Natural mortality, growth parameters, and environmental temperature in fishes revisited

David Griffiths and Chris Harrod

Abstract: Pauly (1980. J. Cons. Int. Explor. Mer, **39**: 175–192) showed that natural mortality rates in fish could be predicted from body growth parameters and environmental temperature but found no evidence for ecological or taxonomic influences. Using an updated database and techniques that avoid some of the earlier analytical problems, we confirm Pauly's conclusion that mortality is correlated with growth and temperature. A path model supports the role of ecological effects on mortality. A phylogenetic effect is also apparent: perciform fishes occupy warmer environments than other species, but in the predator-rich reef habitat they suffer much lower mortality rates. Species that are cryptic or hide in burrows or have morphological defences against predators show the lowest mortality rates.

Résumé : Pauly (1980. J. Cons. Int. Explor. Mer, **39**: 175–192) a montré que les taux de mortalité naturelle chez les poissons pouvaient être prédits à partir des paramètres de croissance corporels et de la température du milieu, mais il n'a trouvé aucun indice de l'influence des caractères écologiques et taxonomiques. À l'aide d'une banque de données renouvelée et de techniques qui permettent d'éviter quelques-uns des problèmes analytiques antérieurs, nous confirmons la conclusion de Pauly qu'il y a une corrélation entre la mortalité, d'une part, et la croissance et la température, d'autre part. Un modèle de trajectoire laisse croire à l'existence d'effets écologiques sur la mortalité. Un effet phylogénétique est aussi apparent: les poissons perciformes vivent dans des milieux plus chauds que les autres espèces; cependant, dans l'habitat de récif qui est riche en prédateurs, ils subissent des taux de mortalité beaucoup moindres. Les espèces qui sont camouflées, qui se cachent dans des gîtes ou qui possèdent des structures morphologiques de défense contre les prédateurs ont les taux de mortalité les plus bas.

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Introduction

Mortality rates are affected by intrinsic and extrinsic tradeoffs. For example, the asymptotic size (W) and growth coefficient (K) of the von Bertalanffy growth equation are negatively correlated because of tradeoffs among energy allocation to growth, maintenance, and reproduction (Roff 1992). There are a number of possible extrinsic mortality agents, both abiotic and biotic. In many aquatic environments, physical and chemical factors are relatively constant (Cyr and Cyr 2003), but biotic factors, particularly predation, are potentially important and variable. Aquatic animals at all trophic levels experience predation risk – food reward tradeoffs (Werner and Gilliam 1984; Werner and Anholt 1993) because food-rich habitats are usually riskier. These intrinsic and extrinsic tradeoffs are at least partially linked. For example, an increased allocation to reproduction will reduce growth rates and, because aquatic mortality rates are strongly size dependent (Peterson and Wroblewski 1984; McGurk 1986), increase mortality rates.

In a widely cited paper, Pauly (1980) analysed the relationships among (adult) natural mortality, growth parameters, and mean water temperature for 175 fish stocks belonging to 113 species (not 84 as stated in the abstract to that paper). He showed that the instantaneous natural mortality rate (M) was well predicted from W and K and water temperature (T). Jensen (2001) reanalysed Pauly's data and suggested that stocks could be divided into two groups, from temperate and warmer waters. Pauly considered various explanations, both direct and indirect, for the surprising role of temperature on mortality rates. He favoured a causal (physiological) effect whereby fishes in warmer waters, because of their higher metabolic rates, need to feed more frequently. Consequently, prey species are exposed more frequently to predators, which in turn impose greater mortality. He noted that with two possible exceptions, mortality rates did not vary with taxonomy or ecology. Since Pauly's paper, additional data have accumulated (available in FishBase 2004), but there has been no subsequent attempt to explore further the roles of environmental temperature, taxonomy, and ecology in affecting mortality

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Table 1. Multiple regression results for natural mortality (M) as a function of size (W), growth coefficient (K), and water temperature (T) for (a) stocks and species treated as independent data points and (b) phylogenetically independent contrasts.

(a) Stocks and species treated independently.

