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Modelling the proximate basis of salmonid life-history variation, with application to Atlantic salmon, *Salmo salar* L.

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Summary

The great diversity of life-history patterns in the salmonids has stimulated many theoretical studies. However, virtually all studies are based on ultimate considerations, in which predictions are made by comparing the expected reproductive success of different developmental or life-history pathways and choosing the one (or ones) with the highest fitness. Such models are *post hoc* because they attribute fitness to individuals at the completion of the particular phase of the life cycle and do not attempt to characterize the mechanisms that animals use to achieve the life-history pattern. We describe a model, based on proximate considerations, for salmonid life histories, focused on Atlantic salmon *Salmo salar* L. The model involves identification of the times at which developmental conversions are initiated or inhibited and the connection between physiological states and the thresholds for such conversions. Developmental paths are based on the comparison of the current physiological status of the fish (and its change of state) with a genetic threshold. The state of the fish and rate of change of state are determined by environmental opportunity, but the threshold is genetic. This approach therefore immediately generates a genotype–environment interaction. We use expected reproductive success to determine the fitness of individuals with different genetically determined thresholds. Instead of finding an optimal life history, our theory generates fitness surfaces for different life histories, so that variation is inherent in this approach. We describe and explain the structure of the model and present evidence on which this structure is based, thus providing a framework within which one can understand how ecology relates to the physiological mechanisms leading to the developmental changes of smolt metamorphosis and maturation.

Keywords: life-history evolution; maturation; phenotypic plasticity; salmonid fish; *Salmo salar*; smolt metamorphosis

Ultimate and proximate life-history models

Life-history strategies are the means by which organisms achieve successful reproduction in varying environments. Life-history theory generally treats such strategies from the viewpoint of their ultimate fitness – measuring fitness in terms of the number of descendants or the number of genes in future generations. The great diversity of life-history patterns among salmonid fishes stimulated various aspects of life-history theory, including studies of age and size at maturity (e.g. Schaffer and Elson, 1975; Schaffer, 1979; Healey and Heard, 1984; Healey, 1986; Bohlin *et al.*, 1990; Holtby and Healey, 1990; Hutchings, 1993), alternative reproductive strategies (Gross, 1985, 1991; Hutchings and Myers, 1988, 1994 and references therein), smolt metamorphosis (Mangel, 1994) and other life-history characteristics (Hutchings and Morris, 1985). These studies all used models based on

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ultimate considerations (McNamara and Houston, 1996): predictions are made by comparing the fitness (expected reproductive success) of different developmental or life-history pathways and choosing the one (or ones) with the highest fitness. Models based on ultimate considerations are *post hoc* because they attribute fitness to individuals at the completion of the particular phase of the life cycle and do not attempt to characterize the mechanisms that animals use to achieve the optimum life-history pattern.

The developmental pathways that salmonids (and many other organisms) use to reach the state necessary for successful reproduction are the consequences of responses to the opportunities the environment offers them. In salmonid ontogeny, there are two major developmental conversions (*sensu* Smith-Gill, 1983): smolt metamorphosis, which is the complex of morphological, physiological and behavioural changes associated with the exchange of the freshwater for the marine environment (Hoar, 1976; Thorpe, 1982; Langdon and Thorpe, 1985), and sexual maturation. Each developmental conversion follows its own annual timetable.

Maturation is regulated by inhibition (Thorpe, 1986, 1994a); that is, maturation in salmonids is not 'switched on' but is continually repressed, until the inhibitor is removed.

Salmon life histories

Smolt metamorphosis

Atlantic salmon, *Salmo salar* L., spawn in the autumn. Embryos develop slowly throughout the winter, hatch in the spring well before the yolk supply is exhausted and begin to feed on external foods in April or May. The determination of whether an individual will undergo smolt metamorphosis (and so emigrate from the river) the following spring occurs soon after midsummer (Metcalfe *et al.*, 1986, 1988; Thorpe, 1986; see below). Some individuals show decline of appetite in late July or August, determined by growth rates and size prior to then. Typically, if appetite is arrested sharply in late July or early August, an individual will cease growth and reduce metabolic demand to a very low level until the following March; smolt metamorphosis does not occur in the following spring. By contrast, individuals who maintain appetite throughout the late summer and autumn usually undergo smolt metamorphosis the following spring.

Maturation

Maturation is a cyclic process that begins at fertilization. Germinal tissue differentiates very early and investment in gonadal growth begins during the embryo stage (Adams and Thorpe, 1989). Hence, the developmental processes associated with sexual maturation begin well before the time of first feeding (Thorpe, 1994a). Adams and Thorpe (1989) showed that females under good growing conditions (water warmed 5°C above Scottish ambient) did not mature in their first year, but did have higher reproductive investment (ovary weight) than those under normal growing conditions. It is therefore possible that females can reach full maturity at age 0+ and we have allowed this possibility. Completion of maturation within the first annual cycle depends upon adequate lipid and possibly other resources in the spring (Rowe and Thorpe, 1990a; Rowe *et al.*, 1991). It is difficult to determine which resource is most critical, since they tend to covary. However, empirical results are generally consistent with the idea that lipid reserves are important, so in this paper we treat them as the limiting resource.

If lipid reserves in the spring are not sufficient, further gonadal investment is arrested until November, which is the beginning of the fish's second year. At this time, if the fish has adequate resources, investment in gonadal tissue will restart (Thorpe, 1994a). Provided that lipid stores remain sufficiently high throughout the winter, and can be replenished during a period of rapid

growth in April and May, maturation will be maintained and the individual will be fully mature by the following November. However, if lipid stores are depleted over the winter to a level where they cannot be replenished in April and May, further gonadal investment is inhibited, and maturation is postponed for another year (Thorpe *et al.*, 1990; Rowe *et al.*, 1991; Thorpe, 1994a).

Individuals can reproduce without emigrating from their juvenile environment but, by definition, cannot reproduce without maturation. Thus, maturation takes evolutionary precedence over smolt metamorphosis (Thorpe, 1994b). Consequently, in November, the choice about restarting investment in gonads must be available to both smolting and non-smolting individuals.

The nature of the physiological assessment that determines the direction of development at the critical times is not clear. However, whether it is the turnover rate or the absolute amount of resources, it appears that the threshold levels vary between individuals, so that there is genetic variation in the thresholds. Hence, the course of the life history is determined by both ultimate regulators which, through natural or artificial selection, set the threshold levels in the genome, and through proximate regulators, which are the environmental opportunities that permit or prevent an individual from reaching the appropriate thresholds at the critical times.

