part of the lake (Eshenroder *et al.* 1995, Sitar *et al.* 1997), and thus longer and more lethal attachments are predicted. Another concern is that the pattern seems to suggest substantial sea lamprey mortality after a wound is observed in the A1 stage on a live lake trout. If true, this conflicts with the assumptions underlying the models used to predict observed marking rates and convert marking rates into estimates of mortality. Anecdotal observations by M.P. Ebener also suggest there may be delayed mortality resulting from sea lamprey attacks. He has observed sampled lake trout in the spring with sea lamprey marks most likely the result of attacks the previous autumn (healed as far as the A3 stage). The blood and flesh of some of these lake trout were pale, suggesting few red blood cells and that these fish would likely die during the next growing season.

Swink's (2003) laboratory studies provide information about the duration of sea lamprey attachments on hosts. These experiments represent the

results of feeding trials where individual sea lamprey were presented with one host. These experiments were done using field-collected sea lamprey and in water drawn from Lake Huron. As a result, sea lamprey size and experimental temperature varied seasonally in a fashion related to the temperature regime in Lake Huron. Results are presented for analyses based on all the experimental trials leading to type A wounds, which consisted of attacks on lake trout, burbot and rainbow trout. Alternative analyses restricted only to non-lethal attacks and analysis only on trials with lake trout (the most commonly used host) produced qualitatively similar results. An initial regression model related attachment time (h) to host weight (WH), sea lamprey weight (WL), experimental temperature (TM), delay time to attack ($delay$) and the instantaneous growth of sea lamprey during its previous attack (G):

$$\ln(h) = a + b \ln\left(\frac{WH}{WL}\right) + c \ln(TM) + d \ln(delay) + e \ln(G) + error \quad (11)$$

The intercept as well as the slope parameters were allowed to vary among host species. Because preliminary analyses indicated no significant effect of the instantaneous growth rate and no difference in slope parameters among host species for the effects of $delay$ and temperature, those terms were dropped from the final model.

The assumed relationship between attachment time and the ratio of host to sea lamprey weight is consistent with predictions regarding the length of time required to reduce host blood quality to a fixed level (Cochran and Kitchell 1989). In addition, including sea lamprey and host weight as individual variables rather than a ratio led to little increase in the amount of variation explained. The assumed multiplicative effect of temperature is consistent with the extended model (Cochran *et al.* 1999). Cochran *et al.* (1999) proposed a domed response to

temperature, but given that the peak of the effect was proposed to occur at 18°C, the assumed monotonic function in the regression model seemed adequate. The *delay* term was included because it might reflect potential motivation or tendency to feed. Growth during the previous feeding trial was considered for inclusion because it could be an indicator of recent food intake.

The fit of the regression model indicated that attachment time increased in response to increases in the host to sea lamprey size ratio and to increases in the delay time, but decreased in response to temperature (Fig. 4, Table 3). In the final model, there was no significant effect of species on the regression intercepts, however, the estimate was highest for lake trout and lowest for burbot. There was a significant effect of species on the estimated slope parameter associated with the size ratio, and the estimated slope was highest for burbot and lowest for lake trout.

It is not clear how to interpret the effect of delay time, and very large differences in delay times were seen for sea lamprey under what seemed like quite similar conditions. Cochran and Kitchell (1989) also noted a positive relationship between attachment time and delay time and discussed potential implications of the lack of a significant difference in delay time between their two treatments. In principle, delay time could reflect either an external (time taken to detect and pursue prey) or an internal (motivation to feed) factor. However in the laboratory studies of Cochran and Kitchell (1989) and Swink (2003), hosts were always available and detection probably did not play a role. If attachments following long delay times represent attacks by sea lamprey with “low motivation” to feed, the associated long attachments may also be associated with relatively low rates of blood removal.

SELECTION OF HOSTS

Host selection by sea lamprey is defined here as the relative rate at which individual hosts of different types are successfully attacked. In the terms of equation 1, selection for prey type *i* is:

$$\alpha_i = \frac{\lambda_i}{\sum_j \lambda_j}$$

This corresponds to the definition of preference given by Manly *et al.* (1972) and developed further by Chesson (1978, 1983). This definition of selection combines together a number of components involved in the predation process.

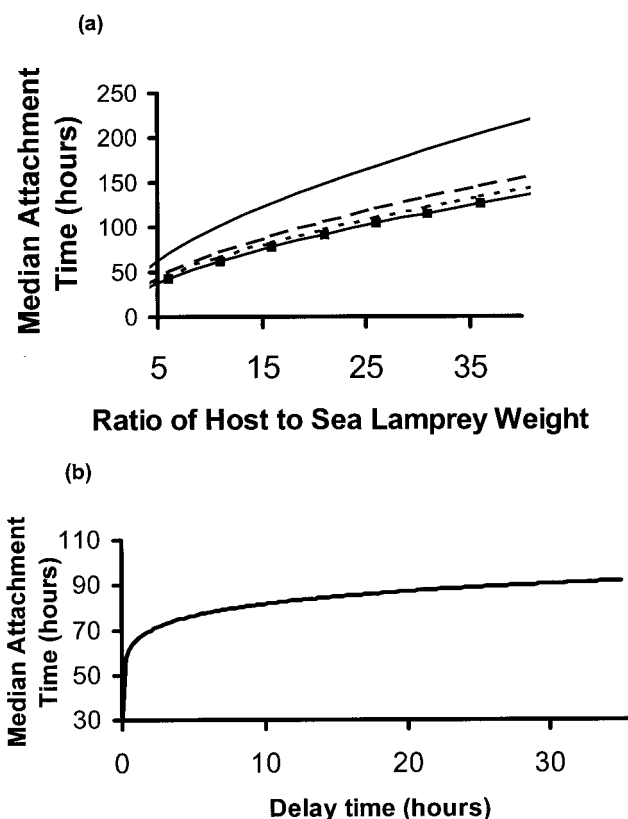


FIG. 4. The relationship between attachment time of a sea lamprey to a host and the ratio of host to sea lamprey weight and temperature (a) and delay time (b). Results are based on lake trout as the host. Panel a assumes a delay time of one day and the four curves are for 1°C (solid line), 5°C (long dashed line), 7.5°C (short dashed line), and 10°C (solid line with squares). Panel b assumes a host to parasite weight ratio of 20. The relationships are based upon a linear regression (Table 3) using the multi-experiment laboratory database described by Swink (2003).

Observations that provide information on host selection come from laboratory experiments and from observations of marking rates in the field. Both have their limitations. In the laboratory, the observations are directly on attack rates and there is no potential confusion caused by differential survival of hosts. However, some components of the selection process (e.g., prey detection) will be substantially altered by the laboratory setting. Inferences from marking rates and observed attachments in samples depend upon assumptions such as the relative survival, attachment times, and healing times for different hosts (equations 5 and 7).

TABLE 3. Results of regression analysis relating attachment time of sea lamprey to host/parasite weight ratio, temperature, and delay time using the same laboratory database analyzed by Swink (2003). Intercept and the slope parameter associated with weight ratio varied among species, with rainbow trout the baseline species and estimated changes from this baseline estimated for lake trout and burbot.

ANOVA Table					
Source	DF	Type III SS	MS	F	Pr > F
ln(weight ratio)	1	108.96	108.96	126.24	< 0.0001
ln(temperature)	1	9.65	9.65	11.18	0.0009
species	2	3.46	1.73	2.0	0.14
ln(weight ratio) x species (=slope)	2	8.04	4.02	4.65	0.0099
ln(delay)	1	9.91	9.91	11.49	0.0007
error	578	498.87	0.863		

Parameter Estimates			
		Estimate	Standard error
Intercept		2.56	0.384
Change in intercept for:			
	burbot	-0.738	0.651
	lake trout	0.322	0.386
ln(temperature) slope		-0.21	0.063
ln(weight ratio) slope		0.867	0.12
Change in ln(weight ratio) slope for:			
	burbot	0.147	0.214
	lake trout	-0.272	0.125
ln(delay) slope		0.096	0.028

Lab Experiments on Host Selection

Laboratory information on host selection is reviewed by Swink (2003). Only the experiment by Farmer and Beamish (1973) appears to provide quantitative information on host species selection. Farmer and Beamish's results differed from what appears to happen in the lakes (Farmer and Beamish 1973, Farmer 1980, Swink 2003). For example, common carp (*Cyprinus carpio*) were preferred over lake whitefish and burbot, whereas in the field the latter two species have experienced higher marking rates and apparently larger effects due to sea lamprey. Other observations were in accord with results from the field. In particular, walleyes (*Stizostedion vitreum*) were least preferred, and in the field, marking of percids is generally quite low except under extreme conditions of host depletion (Lennon 1954). Qualitative observations from earlier laboratory studies (Lennon 1954) also suggest that percids tend to not be selected as hosts.