	Stocks (n = 458, R^2 = 0.706)				Species (n = 173, R^2 = 0.771)			
Effect	Coefficient±SE	Standardized coefficient	t	P	Coefficient±SE	Standardized coefficient	t	P
Intercept	0.034±0.054	—	0.64	0.53	0.102±0.086	—	1.19	0.24
log W	–0.043±0.014	–0.12	3.06	0.002	–0.066±0.020	–0.18	3.25	0.001
log K	0.833±0.045	0.72	18.43	<0.001	0.814±0.067	0.70	12.09	<0.001
log T	0.189±0.040	0.13	4.68	<0.001	0.190±0.065	0.12	2.95	0.004

(b) Phylogenetically independent (n = 68, R^2 = 0.639).

Effect	Coefficient±SE	Standardized coefficient	t	P
log W	–0.084±0.035	–0.25	2.38	0.002
log K	0.625±0.119	0.59	5.23	<0.001
log T	0.150±0.159	0.08	0.94	0.349

rates. Fish exhibit diverse morphological (e.g., spines) and behavioural (e.g., schooling) adaptations to reduce the risk of being eaten, and one would expect these to be reflected in variation in mortality rates. Similarly some environments (e.g., pelagic habitats, tropical reefs) are potentially more hazardous than others (Moyle and Cech 2000). Here we explore the roles of temperature, taxonomy, and ecology on mortality rates using the more extensive data in FishBase (2004).

Central to Pauly's analysis is the assumption that the data points and variables analysed are independent. Jensen (2001) reanalysed Pauly's data because potentially high levels of multicollinearity rendered multiple linear regression suspect, but he came to similar conclusions. However, his reanalysis made no allowance for phylogenetic or multistock non-independence; some species were represented by multiple measurements (e.g., nine for *Gadus morhua*), thereby inflating the number of degrees of freedom, while no allowance was made for phylogenetic relatedness (e.g., 1–11 species/family). Consequently it is not clear to what extent the published relationships are supported statistically, a situation that we attempt to remedy.

Materials and methods

Data

Information from FishBase 2004 on the mortality rates of 458 fish stocks belonging to 173 species was compiled, along with relevant growth and environmental information (Binohlan and Pauly 2000; Froese 2000; Froese et al. 2000; Palomares 2000; Palomares and Pauly 2000; Preikshot et al. 2000). FishBase provides estimates of M derived in a variety of ways. We used only independently estimated values obtained from, for example, plots of total mortality against effort in exploited populations or catch curves in unexploited populations, and not those predicted from empirical relationships for other species, such as was done in Hoenig (1983). Mean values were used whenever there was more than one stock per species.

The Brody growth coefficient K is sometimes referred to as a growth rate but it has dimensions of time^{-1}. Pauly (1994) has argued that the growth performance index (Ø =

Table 2. Comparison of regression models for predicting instantaneous natural mortality rates (M) for species data using Akaike's information criterion (AIC).

Model	AIC$_c$	ΔAIC$_c$	Akaike weights	R^2
WKT	–543.46	0	0.946	0.771
WK	–536.89	6.6	0.035	0.759
KT	–535.10	8.4	0.014	0.757
WT	–437.71	105.8	1.028×10^{-23}	0.573
W	–404.44	139.0	6.137×10^{-31}	0.476
K	–532.63	10.8	0.004	0.750
T	–315.90	227.6	3.657×10^{-50}	0.125

**Note:** All variables were log-transformed, and all regressions were statistically significant ($P < 0.001$). W, size; K, growth coefficient; T, water temperature.