In this paper, we describe a model for the life history of Atlantic salmon based on the proximate mechanisms that determine an individual's developmental pathway. That is, developmental paths are based on the comparison of the current physiological status of the fish (and its change of state) with a genetic threshold (cf. Roff, 1996). The state of the fish and rate of change of state are determined by environmental opportunity, but the threshold is genetic. This approach therefore immediately generates a genotype–environment interaction (Tyler and Rose, 1994).

As with some other life-history approaches, we use expected reproductive success to determine the fitness of individuals with different genetically determined thresholds. However, instead of finding an optimal life history, our theory generates fitness surfaces for different life histories (cf. Mangel and Ludwig, 1992), so that variation is inherent in this approach. Fitness is more easily defined for females (in which reproductive success clearly depends upon gonadal mass) than males (in which reproductive success depends to a varying extent upon the social environment) (Hutchings and Myers, 1988). We therefore concentrate on females, although our approach (and some of the data supporting it) could equally be applied to males.

The aims of this paper are to describe and explain the structure of the model and to present evidence on which this structure is based. Generating predictions using the model and testing these predictions against empirical data is the next stage of the work. Our goal here is to provide a framework within which one can understand how ecology relates to the physiological mechanisms leading to the developmental changes of smolt metamorphosis and maturation.

Developmental switches characterize maturation and emigration

In the model, we assume salmonids function according to developmental switches that control gonadal development and emigration (Mangel, 1994). The timing of these switches is based on current knowledge of Atlantic salmon in Scotland, but these could readily be adapted for other populations or species. We assume that photoperiod is the external cue that synchronizes smolt metamorphosis and maturation (Villarreal *et al.*, 1988; Clarke, 1989; Duston and Saunders, 1990).

Most salmon in Scotland reproduce in November; however, to do so, they must initiate physiological changes the previous November, at which time an individual responds to a developmental switch that determines the maturation process. In the model, this is designated by G_1. The response involves comparing a combination of the absolute level of lipid reserves and rate of change of lipid reserves with a genetically determined maturation threshold, which we designate by M_1. The justification for such a threshold is that lipids are required for both somatic function during the year

and development of gonads, which takes time. Thus, there is a correlation between the lipid state in the current November and the potential level of reproduction the following November. If the combination of lipid and rate of change of lipid is less than M_1, maturation is inhibited; otherwise, gonadal development continues. Thus, we assume that the fish assesses current state and rate of change of state and acts on these to the extent that the current values provide information about future ones.

Maturation can be halted in the following spring if growth performance has deteriorated (we discuss evidence for this below). Thus, in April, a second maturation switch (G_2) occurs and a similar comparison is made between the combination of lipids and rate of change of lipid and a second maturation threshold M_2.

If $G_i = 1$ when the combination of lipid and rate of change of lipid exceeds the threshold, then a fish that matures in November has followed the path $G_1 = 1$ the previous November and $G_2 = 1$ the previous April. A fish that does not mature could have followed either $G_1 = 0$ (in which case $G_2 = 0$ perforce) or $G_1 = 1$ but $G_2 = 0$ (in which case G_1 is reset to 0). The latter case would arise when growth opportunities between November and April were poor, so that by April the fish was no longer on a course to exceed the threshold M_2 associated with G_2. Since it is possible (through photoperiod and temperature manipulations) to produce fish that mature in the first November of their lives, $G_1 = 1$ at the time of fertilization.

The emigration switch (E) occurs in August. At that time, the fish compares its energetic status (for which we use size as a proxy) and rate of change of that status with a genetically determined emigration threshold (R). If the combination of state and rate of change of state exceeds the threshold, the fish follows a pathway leading to emigration the following spring (becoming a fish that metamorphoses into the smolt stage after 1 year in freshwater); otherwise, it follows a pathway leading to residence in the stream for at least another year. We assume that the gonadal switches dominate the emigration switch, so that $G_2 = 1$ implies that $E = 0$. Paths of fish that mature in freshwater when they are 6 months (0+ maturing) or 18 months (1+ maturing) are shown in Table 1; the paths of early and late smolting fish are shown in Table 2.

After a fish moves to the marine environment, the developmental switches G_1 and G_2 still determine the pattern of maturation and return to freshwater for reproduction, although the maturation thresholds may be reset.

We now briefly describe empirical evidence for this formulation. Simpson (1993) and Thorpe (1994a) found that gonadal growth commenced in November, but only in some fish (providing evidence for both the existence and timing of G_1). Stead (1996) also showed that steroid hormone levels in maturing salmon started to increase at this time. Moreover, condition factor (a measure of weight per length) of fish at this time is a good predictor of maturation in the following year (e.g. Bohlin *et al.*, 1994; Thorpe, 1994a). Friedland and Haas (1996) demonstrated that post-smolt

Table 1. Patterns of response to the developmental switches of 0+ and 1+ maturing fish

Year of life	Month	0+ maturing	1+ maturing
First	November	Fertilization, $G_1 = 1$	$G_1 = 1$
	April	Birth, $G_2 = 1$	$G_2 = 0$
	August	$E = 0$	$E = 1$
Second	November	Reproduction	$G_1 = 1 \Rightarrow E = 0$
	April	–	$G_2 = 1$
Third	November	–	Reproduction

Table 2. Patterns of response to the developmental switches of early and late smolting fish

Year of life	Month	Early smolting	Late smolting
First	November	Fertilization, $G_1 = 1$	$G_1 = 1$
	April	Birth, $G_2 = 0$	$G_2 = 0$
	May	–	–
	August	$E = 1$	$E = 0$
Second	November	$G_1 = 0$	$G_1 = 0$
	April	$G_2 = 0$	$G_2 = 0$
	May	Emigration	–
	August	–	$E = 1$
Third	November	–	$G_1 = 0$
	April	–	$G_2 = 0$
	May		Emigration

growth patterns in late summer affect the likelihood of a fish returning after one or two sea-winters, and Kadri *et al.* (1996) showed marked differential growth responses in maturing versus non-maturing fish from November onwards.

The timing of G_2 is supported by the work of Hunt *et al.* (1982), Johnston *et al.* (1987), Rowe and Thorpe (1990b), Kadri *et al.* (1996) and Stead (1996), all of whom showed that, from 1 April, condition factor increases in maturing fish and decreases in non-maturing fish. Similarly, McLay *et al.* (1992) showed that growth rates of maturing females differ significantly from non-maturing fish in March and April. Hunt *et al.* (1982) reported a separation in the condition factor of maturing and non-maturing fish. It is not possible to use their data as a predictive theory, however, because there is no indication of how condition factor and change in condition factor could be combined to predict whether a fish will mature or not.