Several laboratory experiments and observations report on size selection by sea lamprey (Farmer and Beamish 1973, Swink 1991, Cochran and Jenkins

1994). The general result of these studies is that larger hosts are attacked more frequently than smaller ones (Swink 2003), and this agrees with observations from earlier studies (Lennon 1954). The extent of this size selection appears to vary, depending upon context (Swink 2003). Cochran (1985) re-analyzed the data from Farmer and Beamish (1973) and reported new results for chestnut lamprey (*Ichthyomyzon castaneus*). In general the results suggested that selection for hosts increased faster with host size than the surface area of the hosts. This was statistically significant for sea lamprey feeding on lake trout (but not on other host species) and for chestnut lamprey in general. Using larger parasites feeding on a wider range of lake trout sizes, Swink (1991) found significantly greater selection than expected for attacks in proportion to surface area only when the largest lake trout present was greater than 615 mm, TL. Both Cochran (1985) and Swink (1991) interpreted departure from attacks proportional to surface area as strong evidence for active size selection. Swink (1991) argued that such active size selection probably reflected avoidance of small hosts when large hosts were pre-

sent. Cochran and Jenkins (1994) presented evidence for small sea lamprey confined with pairs of small rainbow trout (based on experiments reported by Cochran 1986a and 1986b and Cochran and Kitchell 1989) indicating that they attacked the heavier host significantly more often, even for modest differences in host size.

Observed Patterns of Marking and Attachment among Host Types

There are many reports of marking rates increasing with host size (Royce 1949, Fry 1953, Hall and Elliott 1954, Wigley 1959, Berst and Wainio 1967, Berst and Spangler 1970, Collins 1971). This is not just an aging phenomena, as the pattern is evident for wounds and increments in scars (Royce 1949, Budd *et al.* 1969, Pycha 1980, Schneider *et al.* 1996, Sitar *et al.* 1997, Rutter and Bence 2003). There is strong circumstantial evidence that the observed patterns are at least partly due to sea lamprey generally selecting for larger hosts. The pattern is equally evident for observations collected in the spring (Rutter and Bence 2003) and those collected in the late-summer (Schneider *et al.* 1996), indicating that artifactual patterns related to ongoing attacks and handling time (equation 7) are not principally responsible. Schneider *et al.* (1996) showed that although marking rates were higher on larger fish, the size distribution of lake trout carcasses with wounds was similar to the size distribution of living lake trout with wounds. Thus differential survival did not explain the result.

Comparison of how the relative number of wounds per fish varies with size in different situations also suggest selection for larger hosts, and that this may represent active selection that can vary depending upon conditions. The expectation is that the relative number of wounds on different types of hosts should not change unless there is either a change in selection of different host types, or in the relative survival of different host types. This expectation is derived from equations 1, 2, and 12, which predict that for two host types i and j , the relative wounding rate (\bar{W}_i/\bar{W}_j) will remain constant under these conditions. Rutter and Bence (2003) noted that the general relationship between wounds per fish and length is sigmoid (Fig. 5). However, during 1984 to 2000 in the main basin of Lake Huron, the lake trout size at which the number of wounds increased rapidly shifted toward smaller hosts further north in the lake, where larger lake trout were scarcer (Fig. 5). Rutter and Bence speculated that

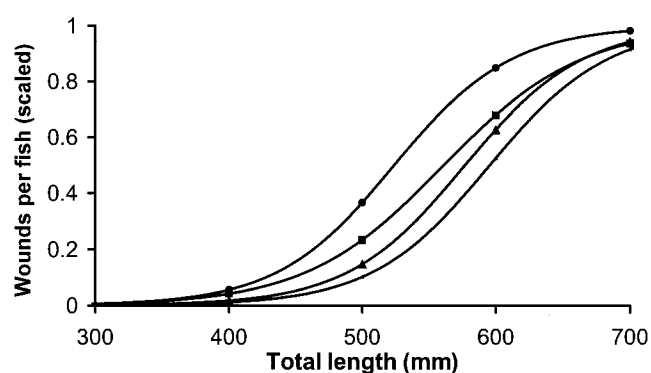


FIG. 5. *Estimated relationships between wounds per lake trout and lake trout length. Shown are three contemporary relationships from three regions of Lake Huron (northern Lake Huron (statistical district MH-1) line marked by circles, central Lake Huron (statistical district MH-2) line marked by triangles, southern Lake Huron (statistical districts MH-3, MH-4, MH-5) line with no symbols), and one relationship from Lake Michigan in 1947 during the period of lake trout collapse (line marked with squares). Contemporary Lake Huron curves based on analyses by Rutter and Bence (2003) are for spring surveys and combined A1 to A3 marks. Curve for Lake Michigan in 1947 is fit of logistic model to proportions wounded, based on data summarized by Randy Eshenroder (Great Lakes Fishery Commission) from original U.S. Fish and Wildlife data records of "fresh marks" in the autumn collected under the direction of J. Van Oosten.*

this might indicate an adaptive behavioral response by the sea lamprey. In any case, it seems very unlikely that the relatively higher wounds per fish seen on small lake trout in northern Lake Huron are the result of a higher probability of surviving an attack; if anything, sea lamprey attacks may be more lethal in that area (see "Attachment Time and the Probability of Surviving an Attack"). Other observations suggest similar dynamic changes in active size selection by sea lamprey. During the period of lake trout recovery in Lake Superior, as larger lake trout became more abundant, there was an increase in the host size at which the number of wounds became substantial (Pycha and King 1975). Simultaneously, survival of small lake trout in Lake Superior appeared to increase (Pycha and King 1975, Lawrie and MacCallum 1980). A similar phenomenon may have also occurred during the 1980s in Lake Ontario (Schneider *et al.* 1996).

A number of authors have reported on the relative number of marks or attachments on different host species (Shetter 1949, Applegate 1950, Lennon 1954, Christie and Kolenosky 1980, Johnson and Anderson 1980, Smith and Tibbles 1980, Ebener *et al.* 1995, Morse *et al.* 2003). These studies indicate that some species, particularly lake trout, are commonly marked at higher rates, whereas other species are rarely marked or at lower rates (percids, common carp, and in general species with heavy scales). While differences in part reflect size related differences among species, the patterns clearly indicate that selectivity for host species varies for other reasons.

Marking and attachment patterns suggest a seasonal pattern to selectivity related to ontogenetic changes in habitat use by sea lamprey (Applegate 1950, Christie and Kolenosky 1980, Johnson and Anderson 1980, Young *et al.* 1996). Although widely distributed, it appears that early in the parasitic phase (through the first spring in the lake) most individuals occupy deep water and tend to attack bottom-associated hosts such as bloater (*Coregonus hoyi*). This tendency of smaller sea lamprey to attack bottom-associated hosts appears to characterize sea lamprey in the marine environment also (Halliday 1991). Johnson and Anderson (1980) reported a general tendency for sea lamprey to move into shallower waters after their first spring as parasites. In addition, they noted a sharp drop in the proportion of parasites recovered from deep water fisheries when parasites attained sizes typical for late autumn. These observations match closely those of Applegate (1950), who noted that starting in September in Lake Huron sea lamprey appeared to concentrate in bays and shoreline indentations. Superficially, these observations appear to conflict with those of Christie and Kolenosky (1980) from Lake Ontario. They reported highest wounding among nearshore species in the spring and autumn, and argued for offshore movements during the summer on the basis of an increase in wounding of lake whitefish at that time, coinciding with a decrease in observed wounding on nearshore species. The apparent discrepancy seems to reflect a difference in the range of host species available. The "offshore" distributed lake whitefish of Christie and Kolenosky would have been classified as a shallower water species by Applegate and Johnson and Anderson. The observation by Christie and Kolenosky of high wounding on nearshore species in the spring probably reflects attacks by larger sea lamprey who have already spent a year or more as

parasites, rather than an indication that young parasites are concentrated in shallow water.