log$K$ + 2/3 log$W$ (Froese and Pauly 2000)), derived from body growth within stocks, with the more usual dimensions of mass·time^{-1} and with a clearer biological meaning (it is the intercept in log$K$, log$W$ regressions), is a more appropriate measure. Ø values were not available in FishBase so we substituted mean K and W values in the above equation to estimate Ø for each species.
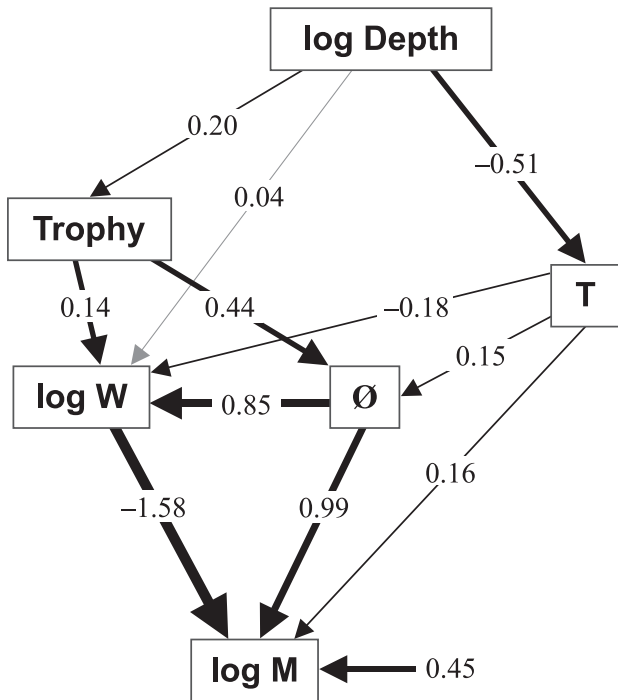
Growth rates vary with food supply, which in turn will vary with trophic level and availability. Primary production is largely restricted to surface waters, and one would expect food availability to decline with increasing depth (Moyle and Cech 2000). FishBase reports the upper, lower, and "common" depths at which many species occur. All three measures are correlated, but most data were available for the lower depth limit, and we used this (log-transformed) as an indicator of food availability. Water temperature is also a function of depth. The Foodtroph index in FishBase was used as an measure of trophic level: Foodtroph is defined as (1 + mean trophic level of the food items), with autotrophs, for example, being assigned to trophic level 1.

Information on the habitats occupied by each species was also obtained from FishBase 2004. Demersal species live and (or) feed on or near the bottom, benthopelagic species occupy a similar zone to demersal species but also occur in midwater, while pelagic species occur mainly in the water

Fig. 1. Path diagrams for the effect of size (*W*), growth coefficient (*K*), and environmental temperature (*T*) on natural mortality rates (*M*) and significant coefficients for (*a*) species and (*b*) phylogenetic data.
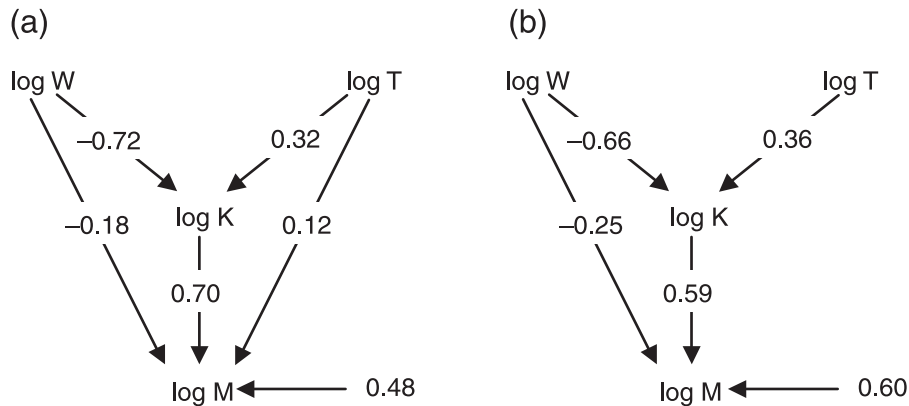
Fig. 2. Path analysis of a model of the effect of size (*W*), growth performance (Ø), untransformed temperature (*T*), trophic level, and maximum depth of occurrence on mortality rates (*M*). The model tested is shown by arrows; nonsignificant path is shown in gray. The path coefficients were estimated by RAMONA (Browne 2004). For clarity of presentation, the path from the residual variable is shown only for *M*. To aid interpretation, the width of the lines is approximately proportional to the size of the path coefficients.

Table 3. A summary of the direct and indirect effects of the predictor variables on mortality rates, as estimated by multiple regression and correlation (*n* = 131).