Food restriction experiments performed by Thorpe *et al.* (1990) and Reimers *et al.* (1993) showed that maturation was suppressed by poor foraging opportunities in February to April; Berglund (1995) showed a similar effect in May and June. The slight discrepancy in the estimated timing of G_2 may be due to the difference in latitude between the two studies (55°N and 64°N, respectively). We anticipate that the precise timing of the developmental switches will depend upon latitude, since this affects the seasonal pattern of food availability. Berglund (1995) also showed that maturation depended upon environmental opportunity in the 3 weeks prior to the start of rapid gonadal growth; this suggests that the length of the assessment window over which the rate of change of lipid or weight is determined (see below) is 3–4 weeks. Moreover, smaller fish were most likely to switch off maturation in response to poor spring conditions, presumably since these were the least likely to attain the reserves needed for successful reproduction.

The timing of the E developmental switch was confirmed in independent studies of appetite by Metcalfe *et al.* (1986, 1988), body growth by Thorpe *et al.* (1989) and otolith growth by Wright *et al.* (1990). In each case, the developmental switches (G_1, G_2 or E) occur well in advance of the life-history event of interest. This sort of process is likely to be common in organisms whenever major physiological changes are required in advance of key life-history events, because such changes probably imply time lags in order for them to be implemented. In general, developmental switches that occur far in advance of the life-history event will use information that is less reliable than switches that occur close to the life-history event. For example, at the time of the G_1 switch, even if the fish has accurate information about its current lipid level and rate of change of lipid level, the actual level the following November will depend upon a myriad of factors, including its

feeding history, social status and the temperature profile during the following year. Thus, developmental processes occur in the face of considerable uncertainty. This has a number of implications. First, there will be a range of circumstances when individuals 'make mistakes', in that a path is chosen in November, for example, that would not have been chosen *post hoc*. Second, there is value in having the opportunity to 'correct' such mistakes. Third, rules of thumb, such as the threshold ones that we propose, are likely to be effective mechanisms for guiding the life history.

A computationally practicable description of the developmental switches and proximate mechanism

There is a difference between the mechanisms that the fish use (proximate factors) and the effect of natural selection on such mechanisms or our ability to describe them (ultimate factors). We now symbolically formalize the description given in the previous section. We consider each developmental switch separately, because the details of each differ in non-trivial ways, and begin with the G_1 switch.

As described above, the G_1 switch occurs in November, at the time that we denote t_1. Because the G_2 switch occurs the following April (at time t_2), we assume that M_1 is a value of lipid in April. That is, if the combination of lipid in November and rate of change of lipid leads to expected lipid levels in April exceeding M_1, maturation is continued at G_1. We assume that information obtained during an assessment window of length T_w preceding t_1 is used to determine the rate of change of lipid.

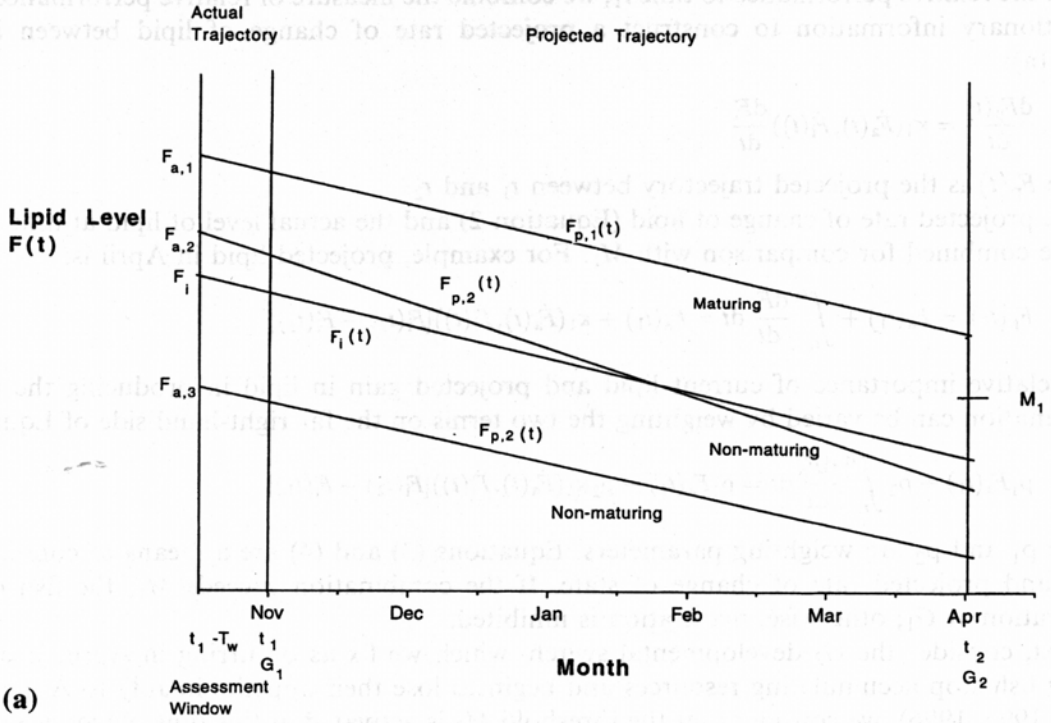
To combine lipid at t_1 and rate of change of lipid for comparison with the threshold at t_2 (Fig. 1), we assume that the fish have inherited an expected lipid trajectory that is characteristic of individual growth in their environment. We denote this inherited lipid trajectory by $F_i(t)$. Performance during the assessment window is measured as the rate of change of lipid during that period:

$$\dot{F}_i(t) = \frac{1}{T_w}[F_i(t_1) - F_i(t_1 - T_w)] \tag{1}$$

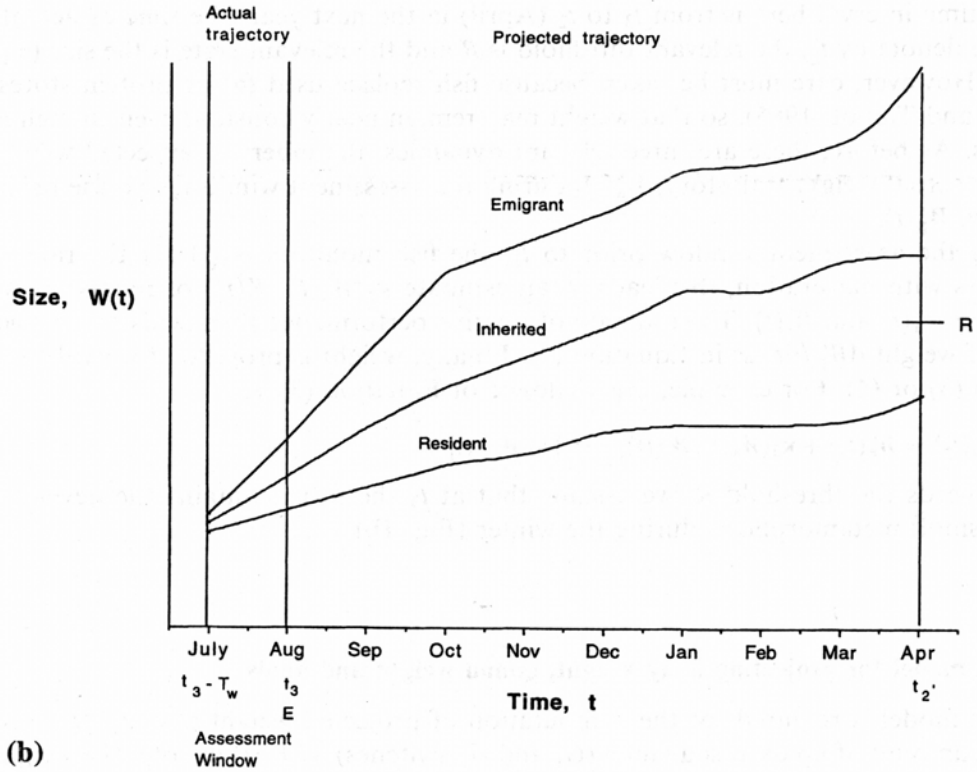
where T_w is the length of the assessment window, $F_i(t_1)$ is the lipid level at t_1, and $F_i(t_1 - T_w)$ is the lipid level at the start of the assessment window. As described above, the work of Berglund (1995) indicates that the length of the assessment window is about 3 weeks.

We assume that the fish monitors its actual rate of change of lipid $\dot{F}_a(t)$ during the assessment window. A comparison of the actual performance and the inherited (predicted) performance provides a measure of relative performance, $\kappa_1(\dot{F}_a(t), \dot{F}_i(t))$. One simple choice, for example, is that $\kappa_1(\dot{F}_a(t), \dot{F}_i(t)) = \dot{F}_a(t)/\dot{F}_i(t)$. In any case, the notion is that if $\kappa_1 = 1$, lipids are projected to change at the same rate as the inherited trajectory. Otherwise, lipids are projected to change at a rate either less than or greater than the inherited trajectory.

Figure 1. Schematic illustration of how the combination of state and rate of change of state is used when the developmental switch points occur. (a) During an assessment window prior to t_1 (November), the actual rate of change of lipid $\dot{F}_a(t)$ is monitored by the fish, compared to a reference trajectory $\dot{F}_i(t)$ and used to create a predicted trajectory $F_p(t)$ of lipid reserves. We show three examples, all of which involve a decrease in lipid during the winter. Fish #1 has lipid in November exceeding the threshold level M_1 and a projected trajectory that maintains lipid levels in April above M_1. Hence, it is on a maturing pathway. Although Fish #2 has lipid in November exceeding M_1, its projected lipid trajectory takes lipid below M_1 in April and hence it switches off maturation at t_1 by setting $G_1 = 0$. The lipid level of Fish #3 lies below M_1 in November and is projected to lie below it in April; hence it too switches off maturation. (b) Similar processes occur at the time of the E developmental switch. For simplicity, we have not labelled the trajectories, other than to indicate the inherited trajectory and those of resident and emigrating fish.


Actual Trajectory
Projected Trajectory
Lipid Level F(t)
$F_{a,1}$
$F_{a,2}$
F_i
$F_{a,3}$
$F_{p,1}(t)$
$F_{p,2}(t)$
$F_i(t)$
$F_{p,2}(t)$
Maturing
Non-maturing
Non-maturing
M_1
Nov
Dec
Jan
Feb
Mar
Apr
$t_1 - T_w$
t_1
G_1
Assessment Window
Month
t_2
G_2

(a)


Actual trajectory
Projected trajectory
Size, W(t)
Emigrant
Inherited
Resident
R
July
Aug
Sep
Oct
Nov
Dec
Jan
Feb
Mar
Apr
$t_3 - T_w$
t_3
E
Assessment Window
Time, t
t_2

(b)

Given the relative performance to time t_1, we combine the measure of relative performance with the evolutionary information to construct a projected rate of change of lipid between t_1 and t_2 (Fig. 1a):

$$\frac{dF_p(t)}{dt} = \kappa_1(\dot{F}_a(t), \dot{F}_i(t))\frac{dF_i}{dt} \tag{2}$$

where $F_p(t)$ is the projected trajectory between t_1 and t_2.

The projected rate of change of lipid (Equation 2) and the actual level of lipid at time t_1, $F_a(t_1)$, can be combined for comparison with M_1. For example, projected lipid in April is:

$$F_p(t_2) = F_a(t_1) + \int_{t_1}^{t_2} \frac{dF_p}{dt}dt = F_a(t_1) + \kappa_1(\dot{F}_a(t), \dot{F}_i(t))[F_i(t_2) - F_i(t_1)] \tag{3}$$

The relative importance of current lipid and projected gain in lipid in producing the predicted combination can be varied by weighting the two terms on the far right-hand side of Equation (3):

$$\rho_1 F_a(t_1) + \rho_2 \int_{t_1}^{t_2} \frac{dF_p}{dt}dt = \rho_1 F_a(t_1) + \rho_2 \kappa_1(\dot{F}_a(t), \dot{F}_i(t))[F_i(t_2) - F_i(t_1)] \tag{4}$$

where ρ_1 and ρ_2 are weighting parameters. Equations (3) and (4) are a means to combine actual state and projected rate of change of state. If the combination exceeds M_1, the fish continues maturation at G_1; otherwise, maturation is inhibited.

Next, consider the G_2 developmental switch, which we fix as occurring in April. Because maturing fish stop accumulating resources and begin to lose their appetite in July to August (Kadri *et al.*, 1995, 1996), we consider that the threshold M_2 is activated at this time, which we denote by t_3. Analogues of Equations (1–4) are used to determine the response to the developmental switch G_2, with the assessment window in this case being an interval during April.

Finally, consider the emigration switch (E) which occurs in July to August, at time t_3. The relevant time interval here is from t_3 to t_2 (April) in the next year (the time of actual migration), which we denote by t_2'; the relevant threshold is R and the relevant state is the size (e.g. weight) of the fish. However, care must be taken because fish replace used fat or protein stores with water (Higgins and Talbot, 1985), so that weight may remain nearly constant even though energy value decreases. As before, there are three relevant dynamics: the inherited expected weight trajectory, $W_i(t)$; the actual weight trajectory, $W_a(t)$, during the assessment window; and the projected weight trajectory, $W_p(t)$.