In spite of striking seasonal patterns, evidence for strong selection for lake trout emerges. Christie and Kolenosky (1980) found higher wounds per fish on lake trout than other species for each month with adequate samples (more than five fish) with the exception of May, and overall wounds per lake trout exceeded the highest numbers seen on the other species, northern pike (*Esox lucius*) and lake whitefish, by 10 and five fold respectively. Johnson and Anderson (1980) reported that the majority of all sea lamprey recovered from commercial fisheries in Lakes Michigan, Huron, and Superior were attached to lake trout, and the only exception was that for small sea lamprey (less than 200 mm) the majority were found attached to deep-water ciscos (*Coregonus* spp.). Morse *et al.* (2003) report higher wounding of lake trout than corresponding sizes of chinook salmon (*Onchorynchus tshawytscha*) or lake whitefish in every year from 1982 through 1999.

Observed patterns in marking among species also suggest that sea lamprey may have changes in selectivity, potentially reflecting dynamic behavioral responses to prey abundances. In situations where

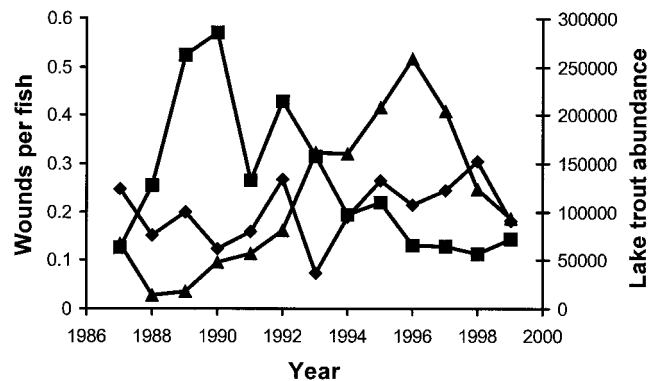


FIG. 6. Wounds (sum of A1 to A3 marks) observed on lake trout (denoted by diamonds for 50 to 60 cm size class) and chinook salmon (denoted by squares for 90 to 100 cm size class) in Chippewa/Ottawa Resource Authority commercial fishery samples from northern Lake Huron during 1987 through 1999, and estimated age-5 and older lake trout abundance (denoted by triangles, unpublished stock assessment, 1836 treaty waters Technical Fisheries Committee Modeling Subcommittee). Lake trout and chinook salmon sizes used to calculate wounding rates were largest size classes for which sample sizes were deemed sufficient.

lake trout are abundant, sea lamprey wounding on lake whitefish is quite low compared to wounding on lake trout, whereas when lake trout are scarce the relative wounding rate on lake whitefish increases (M.P. Ebener, unpublished data), and substantial wounding and sea lamprey induced mortality on lake whitefish are observed (Christie and Kolenosky 1980, Spangler and Collins 1980, Morse *et al.* 2003). Temporal patterns in wounding on chinook salmon and lake trout seen in northern Lake Huron appear to be consistent with the idea of dynamic changes in selection. The wounding rate on large chinook salmon tended to be relatively higher (in comparison with the wounding rate on lake trout) when lake trout were at low abundance (Fig. 6). These apparent changes in selection may in part reflect changes in the habitat overlap resulting from complex dynamics in the spatial distributions of both sea lamprey and hosts.

NUMERICAL RESPONSES

To this point the feeding and growth of parasitic sea lamprey has been concentrated on, without accounting for mortality of sea lamprey during the parasitic phase. There is little information on this topic. The IMSL parasitic submodel assumes constant survival (0.5, Koonce and Locci-Hernandez 1989) from metamorphosis to the summer-autumn period when most losses of hosts occur, with negligible sea lamprey mortality from that point until spawning. This assumed pattern and magnitude of sea lamprey mortality has not been manipulated because both larval and spawning-phase sea lamprey estimates of abundance were considered indices, and thus parameters setting their scales were not distinguishable from the assumed level of survival.

There is, however, some evidence that mortality during the early parasitic stage can be substantial and might vary with conditions. Swink (1995) and Eshenroder *et al.* (1995) speculated that the increase in sea lamprey abundance in Lake Huron seen in the early 1980s was tied to increased recruitment of bloater, which may have been a critical host in the deepwater occupied by this life-stage. Swink (1995) found that young parasites held at about 4°C (typical of bottom water in Lake Huron) grew more and survived better than young parasites held at colder temperatures, and suggested that high overwinter survival might depend upon abundant hosts in this warmer bottom habitat. Young *et al.* (1996) provided additional support for the linkage between sea lamprey and changes in the fish com-

munity in the lake by demonstrating a significant correlation between sea lamprey abundance and both bloater abundance and salmonid stocking in northern Lake Huron. Catch-per-effort in larval sea lamprey surveys was highly variable, and did not suggest sustained increases in larval abundance in the St. Marys River, the likely source of parasitic sea lamprey in northern Lake Huron. This suggests that increased survival during the early parasitic phase may have been responsible for the observed increase in parasite abundance. Additional evidence comes from recovery rates of recently metamorphosed sea lamprey that were marked during the 1960s and 1970s (Hanson and Swink 1989, U.S. Geological Survey Lake Huron Biological Station unpublished data), and more recently (Bergstedt *et al.* 2003b). The recovery rate increased from virtually zero for the 1966 and 1967 parasitic cohort to 4–8% in recent years (Stewart *et al.* 2003). Stewart *et al.* (2003) concluded that these results strongly suggest changes in survival.

Variations in survival of sea lamprey from the time when substantial host mortality occurs until they migrate upstream to spawn, is also possible. Christie and Kolenosky (1980) speculated that sea lamprey might respond to increases in the abundance of large hosts with increased survival and increased growth. However, both observed temporal relationships between spawning runs and the numbers of parasites observed in fisheries (Moore and Lynchwick 1980, Young *et al.* 1996, Sullivan *et al.* 2003) and the fact that large spawning runs have occurred when large hosts are scarce (Morse *et al.* 2003), suggest that such variation may not be a dominant influence on abundance of parasites and resulting spawners. Furthermore, the comparable numbers of estimated spawners (Mullett *et al.* 2003) and parasites (Bergstedt *et al.* 2003b) in northern Lake Huron suggests that under current conditions, survival through the parasitic phase is reasonably high, even in the area where larger hosts are scarcest. In addition sea lamprey growth in this area during the parasitic phase is not substantially lower than in other areas, and is not as low as it has been in the past (Houston and Kelso 1991), so reductions in sea lamprey survival late in the parasitic phase might become apparent under conditions where food is more limiting.

A COMPARISON OF HOST DAMAGE ESTIMATES

Much of the effort directed at the study of the sea lamprey's parasitic phase is ultimately directed to-

ward estimating or predicting damages to hosts, especially lake trout. A number of such estimates exist or can be calculated, and we believe it is worth comparing some of them with respect to the amount of host losses that are estimated or predicted and their underlying assumptions. Swink (2003) calculated and reviewed estimates of host losses based on laboratory studies. A widely cited value is due to Parker and Lennon (1956), who suggested that a sea lamprey in the wild killed up to 16.8 kg of fish before reaching spawning size. They reported an estimate that was double the actual amount killed over the season by the average sea lamprey in the lab. This was likely because the final weight reached by sea lamprey in the laboratory was less than half that of spawning sea lamprey in the wild. Based on Farmer *et al.*'s (1977) laboratory experiments, Swink (2003) calculated that a single sea lamprey would kill on average 11.9 kg of hosts from May through September as it grew from 10 g to 90 g. Swink (2003) argues, however, that both of these earlier studies were situations where relatively small hosts suffered multiple attacks and does not closely resemble current conditions in the Great Lakes. He reports new estimates based on laboratory studies where individual sea lamprey are followed over a feeding season, each host was attacked only once and hosts were substantially larger than those from the earlier studies. There was substantial variability in the weight of hosts killed per sea lamprey, ranging from 1.2 kg to 36 kg among individual sea lamprey and from 6.6 kg to 18.9 kg (equating to 3.3 to 10 hosts) among experiments. Some of this variability seems to be explainable by variation in temperatures and host sizes among experiments. In general, fewer hosts with a lower total weight tended to be killed at lower temperatures. Although fewer hosts tended to be killed per lamprey when the average weight of hosts was higher, Swink (2003) observed that the total weight of hosts killed increased with host size.

