Exploring a first-principles-based model for zooplankton respiration

Ted T. Packard and May Gómez


Oxygen consumption (R) is caused by the respiratory electron transfer system (ETS), not biomass. ETS is ubiquitous in zooplankton, determines the level of potential respiration (RΦ), and is the enzyme system that ultimately oxidizes the products of food digestion, makes ATP, and consumes O2. Current respiration hypotheses are based on allometric relationships between R and biomass. The most accepted version at constant temperature (T) is R = i0M0.75, where i0 is a constant. We argue that, for zooplankton, a Φ-based, O2-consuming algorithm is more consistent with the cause of respiration. Our point: although biomass is related to respiration, the first-principles cause of respiration is ETS, because it controls O2 consumption. Biomass itself is indirectly related to respiration, because it packages the ETS. Consequently, we propose bypassing the packaging and modelling respiration from ETS and hence Φ. This Φ is regulated by T, according to Arrhenius theory, and by specific reactants (S) that sustain the redox reactions of O2 consumption, according to Michaelis–Menten kinetics. Our model not only describes respiration over a large range of body sizes but also explains and accurately predicts respiration on short timescales. At constant temperature, our model takes the form: R = ΦS/(Km + S), where Ea is the Arrhenius activation energy, Rg the gas constant, and Kmp the Michaelis–Menten constant.

Keywords: biomass, electron transfer system, Kleiber’s law, metabolic theory of ecology, metabolism, respiration.

Received 29 June 2007; accepted 6 December 2007; advance access publication 13 February 2008.

Introduction

A first-principles model of respiration should be based mechanistically on the biochemical basis of respiration. Currently, no such model for zooplankton exists, although the metabolic theory of ecology (MTE) by Brown et al. (2004) and the enzyme-kinetic-based models of Packard et al. (1996a,b, 2004) and Roy and Packard (2001) purport to be. Here, we examine the suitability of these two types of metabolic model for predicting zooplankton respiration in different physiological states and on a short timescale. We find the MTE applicable on large scales and the enzyme kinetic approach on small scales.

During the past 10 years, a series of studies has put forth a MTE based on the allometric equation of Kleiber (1932; West et al., 1997, 2000; Enquist et al., 1998; Brown et al., 2004). These studies argue that metabolism is determined by biomass (M), temperature (T), and the flux of elemental materials (i0) through an organism (Brown et al., 2004). They invoke a similarity of the fractal scaling nature of plant and animal circulatory and distribution networks, along with metabolic thermodynamics and kinetics, to explain the widespread applicability of the allometric equation. The mathematical model of the MTE takes the form

\[ R = i_0 M^{3/4} e^{-E_a/kT} \]  

(1)

The biomass part is from Kleiber’s law, R = aM^{3/4} (Kleiber, 1932; Whitfield, 2006), where a = R when M = 1. With an uncertainty of two orders of magnitude, Kleiber’s law holds true over 20 orders of magnitude in biomass (Hochachka and Somero, 2002; Whitfield, 2006). The relative ease with which biomass can be measured, as well as the fact that biomass is a basic property of living systems, explain why Kleiber’s law is so well entrenched in biological thinking and why the MTE is so appealing to ecologists.

Temperature dependence in Equation (1) is based on the atomic-scale Boltzmann factor, e^{-E_a/kT}, where Ea is the Arrhenius energy of activation in electron-volts, k the atomic-scale Boltzmann constant (\( 1.38 \times 10^{-23} \) J atom^{-1} K^{-1} or 0.33 \( 10^{-23}\) cal atom^{-1} K^{-1}), and T is calculated in degrees Kelvin (K). For the stoichiometric factor or resource availability, the MTE uses a constant, i0 (Brown et al., 2004). This MTE equation has been applied to secondary production, respiration (Gillooly et al., 2001), growth, developmental time (Gillooly et al., 2002), etc., in both plants and animals. Recently, it has been applied to the calculation of both phytoplankton productivity and total plankton respiration in the world ocean to demonstrate that respiration will exceed photosynthesis in both the oligotrophic as well as the warming ocean waters (López-Urrutia et al., 2006). On large scales, the MTE provides a basis for improving ocean models.

The models based on enzyme-kinetics of metabolism argue that, at the cellular level, each metabolic process is controlled by the maximum velocity (\( V_{max} \)) of the enzymatic reaction that controls the process (Packard et al., 2004), the temperature, and substrate availability (S). In the case of respiratory oxygen...
consumption ($R_{O_2}$) in zooplankton, this approach asks questions about the biochemistry of respiration and builds the logic of the model on the answers. At constant temperature, what is the direct cause of $R_{O_2}$, and what controls it? Cytochrome oxidase at the end of the electron transport chain is the enzyme that reduces $O_2$ to $H_2O$. However, this $O_2$ reduction rate is controlled at the other end of the electron transport chain by the dehydrogenases that oxidize NADH, succinate, and NADPH and that, in turn, are produced by the Krebs cycle and other metabolic pathways. Packard et al. (1996a, b) argue that it is the $V_{max}$ of these enzymes and the availability of their substrates ($S$) that, according to the theory of Michaelis–Menten, control the in vivo dehydrogenase activity, the electron transfer system (ETS) activity, and potential respiration. The $V_{max}$ of the dehydrogenases and the ETS is largely controlled by $M$, but the substrates are controlled by the organism’s nutritional and physiological state.