Variable	Direct	Indirect	Total
W	−1.59	0.88	−0.71
Ø	1.00	−1.41	−0.40
T	0.15	0.28	0.43
Trophic level	−0.00	−0.40	−0.41
Depth	−0.02	−0.37	−0.39

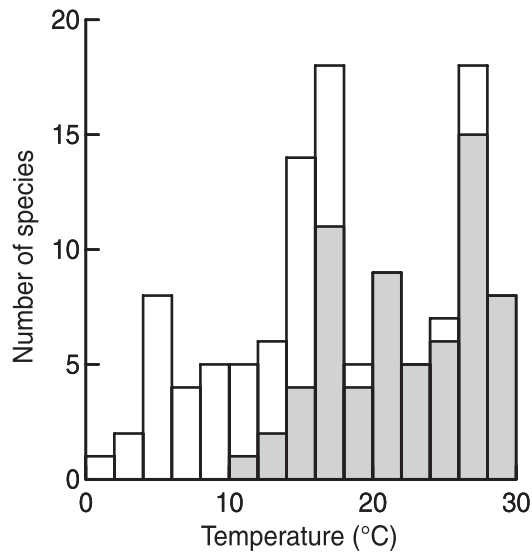**Note:** *W*, size; Ø, growth performance; *T*, untransformed temperature.

strengths of the direct and indirect linkages between any two variables) from standardized regression coefficients (Sokal and Rohlf 1995). In a similar but more complicated analysis, we fitted a path model using the RAMONA procedure in SYSTAT 11.0 (Browne 2004). Each dependent variable has a path coefficient representing the unexplained variation and is referred to here as the residual variable.

We checked if collinearity was a problem with the data by examination of condition indices and tolerance statistics in multiple linear regression and by ridge regression. Since weight and length are expected to be and are strongly correlated ($r = 0.975$, $n = 173$, $P < 0.001$), we, unlike Jensen (2001), used only one size measure per analysis and only present results where *W* was used. All variables were $\log_{10}$-transformed to be consistent with Pauly's analysis and to normalize or improve the normality and linearity of the data; all errors are standard errors. Lowercase *t* in the tables refers to the *t* statistic.

The "best" model (i.e., the one that provides the best fit for the fewest predictor variables) was determined using Akaike's information criterion (AIC) (Burnham and Anderson 1998). The small sample variant (AIC_c) was used because the ratio of number of observations to number of estimated parameters was appreciably below 40. Model fits were then compared with ΔAIC_c and Akaike weights (the relative likelihood of the models) (Burnham and Anderson 1998).

To examine the effects of relatedness (i.e., nonindependence of species values (Harvey and Pagel 1991)), we performed a comparative analysis by independent contrasts

column and do not feed on benthic organisms. Reef-associated species live and (or) feed on reefs. All these species normally occupy the top 200 m of water. Two other habitat categories, occupying deeper water, were infrequently represented in the data (*n* = 1, 6) and were omitted when investigating habitat effects.

Statistical analyses

To estimate the direct and indirect effects of *W*, *K*, and *T* on mortality rates, we calculated path coefficients (i.e., the

Fig. 3. The habitat temperatures experienced by perciform (shaded) and nonperciform (open) marine fishes.

Fig. 4. Mean environmental temperatures (±1 SE) experienced by perciform (●) and nonperciform (△) species in different habitats: bp, benthopelagic; d, demersal; p, pelagic; r, reef-associated.

using the CAIC software (Purvis and Rambaut 1995) and the continuous variable (Crunch) option. Contrasts in the dependent variable were regressed, through the origin, against contrasts in the independent variables. There is no well-established phylogeny for fish, so we used the taxonomy in Froese and Pauly (2000); given the wide range of taxa in the data set and the relatively small numbers of species per genus for most genera, we believe that this procedure will not bias the results appreciably.

Results

T, W, and K all contributed significantly to predicting M. The standardized coefficients are similar for stock and species data (Table 1a) and show K to be the dominant predictor variable. Despite significant correlations between K and W (stocks: –0.706, $n = 485$, $P < 0.001$; species: –0.717, $n = 175$, $P < 0.001$), condition indices and tolerance statistics provided no indication of collinearity in the regressions. Similarly, ridge regression provided no evidence that inclusion of both K and W affected the regression coefficients to any appreciable degree.

Comparison of the full suite of models using AIC shows that the full (WKT) model was better than any of the others in predicting M (Table 2); it was 0.946/0.035 = 27 times more likely to be better than the next best fitting (WK) model.