Although the new laboratory estimates reported by Swink (2003) are based on conditions closer to those currently experienced in the Great Lakes, the peak seasonal temperatures tended to be greater than those that lake trout appear to occupy in northern Lake Huron (Bergstedt *et al.* 2003a), the near constant exposure to hosts may not reflect field conditions, and the amount of growth in the laboratory tended to less than observed in the field (Bergstedt and Swink 1995). For these reasons, some rough approximations of the total number of hosts that could be killed by sea lamprey were calculated.

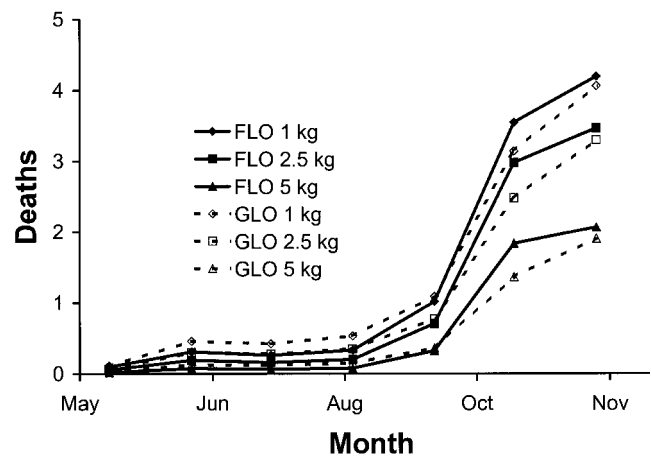


FIG. 7. Calculated numbers of lake trout killed monthly in northern Lake Huron per sea lamprey over the May to November period. For references purposes, in each case all attacks are assumed to be on a single specified type of lake trout. Estimates based on lab derived probability of survival (Fig. 3) taking into account field observations on parasite size and temperature occupied by lake trout, and assumed number of attacks. The assumed number of attacks per month are calculated as the observed field growth from Bergstedt and Swink (1995) divided by a rough estimate of growth per attack based on laboratory observations (Swink 2003). Inspection of the lab data suggested that below 5°C growth averaged about 5 g per attack and above 5°C growth increased approximately 3 g per degree C. Hosts varied by weight (1, 2.5 and 5 kg), and whether they occupied temperatures typical for strains of lake trout of Finger Lakes origin (FLO) or those typical of Great Lakes Origin (GLO) (Bergstedt et al. 2003a).

These calculations used field observations on sea lamprey growth and the temperature occupied by lake trout in northern Lake Huron and Swink's (2003) logistic model for the probability of survival given an attack, and relied on an assumption on the number of attacks required to achieve the observed growth (Fig. 7). The resulting seasonal pattern of numbers of hosts killed per sea lamprey indicates that most deaths would occur in October and November, which is consistent with field observations from Lake Ontario (Bergstedt and Schneider 1988). This pattern is most extreme for larger lake trout and those occupying temperatures typical of strains of Finger Lakes origin (Fig. 7). The cumulative

TABLE 4. Calculated losses of lake trout hosts per feeding sea lamprey when feeding on different host types from May 1 through the end of November given observed temperatures occupied by hosts and growth in northern Lake Huron. For additional details on calculations see Figure 7 legend.

Host strain	Host losses		
	Host size	Number	Weight (kg)
Finger Lakes Origin	1 kg	9.8	9.8
Finger Lakes Origin	2.5 kg	7.8	19.4
Finger Lakes Origin	5 kg	4.5	22.3
Great Lakes Origin	1 kg	9.8	9.8
Great Lakes Origin	2.5 kg	7.5	18.8
Great Lakes Origin	5 kg	4.1	20.3

numbers killed ranged from 4.1 to 9.8 depending upon strain type and host size (Table 4), and these cumulative numbers killed and associated weights (Table 4) were roughly in accord with the direct observations of cumulative host losses per parasite in the laboratory. These high estimates of damages due to sea lamprey contrast with those estimated by Madenjian *et al.* (2003), who estimated less than one host death per sea lamprey, while also taking into account the temperature regime and growth of sea lamprey in northern Lake Huron. Much of this difference stems from the higher probability of surviving an attack combined with fewer and longer attacks implicit in the IBM assumptions (see "Attachment Time and the Probability of Surviving an Attack").

Wounding data have been used to estimate the mortality rate due to sea lamprey that is experienced by lake trout, assuming a probability of survival based on laboratory studies (equation 10). Using wounding data together with estimates of lake trout abundance-at-age from stock assessments, this approach was used to estimate total number of lake trout killed by sea lamprey for eastern Lake Superior, Lake Michigan, and Lake Huron. These were divided by estimates of the number of spawning sea lamprey to calculate the number of lake trout killed per returning spawner (Appendix 2). These calculations resulted in values of 1.32 and 0.75 lake trout deaths per spawner in Lakes Huron and Michigan, respectively. In Lake Superior the number of dead lean lake trout (lean morphotype or form) per spawner was similar to the value for Lake Huron (1.36 per spawner). One possible biological explanation for the lower number of lake trout deaths per spawner for Lake Michigan is

that more attacks are drawn away from lake trout by a more diverse salmonine community. Another possibility stems from the use of autumn wounding data on Lake Michigan and the assumption, based on recent data, that autumn and spring wounds are of comparable magnitudes (Appendix 2). When both have been measured observed ratios have been variable. For example, Moore and Lychwick (1980) adjusted autumn wounding rates in Green Bay, Lake Michigan by assuming that autumn wounding rates were about half those seen in the spring. If this were true, then an adjustment to spring rates would increase the number of lake trout killed per spawner in Lake Michigan to a level exceeding that in Lake Huron. While calculated numbers of lean lake trout killed per sea lamprey in Lake Superior seem consistent with those from the other lakes, when siscowet lake trout (morphotype or form) are included with the lean lake trout as hosts, the result was 89.92 lake trout deaths per spawner, which seems inconsistent with laboratory feeding and survival studies.

Although the estimate of total lake trout deaths in eastern Lake Superior depends on uncertain estimates of lean lake trout abundance, the ratio of siscowet to lean lake trout abundance, and spawner abundance (Appendix 2), it seems unlikely that these uncertainties can reconcile the estimated deaths per spawner with reasonable attack rates. This suggests that either the characteristics of attacks and resulting wounds on siscowet lake trout in Lake Superior (attachment times, healing times, probability of survival) differ from those observed on lean lake trout, or the number of parasitic sea lamprey in Lake Superior far exceeds the number we would expect given the estimated number of spawners, or both.

Estimates of lake trout deaths per spawner from Lake Ontario carcass study (Schneider *et al.* 1996) and abundance of spawning phase sea lamprey from a spawner-discharge regression (Mullett *et al.* 2003) also allows calculation of the number of lake trout deaths per spawner (Appendix 2). The resulting lake trout deaths per spawner of 1.68 is somewhat higher than that calculated for the other lakes (excluding the value for Lake Superior including siscowet lake trout), possibly indicating that sea lamprey are experiencing more abundant hosts in that lake, and thus are feeding at rates closer to their maximum possible rate. It is worth noting that applications of the IMSL model have suggested that sea lamprey are feeding near their maximum rate, but the modeled estimates of returning spawners

were generally less than those estimated by the spawner-discharge regression (Larson *et al.* 2003).

CONCLUDING REMARKS

Predicting how host mortality will respond to changes in sea lamprey control is central to the IPM approach (Sawyer 1980) that has been embraced by the GLFC (Koonce *et al.* 1993). In this paper the large amount of uncertainty surrounding all existing estimates of host damages was emphasized. Furthermore, there are substantial inconsistencies both in underlying assumptions and in conclusions regarding damages among approaches currently being used. The discrepancies are large enough that one could be tempted to give up the enterprise entirely. However, there is reason to take a more optimistic view. The review presented in this paper suggests efforts that would reduce this uncertainty.