During the assessment window prior to t_3, the fish monitors $\dot{W}_a(t)$ for the rate of change in weight. As with maturation, this leads to an estimate $\kappa_3(\dot{W}_a(t), \dot{W}_i(t))$ of relative performance by comparing $\dot{W}_a(t)$ and $\dot{W}_i(t)$. The estimate of relative performance then leads to a projected rate of change of weight dW_p/dt, as in Equation (2). Finally, weight is projected forward to time t_2, as in Equation (3) or (4). For example, the analogue of Equation (3) is:

$$W_p(t_2') = W_a(t_3) + \kappa_3(\dot{W}_a(t), \dot{W}_i(t))[W_i(t_2') - W_i(t_3)] \tag{5}$$

If this exceeds the threshold R, we assume that at t_3 the fish maintains the developmental path towards smolt metamorphosis during the winter (Fig. 1b).

A growth model for projecting body weight, gonad weight and lipids

A growth model is required for the computation of projected weight at emigration (E switch) or projected amount of lipids at spawning (G_1 and G_2 switches). If $W(t)$ denotes the weight of a fish at

time t and $T(t)$ denotes the temperature at time t, we assume that growth follows the von-Bertalanffy growth formula (Reiss, 1989):

$$\frac{dW}{dt} = q\Phi(T(t))W^{2/3} - \alpha e^{0.071T(t)}W \tag{6}$$

In this equation, growth depends upon the difference between anabolic and catabolic terms (Elliott, 1994). The parameter $q = q_i q_e$ reflects individual (q_i) and environmental (q_e) variation in food

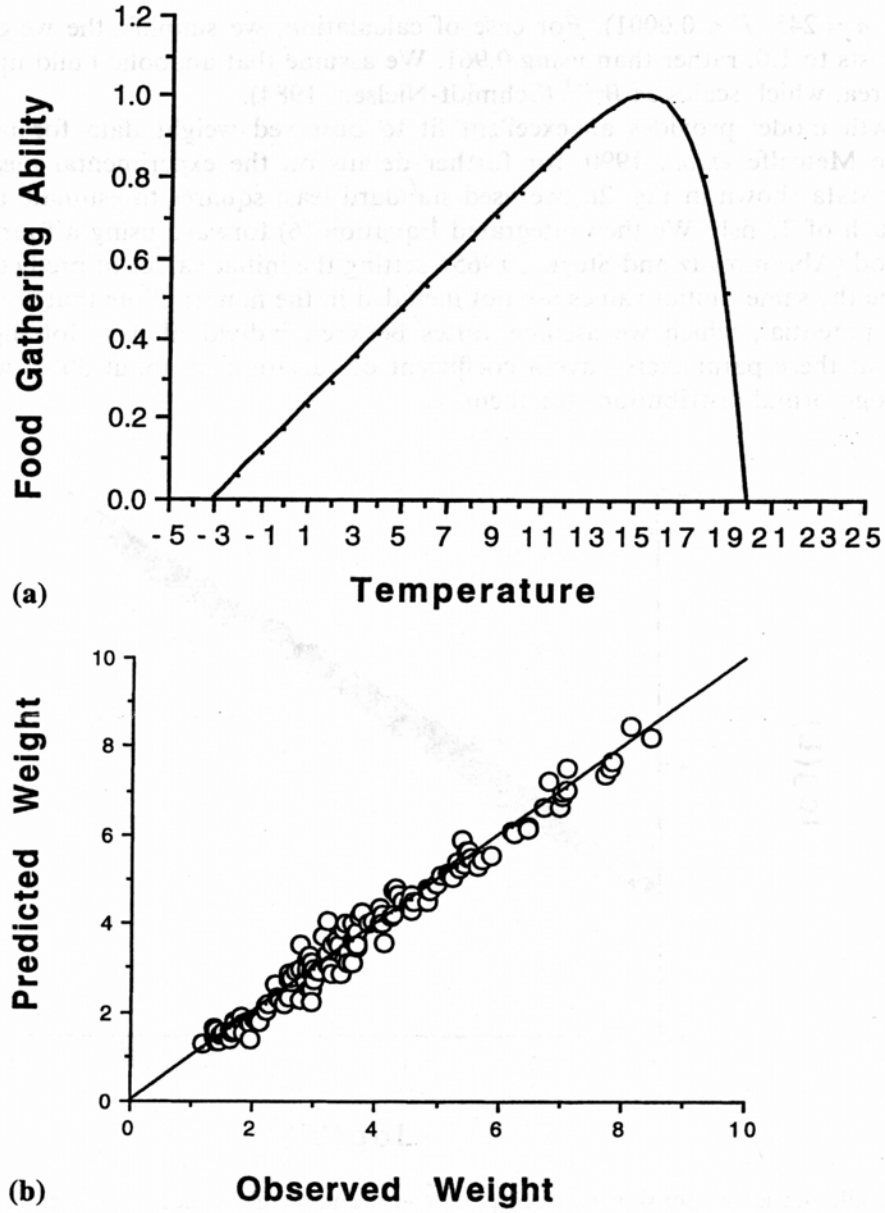


Figure 2. (a) The food-gathering and -processing ability of fish as a function of temperature. (b) The fit between the model given by Equation (6) and the observed weight of individually marked non-anorexic fish between June and November. Data from Metcalfe *et al.* (1990).

finding and processing ability and the parameter α reflects individual variation in metabolic rate. We assume that α and q_i are fixed for an individual fish. Thus, ignoring the temperature dependencies, these predict a fixed asymptotic size.

The function $\Phi(T(t))$ specifies the temperature dependence of the food-gathering and -processing abilities of the fish (Fig. 2a), which we based on the work of Elliott (1994). The assumption that metabolic costs grow exponentially with parameter 0.071 is based on Brett and Groves (1979). C.J. Cutts, N.B. Metcalfe and A.C. Taylor (unpublished) determined the standard metabolic rate (SMR) of Atlantic salmon over a weight range of 1–24 g and found

$$\log(\text{SMR}) = -1.978 + 0.961 \log(W) \tag{7}$$

($r^2 = 0.834$, $n = 245$, $P < 0.0001$). For ease of calculation, we simplify the weight exponent for metabolic costs to 1.0, rather than using 0.961. We assume that anabolic build-up is proportional to surface area, which scales as $W^{2/3}$ (Schmidt-Nielsen, 1984).