Path analysis of species data (Fig. 1a) showed that the direct effects of W and of T on M were much less than the indirect effects (path coefficients W: –0.18, –0.50; T: 0.12, 0.22, respectively). The phylogenetically independent data strengthened the role of W as a significant predictor of mortality rates (Table 1b; Fig. 1b), and the direct and indirect effects are similar (–0.25, –0.39). However, the indirect effect of T was much stronger than the direct effect (0.21, 0.08, respectively), and the total temperature effect was not significant. The total effects for the species and phylogenetic data
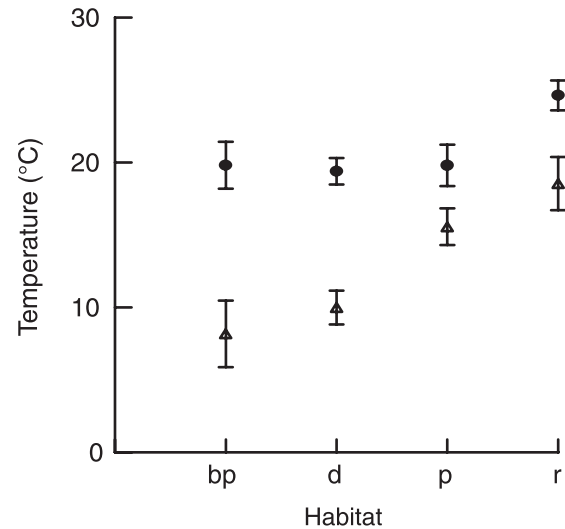
are 0.68 and 0.64 for W and 0.34 and 0.29 for T, respectively.

Food supply was assumed to affect size and growth rates and consequently influence mortality rates. In turn, food supply should vary with trophic level and with water depth. Water depth should also influence mortality rates by its effect on water temperature. We tested a model incorporating these effects and using untransformed temperature (T, since this was closer to normality in its distribution) by path analysis (Fig. 2). Although the direct effect of depth on W was not significant, the model provided an excellent fit to the data (root mean square error of approximation = 0.03). Size and T had large effects on M, but the indirect effects of Ø, trophic level, T, and depth were stronger than the direct effects (Table 3). Similar results were obtained when the analysis was restricted to marine species.

The conflicting temperature effects in the species and phylogenetically independent contrast analyses could reflect heterogeneity in the thermal preferences of different lineages. Bimodality is apparent in the habitat temperatures experienced (Fig. 3). Some of this bimodality is because of differences between taxa (perciform vs. nonperciform fishes), but note that bimodality is still apparent within perciform fishes, the taxon that contributes most to the overall bimodality. W, K, and T contributed significantly to predicting M in perciform fishes ($R^2 = 0.80$, $n = 95$), but in nonperciforms ($R^2 = 0.76$, $n = 78$), T was not a significant predictor. The mean temperatures that these taxa occupy are significantly different (least squares adjusted means all salinities: perciforms, 21.1 ± 0.62 °C, $n = 132$; nonperciforms, 13.7 ± 0.66 °C, $n = 115$ ($F_{[1,245]} = 67.71$, $P < 0.001$; marine species: 21.1 ± 0.63 °C, $n = 111$; 12.0 ± 0.76 °C, $n = 75$, respectively ($F_{[1,184]} = 73.01$, $P < 0.001$)).

The environmental temperatures experienced by marine species (i.e., excluding freshwater and estuarine species) varied across taxonomic group and habitat, and there was a marginally significant interaction term ($F_{[3,171]} = 2.38$, $P = 0.07$). In each habitat, perciforms occupied warmer environ-

Table 4. (*a*) General linear model showing the contributions of size (*W*), growth performance (Ø), temperature (*T*), habitat, and taxon to mortality rate and (*b*) a summary of the mortality regression results for perciform and nonperciform fishes.

(*a*) General linear model.

Source	df	Mean square	F	P
W	1	8.105	219.345	<0.001
Ø	1	2.633	71.246	<0.001
T	1	0.244	6.593	0.011
Habitat	3	0.117	3.16	0.027
Taxon	1	0.265	7.162	0.008
Habitat × Taxon	3	0.105	2.841	0.041
T × Taxon	1	0.264	7.146	0.009
Error	124	0.037	—	—

(*b*) Mortality regression.