More than a quarter of a century ago Farmer *et al.* (1975) examined the relationship between sea lamprey feeding rate and the time from initiation of an attachment to host death. These results were used by Farmer and others (Farmer *et al.* 1975, Farmer 1980, Cochran and Kitchell 1989, Kitchell 1990, Madenjian *et al.* 2003) to infer the potential lethality of sea lamprey attacks and to predict damages on host populations. This work has evolved from simple calculations based on averages toward more complex models taking into account bioenergetic relationships, seasonal temperature and growth patterns, and variation in feeding rates and attachment times. This approach has much promise as it is practical to measure key information such as the total growth achieved during the parasitic phase (Kitchell 1990, Houston and Kelso 1991, Bergstedt and Swink 1995) and temperature occupied by hosts (Bergstedt *et al.* 2003a). However, the apparently higher probability of surviving sea lamprey attacks that have been predicted from these models contrasts with estimates based on laboratory data summarized by Swink (2003) and with some calibration of models to field observations (Schneider *et al.* 1996, Larson *et al.* 2003), and this needs to be resolved. To some extent the difference could be due to delayed mortality following detachment (Madenjian *et al.* 2003). There remains, however, great uncertainty in the understanding of the relationship between blood loss and host mortality. Farmer's widely used relationship between feeding rate and days to death was estimated for a group of 16 fish representing rainbow trout and lake trout exposed to blood loss ranging from 25% to the

equivalent of over 100% of their blood volume in a day. By necessity, these results have been extrapolated to much lower percentage rates of host blood loss characteristic of single attacks on lake trout in the field. Additional laboratory study of the response of hosts to varying controlled rates of blood loss, including characterization of ability of hosts to maintain blood quality and post attachment responses of initially surviving hosts, would help resolve these uncertainties.

Another important area of uncertainty is in inferring sea lamprey mortality from observed numbers of sea lamprey marks on surviving hosts. Theory underlying existing procedures has been laid out here, and it was noted that the connection between sea lamprey-induced mortality and wounding rates depends upon how rapidly wounds heal, the average length of attachment, and the probability of surviving an attack. There is some potential to extract additional information on the lethality of sea lamprey attacks from existing data, but this will require a comprehensive and simultaneous analysis of multiple data sources to obtain sufficient contrast (Stewart *et al.* 2003). Furthermore, some existing procedures assume that mortality occurs at the completion of an attachment and not after a substantial delay. Tagging studies have some potential to provide information on both the duration of sea lamprey marks in various stages and the latent effects after a detachment. Such studies could allow examination of the same wound on a tagged lake trout after some time at large, and also a comparison of survival of fish tagged when they have a fresh A1 wound versus survival of fish that had not recently been wounded. Marking data collected from surviving hosts remains a critical information source, and because of issues associated with the quality of these data (Ebener *et al.* 2003), investment in continual standardized training of observers is also recommended.

Some evidence suggestive of dynamic behavioral responses by sea lamprey to host availability was presented. However, this evidence comes from a contrast of conditions where preferred hosts such as large lake trout were extremely scarce and conditions where a potential spawning stock of lake trout is developing. The complexity involved in incorporating such behavior in management models might not be necessary if the scope of application of the predictive models is to lake trout populations approaching target abundances and size-compositions. This view contrasts sharply with suggestions in SLIS-I, that dynamic behavior of sea lamprey might

cause a shift from a predatory to a parasitic mode as lake trout become rehabilitated (Christie and Kolenosky 1980, Kitchell and Breck 1980, Walters *et al.* 1980). Current management models are extravagant simplifications of the spatial relationships between sea lamprey and hosts, but this is an area that has not received much attention since SLIS-I. In the context of dynamic behavior, it seems likely that sea lamprey change their spatial distributions in response to host abundances and distributions. Dynamic behavior in general is an area needing further consideration. In this regard, an improved understanding of the apparently anomalously high marking rates seen in Cayuga Lake, Lake Champlain, and on Siscowet lake trout on Lake Superior is critical.

Similarly, sea lamprey may have a substantial numerical response due to a relationship between survival of young parasites and the availability of suitable hosts. Jensen (1994) modeled uncontrolled sea lamprey populations feeding on lake trout and alternative prey and included a numerical response to lake trout abundance. The review presented here supports Stewart *et al.*'s (2003) recommendation to take advantage of improved estimates of both spawner abundance (e.g., Mullett *et al.* 2003) and the abundance of recently metamorphosed parasites (e.g., Slade *et al.* 2003) to examine how the ratio of spawner to metamorphosing parasite abundance varies over time within and among the Great Lakes, especially as host abundance changes.

This review has covered a number of topics and identified areas of uncertainty associated with each. It has indicated that the existing IMSL submodel for sea lamprey parasite-host interactions is a simplification of the actual processes in the Great Lakes, and considered the value of detailed models of the seasonal dynamics of the predation process, such as IBMs. It is not being suggested that complex individual-based models replace simplified management models. Instead, a major contribution of detailed IBMs might be to help improve the parameterization of simpler models used in management. It is hoped that the review presented here will encourage some key data collection activities and analyses, and promote further exploration of the sensitivity of existing models to their underlying assumptions.

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REFERENCES

- Applegate, V.C. 1950. *Natural history of the sea lamprey*, *Petromyzon marinus*, in Michigan. United States Fish and Wildlife Service Special Scientific Report: Fisheries No. 55.
- Bergstedt, R.A., and Schneider, C.P. 1988. Assessment of sea lamprey (*Petromyzon marinus*) predation by recovery of dead lake trout (*Salvelinus namaycush*) from Lake Ontario, 1982–85. *Can. J. Fish. Aquat. Sci.* 45:1406–1410.
- , and Seeley, J.G. 1995. Evidence for lack of homing by sea lampreys. *Trans. Am. Fish. Soc.* 124: 235–239.
- , and Swink, W.D. 1995. Seasonal growth and duration of the parasitic life stage of landlocked sea lampreys (*Petromyzon marinus*). *Can. J. Fish. Aquat. Sci.* 52: 1257–1264.
- , Argyle, R.L., Seelye, J.G., Scribner, K.T., and Curtis, G.L. 2003a. *In situ* determination of the annual thermal habitat used by lake trout (*Salvelinus namaycush*) in Lake Huron. *J. Great Lakes Res.* 29 (Suppl. 1):347–361.
- , McDonald, R.B., Mullett, K.M., Wright, G.M., Swink, W.D., and Burnham, K.P. 2003b. Mark-recapture population estimates of parasitic sea lampreys (*Petromyzon marinus*) in Lake Huron. *J. Great Lakes Res.* 29 (Suppl. 1):226–239.
- Berst, A.H., and Spangler, G.R. 1970. Population dynamics of F1 splake (*Salvelinus fontinalis* X *S. namaycush*) in Lake Huron. *J. Fish. Res. Board Can.* 27: 1017–1032.
- , and Wainio, A.A. 1967. Lamprey parasitism of rainbow trout in southern Georgian Bay. *J. Fish. Res. Board Can.* 24: 2539–2548.
- Budd, J.C., Fry, F.E.J., and Pearlstone P.S.M. 1969. Final observations on the survival of planted lake trout in South Bay, Lake Huron. *J. Fish. Res. Board Can.* 26:2413–2424.
- Chesson, J. 1978. Measuring preference in selective predation. *Ecology* 59:211–215.