This growth model provides an excellent fit to observed weight data for non-anorexic fish (Fig. 2b; see Metcalfe *et al.*, 1990, for further details on the experimental measurements). To generate the data shown in Fig. 2b, we used standard least squares to estimate the parameters q and α for each of 27 fish. We then integrated Equation (6) forward using a fourth-order Runge-Kutta method (Abramowitz and Stegun, 1965), setting the initial values of predicted and observed weights to be the same (initial values are not included in the figure). Note that q/α is a measure of the growth potential, which we assume varies between individual fish. Jobling (1994, p. 178) suggested that these parameters have a coefficient of variation of about 30%; we assume either normal or log-normal distributions for them.

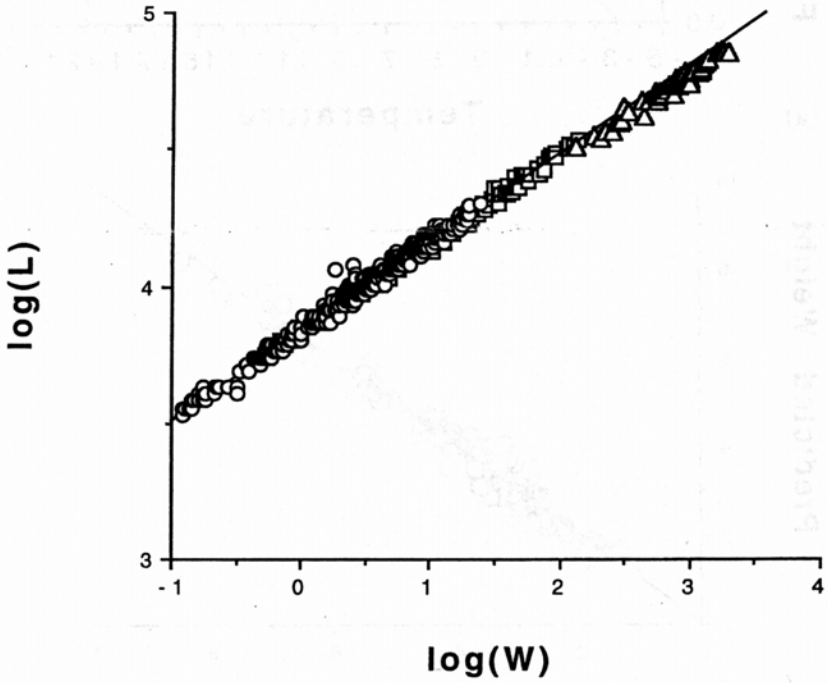


Figure 3. The allometric relationship between parr length and weight is constant over the early life-history stages. Fish that emigrated at age 1 are denoted by squares, those that emigrated at age 2 by circles in their first year and triangles in their second autumn. The allometric relationship is $\log(L) = 3.8463 + 0.31488 \log(W)$; $r^2 = 0.995$.

Length, $L(t)$, at time t is given by an allometric relationship with respect to body weight (Fig. 3) with the constraint that, although fish may lose weight, length cannot decrease. Further details concerning the measurement of length and weight can be found in Metcalfe *et al.* (1990) and Simpson (1992). Length is measured in millimetres and weight in grams. The basic growth model is modified during winter to take account of fish becoming anorexic (see below).

To compute gonad weight from total body weight, we use the data of Sutterlin and MacLean (1984). Based on their work, the gonadal mass $G(W)$ of a ripe mature female of total body weight W (Mangel, 1996) is:

$$G(W) = -0.3255 + 194.45W \tag{8}$$

where gonadal mass is measured in grams and mass W is measured in kilograms.

Fat levels are estimated using a simplified version of the equation presented in Bull *et al.* (1996): fat reserves $F(t)$ in grams at time t are given by

$$F(t) = 0.268 - 0.00683L(t) + 0.121W(t) \tag{9}$$

($r^2 = 0.617$, $P < 0.093$ for length and $P < 0.001$ for weight). Equation (9) allows us to use weight and length of a fish to predict its fat content. The usefulness of a non-destructive measure of fat (Simpson *et al.*, 1992) as a predictor of life histories has been demonstrated by Simpson (1992) and Beddow and Ross (1996).

The feeding rule for overwintering fish determined by a performance threshold

Juvenile salmonids often exhibit a suppressed appetite over winter (Metcalfe *et al.*, 1986; Metcalfe and Thorpe, 1992; Bull *et al.*, 1996; Simpson *et al.*, 1996). We incorporate this into the model by assuming that feeding intensity over the winter (1 October to 1 April) is based on a performance threshold (P). The measure of performance involves the ratio of mortality rate to specific growth rate. The mortality rate, m, for a fish feeding the entire day has size-independent (m_0) and size-dependent (m_1) components:

$$m = m_0 + m_1 W^{-0.37} \tag{10}$$

The form of the size-dependent component is based on McGurk (1996) and can be derived from first principles (Peterson and Wroblewski, 1984). Specific growth rate is given by

$$g(W) = \frac{1}{W}\frac{\mathrm{d}W}{\mathrm{d}t} \tag{11}$$

We assume that a fish feeds the entire day (and grows as determined by Equation 6) if:

$$\frac{m}{g(W)} < P_{\mathrm{E}} \quad \text{(for a fish on the emigration path)}$$

or

$$\frac{m}{g(W)} < P_{\mathrm{R}} \quad \text{(for a fish on the non-emigration path)} \tag{12}$$

The performance thresholds P_{E} and P_{R} are assumed to be genetically determined; their units are %mortality/%growth. We assume that $P_{\mathrm{R}} < P_{\mathrm{E}}$, so that non-emigrating fish feed less over the winter than emigrants (Huntingford *et al.*, 1992). In the extreme, $P_{\mathrm{E}} = \infty$ (so that emigrating fish feed all day) and $P_{\mathrm{R}} = 0$ (so that non-emigrants are in a state of anorexia, feeding at minimal rate).

Bull *et al.* (1996) demonstrated that the overwinter feeding behaviour of non-emigrating fish can be effectively described in terms of a dynamic state variable model (Mangel and Clark, 1988) in which we assume that the fish maximize survival probability while using fat reserves until the water

temperature warms and food becomes more plentiful in the spring. Jobling and Miglavs (1993) envision a similar role of fat in regulating feeding behaviour in Arctic charr, although they report a set point, whereas in our model there is a time-dependent trajectory.

The dynamic state variable model used by Bull *et al.* (1996) generates an optimal trajectory $F^*(t)$ for the use of fat reserves during the winter. If $m/g(W) > P_E$ or P_R, the fish becomes anorexic and feeds just enough to ensure that its fat level $F(t) = F^*(t)$. We let $v(t)$ denote the fraction of the day that it forages during such anorexic periods ($v(t) = 1$ during all other intervals). During the anorexic periods, it does not gain length or weight (indeed, fish may lose weight); mortality occurs, but at a reduced level compared to Equation (10) because the fish forages for only part of the day.