Taxon	Habitat	Intercept	*W*	Ø	*t*	*n*	R^2
Perciforms	All	0.148	–0.616	0.780	0.014	83	0.81
Nonperciforms*	Benthic–deep	0.230	–0.499	0.567	—	57	0.80
Nonperciforms*	Pelagic	0.367	–0.499	0.567	—	57	0.80
Nonperciforms*	Reef	0.586	–0.499	0.567	—	57	0.80

*The equations for nonperciforms are for three habitat categories, which were coded as dummy variables.

ments than nonperciform fishes ($F_{[1,171]} = 54.17$, $P < 0.001$) (Fig. 4). The temperatures decreased with increasing depth (pelagic, p; demersal, d; benthopelagic, bp) for nonperciform fishes but were independent of depth for perciforms ($F_{[1,64]} = 3.90$, $P = 0.05$; $F_{[1,107]} = 0.01$, $P = 0.93$); reef-associated (r) fish in each taxon lived in warmer waters than in the other habitats ($F_{[1,64]} = 9.74$, $P < 0.01$; $F_{[1,107]} = 17.85$, $P < 0.001$).

An a priori model of mortality rates using *W*, Ø, untransformed *T*, and the taxonomic and habitat categories (Table 4*a*) explained 81% of the variance (marine species only, *n* = 136). The inclusion of taxonomic and habitat variables resulted in only a small increase in the variation explained (0.78 to 0.81). However, random sorting of the taxonomic variable supported the conclusion that this was a significant contributor to mortality rates. Mortality rates differed across habitats in nonperciform fishes ($F_{[3,46]} = 3.53$, $P < 0.05$) but not in perciforms ($F_{[3,76]} = 0.30$) (Fig. 5; Table 4*b*). Perciforms suffered higher mortality rates when living in demersal habitats, but nonperciforms had higher mortality rates when associated with reefs ($F_{[1,124]} = 3.79$, $P = 0.05$; $F_{[1,124]} = 4.81$, $P < 0.05$, respectively).

These species belonged to 48 families. Least squares adjusted means were calculated for families, with *W*, Ø, and untransformed *T* as covariates. 15 of the 18 species belonging to the 11 families with the lowest mortality rates showed adaptations that would be expected to reduce their vulnerability to predators; 11 species are flatfish (pleuronectiforms), which are cryptic and lie at least partly buried in the substrate, two occupy burrows (cheilodactylid, malacanthid), one is heavily armoured (gasterosteid), and one is armed with poisonous spines (sebastid). There was no obvious reason why the remaining three species (acipenserid, lethrinid, triakid) should have low mortality rates. Pauly (1980) noted that 25 clupeid stocks (belonging to 14 species) tended to have *M* values less than those predicted by his equation (using *W*, *K*, *T* as predictor variables), whereas engraulids,

Fig. 5. Least squares adjusted instantaneous mean mortality rates (*M*) (±1 SE) of perciform (●) and nonperciform (△) species in different habitats: bp, benthopelagic; d, demersal; p, pelagic; r, reef-associated.
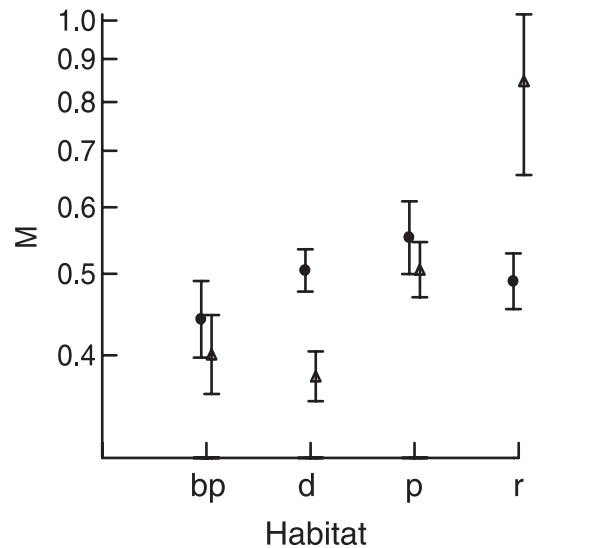
which also strongly school, did not. We found no significant difference in *M* among the 14 clupeid and 8 engraulid species ($F_{[1,17]} = 0.27$; *W*, *K*, *T* as covariates).

Discussion

Stock and species data regressions using *W*, *K*, and *T* to predict mortality rates showed good agreement. Our standardized coefficients for stocks were also similar to those obtained by Pauly (1980, his table 6), although the temperature effect was stronger for his smaller data set. There was agreement also in the importance of *W* and *K* for phylogen-

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etically independent contrasts, but the direct effect of temperature was no longer significant.