- . 1983. The estimation and analysis of preference and its relationship to foraging models. *Ecology* 64:1297–1304.
- Christie, W.J., and Kolenosky, D.P. 1980. Parasitic phase of the sea lamprey (*Petromyzon marinus*) in Lake Ontario. *Can. J. Fish. Aquat. Sci.* 37:2021–2038.
- Cochran, P.A. 1985. Size-selective attack by parasitic lampreys: consideration of alternate null hypotheses. *Oecologia* (Berl.) 67:137–141.
- . 1986a. The daily timing of lamprey attacks. *Env. Biol. Fishes* 16:325–329.
- . 1986b. Attachment sites of parasitic lampreys: comparisons among species. *Env. Biol. Fishes* 17:71–79.
- . 1994. Why lampreys and humans “compete” (and when they don’t): toward a predictive theory of host species selection by parasitic lampreys. In *Theory and application in fish feeding ecology*, eds. D.J. Stouder, K.L. Fresh, and R.J. Feller, pp. 329–345. Belle W. Baruch Library in Marine Science, No. 18. University of South Carolina, Columbia, SC.
- , and Jenkins, R.E. 1994. Small fishes as hosts for parasitic lampreys. *Copeia* 1994:499–504.
- , and Kitchell, J.F. 1986. Use of modelling to investigate potential feeding strategies of parasitic lampreys. *Env. Biol. Fishes* 16:219–223.
- , and Kitchell, J.F. 1989. A model of feeding by parasitic lampreys. *Can. J. Fish. Aquat. Sci.* 46:1845–1852.
- , Swink, W.D., and Kinziger, A.P. 1999. Testing and extension of a sea lamprey feeding model. *Trans. Am. Fish. Soc.* 128:403–413.
- , Hodgson, J.Y., and Kinziger, A.P. 2003. Change in energy density of the sea lamprey (*Petromyzon marinus*) during its parasitic phase: implications for modeling food consumption and growth. *J. Great Lakes Res.* 29 (Suppl. 1):297–306.
- Collins, J.J. 1971. Introduction of Kokanee Salmon (*Oncorhynchus nerka*) into Lake Huron. *J. Fish. Res. Board Can.* 28:1857–1871.
- Cook, R.M., and Cockrell, B.J. 1978. Predator ingestion rate and its bearing on the theory of optimal diets. *J. Anim. Ecol.* 47:529–549.
- Ebener, M.P. 1995. *Methods used to estimate population size, mortality and growth of predatory fishes in western U.S. waters of Lake Superior*. Report submitted to Red Cliff Band of Lake Superior Chippewas.
- , Selgeby, J., Gallinat, M., and Donofrio, M. 1989. *Methods for determining total allowable catch of lake trout in the 1842 treaty-ceded area within Michigan waters of Lake Superior, 1990–1994*. Great Lakes Indian Fish and Wildlife Commission, Administrative Report 89-11, Odanah, Wisconsin.
- , Johnson, J.E., Reid, D.M., Payne, N.P., Argyle, R.L., Wright, G.M., Kruger, K., Baker, J.P., Morse, T., and Weise, J. 1995. Status and future of Lake Huron fish communities. In *The Lake Huron Ecosystem: Ecology, Fisheries, and Management*, eds. M. Munawar, T. Edsall, and J. Leach, pp. 125–169. Amsterdam, The Netherlands. Ecovision World Monograph Series, SPB Academic Publishing.
- , Bence, J.R., Bergstedt, R.A., and Mullett, K.M. 2003. Classifying sea lamprey marks on Great Lakes lake trout: observer agreement, evidence on healing times between classes, and recommendations for reporting of marking statistics. *J. Great Lakes Res.* 29 (Suppl. 1):283–296.
- Eshenroder, R.L., and Koonce, J.F. 1984. *Recommendations for standardizing the reporting of sea lamprey marking data*. Great Lakes Fishery Commission Spec. Publ. No. 84-1.
- , Payne, N.R., Johnson, J.E., Bowen, C., II, and Ebener, M.P. 1995. Lake trout rehabilitation in Lake Huron. *J. Great. Lakes Res.* 21(Supplement 1):108–127.
- Farmer, G.J. 1980. Biology and physiology of feeding in adult lampreys. *Can. J. Fish. Aquat. Sci.* 37:1751–1761.
- , and Beamish, F.W.H. 1973. Sea lamprey (*Petromyzon marinus*) predation on freshwater teleosts. *J. Fish. Res. Board Can.* 30:601–605.
- , Beamish, F.W.H., and Robinson, G.A. 1975. Food consumption of the adult landlocked sea lamprey, *Petromyzon marinus*, L. *Comp. Biochem. Physiol.* 50A:753–757.
- , Beamish, F.W.H., and Lett, P.F. 1977. Influence of water temperature on the growth rate of the landlocked sea lamprey (*Petromyzon marinus*) and the associated rate of host mortality. *J. Fish. Res. Board Can.* 34:1373–1378.
- Fetterolf, C.M. 1980. Why a Great Lakes Fishery Commission and why a Sea Lamprey International Symposium. *Can. J. Fish. Aquat. Sci.* 37:1588–1593.
- Fry, F.E.J. 1953. The 1944 year class of lake trout in South Bay, Lake Huron. *Trans. Am. Fish. Soc.* 82:178–192.
- Greig, L., Meisner, D., and Christie, G. 1992. *Manual for the management protocol for the implementation of integrated management of sea lamprey in the Great Lakes Basin*. Version 1.1. Great Lakes Fishery Commission.
- Hall, A.E. Jr., and Elliott, O.R. 1954. Relationship of length of fish to incidence of sea lamprey scars on white suckers, *Catostomus commersoni*, in Lake Huron. *Copeia* 1954:73–74.
- Halliday, R.G. 1991. Marine distribution of the sea lamprey (*Petromyzon marinus*) in the northwest Atlantic. *Can. J. Fish. Aquat. Sci.* 48:832–842.
- Hanson, L.H., and Swink, W.D. 1989. Downstream migration of recently metamorphosed sea lampreys in the Ocqueoc River, Michigan, before and after treatment with lampricides. *N. Am. J. Fish. Manage.* 9:327–331.
- Heinrich, J.W., Weise, J.G., and Smith, B.R. 1980.

- Changes in biological characteristics of the sea lamprey (*Petromyzon marinus*) as related to lamprey abundance, prey abundance, and sea lamprey control. *Can. J. Fish. Aquat. Sci.* 37:1861–1871.
- Holling, C.S. 1959. The components of predation as revealed by a study of small mammal predation of the European pine sawfly. *Can. Ent.* 91: 293–320.
- . 1961. Principles of insect predation. *Ann. Rev. Entomol.* 6: 163–182.
- Houston, K.A., and Kelso, J.R.M. 1991. Relation of sea lamprey size and sex ratio to salmonid availability in three Great Lakes. *J. Great Lakes Res.* 17:270–280.
- Jacobson, L.D. 1989. New approach to measuring the frequency of sea lamprey wounds in fish stocks in the Great Lakes. *N. Am. J. Fish. Manage.* 9:23–34.
- Jensen, A.L. 1994. Larkin's predation model of lake trout (*Salvelinus namaycush*) extinction with harvesting and sea lamprey (*Petromyzon marinus*) predation: a qualitative analysis. *Can. J. Fish. Aquat. Sci.* 51:942–945.
- Johnson, B.G.H., and Anderson, W.C. 1980. Predatory-phase sea lampreys (*Petromyzon marinus*) in the Great Lakes. *Can. J. Fish. Aquat. Sci.* 37:2007–2020.
- Jones, M.J., Koonce, J.F., and O'Gorman, R. 1993. Sustainability of hatchery-dependent salmonine fisheries in Lake Ontario: the conflict between predator demand and prey supply. *Trans. Am. Fish. Soc.* 122:1019–1030.
- Juanes, F., and Smith, L.D. 1995. The ecological consequences of limb loss in decapod crustaceans: a review and prospectus. *J. Exp. Mar. Bio. Ecol.* 193:197–223.
- King, E.L. 1980. Classification of sea lamprey *Petromyzon marinus* attack marks on Great Lakes lake trout. *Can. J. Fish. Aquat. Sci.* 37:1989–2000.
- Kitchell, J.F. 1990. The scope of mortality caused by sea lamprey. *Trans. Am. Fish. Soc.* 119:642–648.
- , and Breck, J.E. 1980. Bioenergetics model and foraging hypothesis for sea lamprey (*Petromyzon marinus*). *Can. J. Fish. Aquat. Sci.* 37: 2159–2168.
- Koonce, J.F. 1982. A review of the adaptive management workshop addressing salmonid/lamprey management in the Great Lakes. Great Lakes Fishery Commission, Ann Arbor, Michigan, Special Publication 82-2.
- . 1986. Application of the results of adaptive environmental assessment methodology workshops sponsored by the Great Lakes Fishery Commission to the development and practice of multispecies fishery management. Great Lakes Fishery Commission, Ann Arbor, Michigan, Research Completion Report.
- . 1987. Application of models of lake trout/sea lamprey interaction to the implementation of integrated pest management of sea lamprey in Lake Ontario. Great Lakes Fishery Commission, Ann Arbor, Michigan, Project Completion Report.
- , and Locci-Hernandez, A.B. 1989. A decision support system for the integrated management of sea lamprey. Great Lakes Fishery Commission, Ann Arbor Michigan, Special Publication 89-1.
- , and Pycha, R.L. 1985. Observability of lake trout mortality due to attacks of sea lamprey. Great Lakes Fishery Commission, Ann Arbor, Michigan, Research Completion Report.
- , Schneider, C.P., and Dentry, W. 1987. Effects of fishing mortality on estimation of lethality of sea lamprey attacks on lake trout. Great Lakes Fishery Commission, Ann Arbor, Michigan, Research Progress Report.
- , Christie, G.C., and Eshenroder, R.L. 1993. An economic injury level approach to establishing the intensity of sea lamprey control in the Great Lakes. *N. Am. J. Fish. Manage.* 13:1–14.
- Kowalewski, M., and Flessa, K.W. 2000. Seasonal predation by migratory shorebirds recorded in shells of lingulid brachiopods from Baja California, Mexico. *Bull. Mar. Sci.* 66:405–416.
- Larson, G.L., Christie, G.C., Johnson, D.A., Koonce, J.F., Mullett, K.M., and Sullivan, W.P. 2003. The history of sea lamprey control in Lake Ontario and updated estimates of suppression targets. *J. Great Lakes Res.* 29 (Suppl. 1):637–654.
- Lawrie, A.H., and MacCallum, W. 1980. On evaluating measures to rehabilitate lake trout (*Salvelinus namaycush*) of Lake Superior. *Can. J. Fish. Aquat. Sci.* 37:2057–2062.
- Lennon, R.E. 1954. Feeding mechanism of the sea lamprey and its effect on host fishes. *U.S. Fish Wildl. Serv. Fish. Bull.* 56:247–293.
- 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.
- , Cochran P.A., and Bergstedt, R.A. 2003. Seasonal patterns in growth, blood consumption, and effects on hosts by parasitic-phase sea lampreys in the Great Lakes: an individual-based model approach. *J. Great Lakes Res.* 29 (Suppl. 1):332–346.
- Manly, B.F.J., Miller, P., and Cook, L.M. 1972. Analysis of a selective predation experiment. *Am. Nat.* 106:719–736.
- Marsden, J.E., Chipman, B.D., Nashett, L.J., Anderson, J.K., Bouffard, W., Durfey, L., Gersmehl, J.E., Schoch, W.F., Staats, N.R., and Zerrenner, A. 2003. Sea lamprey control in Lake Champlain. *J. Great Lakes Res.* 29 (Suppl. 1):655–676.
- Moore, J.D., and Lychwick, T.J. 1980. Changes in mortality of lake trout (*Salvelinus namaycush*) in relation to increased sea lamprey (*Petromyzon marinus*) abundance in Green Bay, 1974–78. *Can. J. Fish. Aquat. Sci.* 37:2052–2056.
- Morin, P.J. 1985. Predation intensity, prey survival and