Evaluating the fitness of a suite of developmental thresholds and growth parameters

According to our model, a fish is characterized by a genetically determined set of parameters and thresholds: $\{q_i, \alpha, R, M_1, M_2, P_E, P_R\}$. (The parameters q_i and α determine weight through Equation 6 and lipid levels through Equation 9.) There exists information on the genetic correlation between some of these parameters (e.g. Saxton *et al.*, 1984; Gjerde *et al.*, 1994; Heath *et al.*, 1994), but the model also allows us to treat them flexibly, ranging from being orthogonal to completely correlated. The environment is characterized by $\{q_e, T(t), m_0, m_1\}$.

The fitness associated with a given suite of parameters and thresholds is expected gonadal mass; that is, survival until spawning multiplied by projected gonadal mass at that time. We are thus able to create a fitness surface (Mangel and Ludwig, 1992) associated with a set of parameters and thresholds in a particular environment.

If the fish matures in freshwater, without ever having gone to sea, gonadal mass is related to weight at reproduction by Equation (8). Survival to reproduction at time t_m is given by:

$$\mathscr{S}_m = \exp\left\{-\int_0^{t_m} v(t)(m_0 + m_1 W(t)^{-0.37})\mathrm{d}t\right\} \tag{14}$$

In this case, the fitness of the set of parameters $\{q_i, \alpha, R, M_1, M_2, P_E, P_R\}$ is $\mathscr{S}_m G(W_m)$, where W_m is weight at the time of maturity in freshwater.

Alternatively, when a fish emigrates rather than matures in freshwater, there is an associated expected reproductive success as a returning adult. Mangel (1996) describes a method based on empirical data for computing expected reproductive success $\mathscr{E}(L_s)$ as a function of smolt length L_s. By using this, we avoid the problem of having to reset the maturity thresholds and explicitly modelling the fish when it is in sea water.

Smolt metamorphosis involves a biomechanical transformation in which fish become longer and slimmer at a given weight (Fig. 4). At this time, we are unable to model the parr–smolt transformation, so we use a regression model to calculate the length of a smolting fish at the start of May from its weight. The transformation may be due to the increased somatic cost of maintaining the Na-K ATPase pump (B. McFarlane, personal communication), although recent work shows that the width of the first vertebra differs between parr and smolts (Armstrong and Stewart, 1996), suggesting that this is a true mechanical transformation.

To obtain a wide range of weight values for parr (Fig. 4), we used data on non-anorexic fish in their first year of life, on anorexic fish in their second year of life, and on non-sibling fish. Wankowski and Thorpe (1979) showed that a similar calculation for sibling fish had increasing separation of the regression lines over the winter. For our purposes, the important point is that, over the range of smolt sizes, the lines are essentially parallel.

Assuming that survival to smolting is $\mathscr{S}_e$, the fitness associated with smolting is $\mathscr{S}_e\mathscr{E}(L_s)$. If emigration takes place at time t_e,