Savage et al. (2004) tested the metabolic theory of ecology with Pauly's (1980) data and found broad agreement; the slope of the size-corrected mortality rate and temperature gave an activation energy somewhat below the predicted value (–0.45 eV vs. 0.6–0.7 eV), but the slope of the temperature-corrected mortality rate as a function of body mass (–0.23) was very close to the predicted value of –0.25. Using species rather than stock data gives the expected slope (–0.26), but the activation energy of 0.33 eV is only half that expected. Charnov and Gillooly (2004), using a model in which temperature is assumed to affect only growth, argue that the temperature effect on mortality could be indirect, via life history optimization, in line with the analysis presented here.

Depth in the path model is a surrogate for both temperature and food supply. We expected mean trophic level to increase with depth if only because of the loss of herbivorous species. In deep waters, productivity is limited, and coupled with the low temperatures, this should favour large, slowly growing species (Moyle and Cech 2000). We also expected positive correlations between trophic level and body size (because piscivorous fish are usually larger than their prey) and between trophic level and growth performance (because piscivores are nutritionally more similar to their prey than lower trophic levels). All, except the direct size–depth relationship, were found.

Pauly (1980) considered various direct and indirect effects of T on M. He favoured the notion that fish in warm waters are more likely to encounter predators that are hungry because of higher metabolic rates at higher temperatures. The results presented here are at least partly consistent with this possibility. Mean mortality rates decline from 0.76 (T = 21.6 °C) in reef to 0.59 (T = 17.7 °C) in pelagic to 0.42 in demersal (T = 14.7 °C) and benthopelagic habitats (T = 14.0 °C). However, the significant effect of taxonomic group on mortality rates suggests that indirect effects are also important. We expected predation risk to differ across habitats, dependent on the number of predators and the availability of hiding places. The high productivity relative to surrounding waters, the large number of predatory species, and the many defensive mechanisms found suggest that piscivory is intense on tropical reefs (Hixon 1991; Moyle and Cech 2000), despite there being many potential refuges from predators. In contrast, pelagic species live in an environment lacking hiding places, but the frequency of schooling in these species suggests that predation is also an important factor there. Excluding temperature and its interaction with taxon from the model in Table 4 significantly reduced the variance explained (R^2 = 0.79, ΔAIC_c = 9.94 for the reduced model), suggesting that both habitat and temperature effects are important. This analysis shows that mortality rates are correlated with taxon and habitat in a way that reflects a more direct role for ecology than implied by the physiological effects of temperature. The low mortality rates for species that are hidden or morphologically protected from predators supports this interpretation.

Perciforms occupy substantially warmer habitats than nonperciform species, and with the exception of reef species, they tend to suffer higher mortality rates, consistent with Pauly's explanation. Moyle and Cech (2000) attribute the success of perciforms as a group to a number of factors, including the presence of spines and high manoeuvrability. They suggest that these features enable the fish to occupy riskier, more exposed, habitats (e.g., shallow or surface waters) than other taxa; these will be warmer than deeper or more offshore waters. However, if such defences were highly effective, one would expect lower mortality rates in this group than in nonperciforms. Perciforms suffer higher mortality rates than other fishes in demersal habitats, but the markedly higher temperatures they experience suggest that they occupy rather different demersal habitats from the nonperciforms analysed. The much higher mortality rates experienced by nonperciforms in what is probably the most dangerous habitat (i.e., tropical reef) is consistent with an effective defensive role for perciform spines.

While Pauly (1980) and the equations generated here explain much of the variance in mortality rates, they are poor predictors; for example, the median and 90th percentile jackknife prediction errors for perciform fish, calculated using the procedure in Pascual and Iribarne (1993), were 20% and 76%, respectively. Pauly's stock-based equation explains almost as much of the variance as the species versions generated here; the original conclusions were not affected by the nonindependence of many of the data points. However, both ecology and taxonomy do influence fish mortality rates, although taxonomy does not necessarily closely map to phylogeny (Harvey and Pagel 1991). While phylogeny has been taken into account in some comparative analyses of marine fish biology (e.g., Block and Finnerty 1994), in many others it has not, with the possibility that valuable insights will be lost. Such analyses require a well-established phylogeny, something that is lacking for many fish groups.

Acknowledgements

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