- injury frequency in an amphibian predator-prey interaction. *Copeia* 1985: 638–644.
- Morse, T.J., Ebener, M.P., Koon, E.M., Morkert, S.B., Johnson, D.A., Cuddy, D.W., Weisser, J.W., Mullett, K.M., and Genovese, J.H., 2003. A case history of sea lamprey control in Lake Huron: 1979 to 1999. *J. Great Lakes Res.* 29 (Suppl. 1):599–614.
- Mullett, K.M., Heinrich, J.W., Adams, J.V., Young, R.J., Henson, M.P., McDonald, R.B., and Fodale, M.F. 2003. Estimating lake-wide abundance and spawning-phase sea lampreys (*Petromyzon marinus*) in the Great Lakes: extrapolating from sampled streams using regression models. *J. Great Lakes Res.* 29 (Suppl. 1):240–252.
- Murdoch, W.W. 1971. The developmental response of predators to changes in prey density. *Ecology* 52:132–137.
- . 1973. The functional response of predators. *J. Applied Ecology* 10:335–342.
- Murtaugh, P.A. 1981. Inferring properties of mysid predation from injuries to *Daphnia*. *Limnol. Oceanogr.* 26:811–821.
- Mushinski, H.R., and Miller, D.E. 1993. Predation on water snakes—ontogenetic and interspecific considerations. *Copeia* 1993:660–665.
- Oji, T. 1996. Is predation intensity reduced with depth? Evidence from the west Atlantic stalked cinoid *Endoxocrinus parrae* (Gervais) and implications for the Mesozoic marine revolution. *Paleontology* 22: 339–351.
- Parker, P.S., and Lennon, R.E. 1956. *Biology of the sea lamprey in its parasitic phase*. U.S. Fish Wildl. Serv. Res. Rep. 44.
- Pearce, W.A., Braem, R.A., Dustin, S.M., and Tibbles, J.J. 1980. Sea lamprey (*Petromyzon marinus*) in the Lower Great Lakes. *Can. J. Fish. Aquat. Sci.* 37:1802–1810.
- Peddle, S.M., and Larson, D.J. 1999. Cuticular evidence of traumatic experiences of water beetles (Coleoptera: Dytiscidae, Hydophilidae). *Coleopt. Bull.* 53:42–51.
- Pycha, R.L. 1980. Changes in mortality of lake trout (*Salvelinus namaycush*) in Michigan waters of Lake Superior in relation to sea lamprey (*Petromyzon marinus*) predation, 1968–78. *Can. J. Fish. Aquat. Sci.* 37:2063–2073.
- , and King, G.R. 1975. *Changes in the lake trout population of southern Lake Superior in relation to the fishery, the sea lamprey, and stocking, 1950–70*. Great Lakes Fishery Commission, Ann Arbor, Michigan, Tech. Rep. No. 28.
- Quinn, T.J., and Deriso, R.B. 1999. *Quantitative Fish Dynamics*. New York: (Oxford University Press.)
- Ricker, W.E. 1975. *Computation and interpretation of biological statistics of fish populations*. Fish. Res. Board Can. Bull. 191.
- Robson, D.S. 1970. *Estimation of instantaneous lamprey attack rate. (Based on a Markov model of lamprey predation with non-stationary attack rates and survival rates.)* Biometrics Unit Technical Report, Mimeo Series. BU-276-M.
- Ross, S.M. 1980. *Introduction to probability models*. Second edition. Orlando, Florida. Academic Press, Inc.
- Royce, W.F. 1949. The effects of lamprey attacks upon lake trout in Seneca Lake, New York. *Trans. Am. Fish. Soc.* 79:71–75.
- Rosenzweig, M. 1971. Paradox of enrichment: destabilization of exploitation ecosystems in ecological time. *Science* 171:385–387.
- Rutter, M.A., and Bence, J.R. 2003. An improved method to estimate sea lamprey wounding rate on hostes with application to lake trout in Lake Huron. *J. Great Lakes Res.* 29 (Suppl. 1):320–331.
- Sawyer, A.J. 1980. Prospects for integrated pest management of the sea lamprey (*Petromyzon marinus*). *Can. J. Fish. Aquat. Sci.* 37:2081–2092.
- Schneider, C.P., Owens, R.W., Bergstedt, R.A., and O’Gorman, R. 1996. Predation by sea lamprey (*Petromyzon marinus*) on lake trout (*Salvelinus namaycush*) in southern Lake Ontario, 1982–1992. *Can. J. Fish. Aquat. Sci.* 53:921–932.
- Schoener, T.W. 1979. Inferring the properties of predation and other injury-producing agents from injury frequencies. *Ecology* 60:1110–1115.
- , and Schoener, A. 1982. Ecological and demographic correlates of injury rates in some Bahamian *Anolis* lizards. *Copeia* 1980:839–850.
- Shargal, E., Rath-Wolfson, L., Kronfeld, N., and Dayan, T. 1999. Ecological and histological aspects of tail loss in spiny mice (Rodentia: Muridae, Acomys) with review of its occurrence in rodents. *J. Zool.* 249:187–193.
- Shetter, D.S. 1949. A brief history of the sea lamprey problem in Michigan waters. *Trans. Am. Fish. Soc.* 76:160–176.
- Sih, A. 1980. Optimal foraging: partial consumption of prey. *Am. Nat.* 116:281–290.
- Sitar, S.P., Bence, J.R., Taylor, W.W., and Johnson, J.E. 1997. Sea Lamprey Wounding Rates on Lake Trout in Lake Huron, 1984–1994. *Michigan Academician* XXIX:21–37.
- , Bence, J.R., Johnson, J.E., Ebener, M.P., and Taylor, W.W. 1999. Lake trout mortality and abundance in southern Lake Huron. *N. Am. J. Fish. Manage.* 19:881–900.
- Slade, J.W., Adams, J.V., Christie, G.C., Cuddy, D.W., Fodale, M.F., Heinrich, J.W., Quinlan, H.R., Weise, J.G., Weisser, J.W., and Young, R.J. 2003. Techniques and methods for estimating abundance of larval and metamorphosed sea lampreys in Great Lakes tributaries, 1995 to 2002. *J. Great Lakes Res.* 29 (Suppl. 1):137–151.
- Smith, B.R. 1980. Introduction to the proceedings of the 1979 sea lamprey international symposium (SLIS). *Can. J. Fish. Aquat. Sci.* 37:1585–1587.