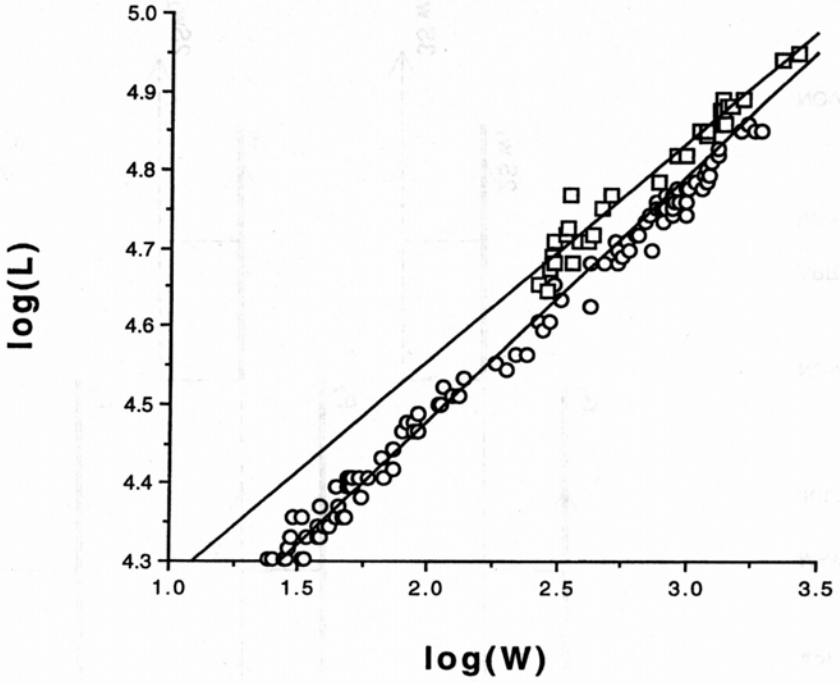


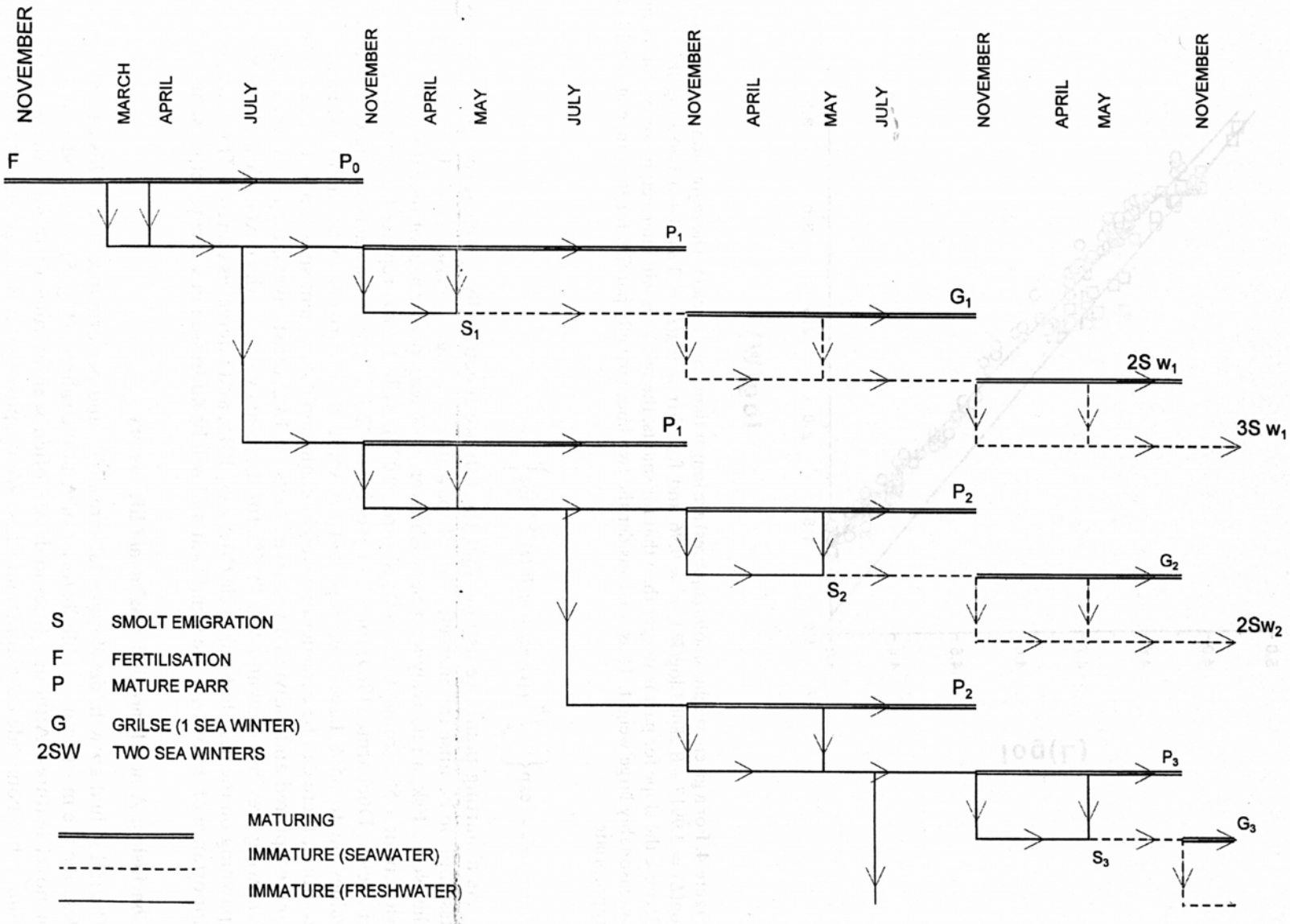
Figure 4. For a given weight, smolts (squares) are longer than parr (circles). The regression equation for smolts is $\log(L) = 3.9917 + 0.28068 \log(W)$, $r^2 = 0.96$ and for parr is $\log(L) = 3.8463 + 0.31488 \log(W)$, $r^2 = 0.995$. Because the slope for parr is steeper than that for smolts, the two lines will intersect; however, this occurs at an unreasonably large weight. Thus, we consider the two lines virtually parallel for the range of weights that are appropriate.

$$\mathscr{S}_e = \exp\left\{-\int_0^{t_e} v(t)(m_0 + m_1 W(t)^{-0.37})\mathrm{d}t\right\} \tag{15}$$

As a starting point, we assume that the fish are functionally semelparous; this is a reasonable assumption because the energetic cost of spawning is so high (Jonsson *et al.*, 1991). For example, on the North Esk, a relatively long Scottish river, the mean percentage of repeat spawners in 1963–70 was about 1.5% (range 0.3–2.4%; Shearer, 1972). Mills (1989) reported a general value of 3–6%. However, Ducharme (1969) found an average of 42.5% repeat spawners in one Canadian river and values up to 35% have been reported for some shorter rivers in Scotland (Menzies, 1915). In addition, there can be considerable weight change in subsequent spawnings. For example, for over 100 recaptured and individually marked kelts on 13 Scottish rivers, the mean wet weight increases following previous spawning was 78.5% for those spawning on the next cycle and 172.9% for those spawning on the next-but-one cycle (Menzies, 1915, and references therein). Thus, the potential and importance of iteroparity are significant and will be addressed in subsequent versions of the model.

Conclusion: A novel, predictive salmonid life history

The individual growth, developmental thresholds and performance thresholds can be combined to describe the most common life histories of Atlantic salmon (Fig. 5). Our model involves a number of novel features. Although thresholds are often seen as rules of thumb, these are not rules of thumb, but bona fide developmental rules. We propose that it is the genetic thresholds, interacting with the environment via growth, that cause the genotype by environment interaction.


NOVEMBER
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P0
P1
S1
G1
2S w1
3S w1
P1
P2
S2
G2
2Sw2
P2
P3
S3
G3
S SMOLT EMIGRATION
F FERTILISATION
P MATURE PARR
G GRILSE (1 SEA WINTER)
2SW TWO SEA WINTERS
MATURING
IMMATURE (SEAWATER)
IMMATURE (FRESHWATER)

Our use of the combination of state (lipid or weight) and rate of change of state avoids the general question and discussion in the literature about singling out whether it is growth rate or size that triggers life-history events (e.g. Berglund, 1992; Bohlin *et al.*, 1993, 1996; Jonsson and Jonsson, 1993; Økland *et al.*, 1993). Based on the model proposed here, we suggest that both are involved. Using the model, we will be able to identify conditions under which it will appear that size is the trigger for life-history events and other conditions under which it will appear that growth rate is the trigger.

Because this is a life-history model, with a computation of fitness, it can be used to understand why certain life-history patterns are virtually never observed. For example, underyearling fish that adopt the anorexic overwinter path are never observed to undergo smolt metamorphosis the following spring; our model can be used to determine the fitness consequence of smolting in such a fish. The prediction is that fish with small thresholds for both R and P_E will have low fitness.

Our model has some similarities with that of Hutchings and Myers (1994), who assumed that maturation in parr is determined by a polygenic threshold based on growth rate and energy reserves, and who provided an indirect test for the existence of a threshold. However, there are a number of differences in approach: their model is based solely on growth rate; they have a single maturation switch (a little bit later than our G_2); and they use fitness defined as the intrinsic rate of increase from the Euler-Lotka equation.

This paper is built on the foundation laid by Thorpe (1986, 1994a), who presented a conceptual model for the life history of salmonids. Thorpe's model was similar to the one presented by Kubo (1980) for masu salmon. The difference between the current and earlier models is that the current one is quantitatively predictive. The ultimate success or failure of this model will be judged in terms of its predictive capability, new understanding derived from it, and new experiments motivated by it.

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Figure 5. Schematic representation of the life-history model based on proximate mechanisms proposed in this paper. We show the timing of the thresholds and the possible life-history routes determined by the responses to those thresholds for fish that mature in freshwater at ages 0, 1 or 2 (P_0, P_1, P_2), that mature after one sea-winter (G) or two sea winters (2SW), and of fish that smolt; subscripts indicate the number of years spent in freshwater prior to smolting. The maturation switches G_1 and G_2 occur in November and April, respectively, and the emigration switch E occurs in August.

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