- _____, and Tibbles, J.J. 1980. Sea lamprey (*Petromyzon marinus*) in Lakes Huron, Michigan and Superior: history of invasion and control, 1936–78. *Can. J. Fish. Aquat. Sci.* 37: 1780–1801.
- _____, Tibbles, J.J., and Johnson, B.G.H. 1974. *Control of the sea lamprey, Petromyzon marinus, in Lake Superior, 1953–70*. Great Lakes Fishery Commission, Ann Arbor, Michigan, Technical Report 26.
- Solomon, M.E. 1949. The natural control of animal populations. *J. Anim. Ecol.* 18:1–35.
- Spangler, G.R., and Collins, J.J. 1980. Response of lake whitefish (*Coregonus clupeaformis*) to the control of sea lamprey (*Petromyzon marinus*) in Lake Huron. *Can. J. Fish. Aquat. Sci.* 37:2039–2046.
- _____, and Jacobson, L.D. (eds.) 1985. *A workshop concerning the application of integrated pest management (IPM) to sea lamprey control in the Great Lakes*. Great Lakes Fishery Commission, Ann Arbor, Michigan. Special Publication 85-2.
- _____, Robson, D.S., and Regier, H.A. 1980. Estimates of lamprey-induced mortality in whitefish, *Coregonus clupeaformis*. *Can. J. Fish. Aquat. Sci.* 37:2146–2150.
- Stewart, T.J., Bence, J.R., Bergstedt, R.A., Ebener, M.P., Lupi, F., and Rutter, M.A. 2003. Recommendations for assessing sea lamprey damages: toward optimizing the control program in the Great Lakes. *J. Great Lakes Res.* 29 (Suppl. 1):783–793.
- Sullivan, W.P., Christie, G.C., Cornelius, F.C., Fodale, M.F., Johnson, D.A., Koonce, J.F., Larson, G.L., McDonald, R.B., Mullett, K.M., Murray, C.K., and Ryan, P.A. 2003. The sea lamprey in Lake Erie: a case history. *J. Great Lakes Res.* 29 (Suppl. 1):615–636.
- Swanson, B.L., and Swedberg, D.V. 1980. Decline and recovery of the Lake Superior Gull Island Reef lake trout (*Salvelinus namaycush*) population and the role of sea lamprey (*Petromyzon marinus*). *Can. J. Fish. Aquat. Sci.* 37:2074–2080.
- Swink, W.D. 1990. Effect of lake trout size on survival after a single sea lamprey attack. *Trans. Am. Fish. Soc.* 119:996–1002.
- _____. 1991. Host-size selection by parasitic sea lampreys. *Trans. Am. Fish. Soc.* 120:637–643.
- _____. 1995. Growth and survival of newly parasitic sea lampreys at representative winter temperatures. *Trans. Am. Fish. Soc.* 124:380–386.
- _____. 2003. Host selection and lethality of attacks by Sea Lamprey (*Petromyzon marinus*) in laboratory studies. *J. Great Lakes Res.* 29 (Suppl. 1):307–319.
- _____, and Hanson, L.H. 1986. Survival from sea lamprey (*Petromyzon marinus*) predation by two strains of lake trout (*Salvelinus namaycush*). *Can. J. Fish. Aquat. Sci.* 43:2528–2531.
- _____, and Hanson, L.H. 1989. Survival of rainbow trout and lake trout after sea lamprey attack. *N. Am. J. Fish. Manage.* 9:35–40.
- Walters, C.J., and Post, J.R. 1993. Density-dependent growth and competitive asymmetries in size-structured fish populations—a theoretical-model and recommendations for field experiments. *Trans. Am. Fish. Soc.* 122:34–45.
- _____, Spangler, G., Christie, W.J., Manion, P.J., and Kitchell, J.F. 1980. A synthesis of knowns, unknowns, and policy recommendations from the Sea Lamprey International Symposium. *Can. J. Fish. Aquat. Sci.* 37:2202–2208.
- Wigley, R.L. 1959. Life history of the sea lamprey of Cayuga Lake, New York. *U.S. Fish. Wildl. Serv. Fish. Bull.* 59:559–617.
- Young, R.J., Christie, G.C., McDonald, R.B., Cuddy, D.W., Morse, T.J., and Payne, N.R. 1996. Effects of habitat change in the St. Marys River and northern Lake Huron on sea lamprey (*Petromyzon marinus*) populations. *Can. J. Fish. Aquat. Sci.* 53 (Suppl. 1):99–104.
- Youngs, W.D. 1980. Estimate of lamprey-induced mortality in a lake trout (*Salvelinus namaycush*) population. *Can. J. Fish. Aquat. Sci.* 37:2151–2158.

APPENDIX 1

Here the assumption of constant attack rate and probability of surviving an attack is relaxed. If for some arbitrarily short interval of length δ , starting at time t , the probability of an attack is $A(t)$, the probability of multiple attacks is low, and the probability of attacks is independent of previous attacks, then the accumulation of attacks is a nonhomogeneous Poisson process (Ross 1980). If survival of an attack is independent of previous attacks, the accumulation of wounds will also be a nonhomogeneous Poisson process, even when $P_S(t)$ varies over time also. This can be seen by noting that lethal and non-lethal attacks occur independently, so the rate of accumulation of non-lethal attacks is $A(t)P_S(t)$, and this is the same on survivors as on all fish and meets the conditions for a non-homogenous Poisson process. Note that no new assumptions have been imposed, the previous assumptions have been modified to allow for time varying rates.

Now, with the definition

$$\Lambda(t+H) = \int_0^H A(t+s)ds \quad (A1)$$

the expected attacks from time t to time $t+H$ (given that the fish survives the period) is $\Lambda(t+H)$ and the number of attacks will have a Poisson distribution (Ross 1980). Likewise,

$$\Omega(t+H) = \int_0^H A(t+s)P_S(t+s)ds \quad (A2)$$

is the expected number of wounds on surviving fish and wounds per fish also has a Poisson distribution.

With the mean attack rate for the period defined as the time average:

$$\bar{A}(t+H) = \Lambda(t+H) / H \quad (\text{A3})$$

and the mean probability of surviving an attack as the average over all attacks during the period:

$$\bar{P}_S(t+H) = \frac{\Omega(t+H)}{\Lambda(t+H)} \quad (\text{A4})$$

the averages defined in equations A3 and A4 can replace A and P_S in the relationships between mortality and wounds per fish given in the main text, provided that total mortality rates are not too high (generally $Z < 1.0$). This claim is based on similar observations regarding time-varying fishing rates, which have modest effect on total annual mortality unless total mortality rates are quite high. Strictly speaking M_L (sea lamprey mortality) calculated from these averages is not the instantaneous rate of sea lamprey mortality (since that is varying) and adequacy of the approximation refers to the closeness of the total fraction of fish that will die over a time period of length Y and $1 - e^{-(F+M+M_L)Y}$.

APPENDIX 2

Here the various estimates of lake trout deaths and sea lamprey spawning populations, and use of these to calculate lake trout deaths per spawner are described. For Lake Huron, Lake Michigan, and

Lake Superior lake trout deaths are averages for 1990 to 1998, and are based on unpublished lake trout stock assessments developed in 2000 by the Technical Fisheries Review Committee's Modeling Group and by the Michigan DNR. Sea lamprey mortality rates were calculated following equation 10, as described by Sitar *et al.* (1999). Wounding rates came from spring or spring/early summer survey data for Lake Huron and Lake Superior and autumn surveys in Lake Michigan. Use of autumn wounding data for Lake Michigan was justified based on the observations that wounding in autumn and spring were of comparable magnitude, and Lake Michigan estimates were extrapolated to the entire lake (Madenjian *et al.* 2002). Siscowet abundance in Lake Superior was estimated by depth and size class based on relative catch per effort of siscowet and lean lake trout as by Ebener (1995) and a natural mortality rate of 0.1 was assumed in application of the Baranov catch equation. Estimates of lake trout deaths due to sea lamprey for Lake Ontario are based on Schneider *et al.*'s (1996) carcass estimates averaged for the years they report results for and expanded by a factor of 4.0 to account for decomposition and unsampled Ontario waters. Sea lamprey spawning estimates are averages of discharge regression estimates (Mullett *et al.* 2003) for the cohorts producing the reported lake trout loss estimates.

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