The equation, for the enzyme-kinetic-based model for any oxygen consumption rate ($R_I$) at temperature $T_I$ as a function of $V_{max}$ measured at another temperature ($T_0$), takes the form

$$ R_I = \frac{S(V_{max} e^{-\lambda})}{K_m + S} \tag{2} $$

$V_{max}$ is the potential rate of the process (in the same units as the physiological rate, $R_I$) and $\lambda = E_a(1/T_I - 1/T_0)/R_0$, an exponent derived from the Arrhenius equation ($R_0 = Ae^{E_a/K_BT_0}$ and $R_I = Ae^{-E_a/K_BT_I}$), where $A$ is the Arrhenius frequency factor (a reaction constant), $E_a$ the molar-scale Arrhenius energy of activation ($\approx 0.27 \text{ kJ} \text{ mol}^{-1} \text{ K}^{-1}$ or 15 kcal mol$^{-1}$ K$^{-1}$; Packard et al., 1971, 1975), $R_0$ the molar-scale gas constant (8.31 J mol$^{-1}$ K$^{-1}$ or 1.987 cal mol$^{-1}$ K$^{-1}$), and $T_0$ and $T_I$ the temperatures (K) of the measured potential rate and the predicted rate, respectively, between 0°C and the temperature optimum of $R_{O_2}$. The term $e^{-\lambda}$ represents the fraction of the measured $\Phi_0$ that is available to function at $T_I$. If $T_I > T_0$ then $\Phi_I > \Phi_0$ and $R_I > R_0$ if $T_I < T_0$ then $\Phi_I < \Phi_0$ and $R_I < R_0$ as observed in nature, up to the optimum temperature for the organism’s $R_{O_2}$. Because $A$ is a constant for a particular reaction, it is eliminated when solving $R_I = f(R_0)$, i.e., $R_I = R_0 e^{-E_a(1/T_I-1/T_0)/R_0}$. The concentration of the substrate, $S$, represents the reactant of the enzyme reaction controlling $R_{O_2}$. The constant, $K_m$, for a single-substrate reaction ($S\rightarrow P$), where $P$ is the product of the reaction, is the Michaelis–Menten constant. In the case of a bisubstrate reaction as in the ETS (Savenkov et al., 1995; Gómez et al., 1996), ($S_1 + S_2 \rightarrow P_1 + P_2$), $S$ becomes $[S_1S_2]$ and $K_m$ becomes $K_m = (K_{S_1}) (K_{S_2}) / (K_{S_1} + (K_{S_2}) + [S_1] + [S_2])$, where $K_{S_1}$ and $K_{S_2}$ are the Michaelis–Menten constants for $S_1$ and $S_2$, and $K_{mS}$ the dissociation constant for the enzyme-S complex (Packard et al., 1996a, b, 2004). Note that, if $T_0$ and $T_I$ are equal, $\lambda$ becomes 0, and $e^{-E_a(1/T_I-1/T_0)/R_0}$ becomes 1, $R_I = R_0$, and Equation (2) simplifies to

$$ R_I = R_0 = \frac{S V_{max}}{K_m + S} \tag{3} $$

Note the different between $e^{-E_a(1/T_I-1/T_0)/R_0}$ in Equation (2) and $e^{-E_a/kT}$ in Equation (1). Both exponential distribution functions trace back to Maxwell in 1859, but Maxwell, Boltzmann, van’t Hoff, and Arrhenius used them for different purposes. It was Arrhenius who used this function to predict the temperature dependence of biological processes, as well as chemical and biochemical reactions (Arrhenius, 1915). At a numerical level, $R_0$ in Equation (2) is greater than $k$ in Equation (1) by 23 orders of magnitude. This is because $k = R_g/N$, where $N = $ Avogadro’s number, 6.022 $\times 10^{23}$ atoms mol$^{-1}$. This would not be a problem if $E_a$ values were reported in molecular units, but historically biologists, biochemists, and chemists, following Arrhenius (1889, 1915), have reported their measurements of $E_a$ in molar units (Martínez and Estrada, 1992; Treacy et al., 1997; Oxtoby et al., 1999; Ramirez et al., 2006; Angelova et al., 2007).

Previously, this enzyme-kinetic-based model predicted respiratory CO$_2$ production rates in bacteria from $V_{max}$ measurements of isocitrate dehydrogenase activity, a proxy for potential respiratory CO$_2$ production (Figure 1a, and Roy and Packard, 2001). About the same time, it was applied to respiratory oxygen consumption in bacteria, as we plan to do here for zooplankton. The model predicted rates in bacteria from $V_{max}$ measurements of respiratory ETS activity, kinetic constants from the literature, and modelled time courses of the two main ETS electron donors (reactants), NADH and NADPH (Figure 1a, and Packard et al., 1996b). Here, we argue that this ETS-based model can be used to predict zooplankton respiratory oxygen consumption.

**Material and methods**

The data used in this paper have been drawn from the zooplankton studies of King and Packard (1975) and the bacterial studies of Berdalet et al. (1995) and Packard et al. (1996a, b). The older ETS data have been converted to values consistent with the newer methodology, using the correction factor of 3.3 according to Owens and King (1975). The use of the bacterial studies is based on the understanding that, because of the universal nature of the relationship between respiration and the respiratory electron transport system in bacteria and zooplankton, lessons learned from a study of respiration in bacteria can guide our thinking about respiration in zooplankton.

The zooplankton respiration and ETS activity measurements are described in Packard et al. (1974) and King and Packard (1975). Bacterial respiration in Pseudomonas nautica (strain 617) was measured according to Berdalet et al. (1995), and the bacterial ETS was measured kinetically using the method of Packard and Williams (1981).

**Results**

The two metabolic models, as defined by Equations (1) and (2), have three components. Both models claim to be based on first principles, and both claim to be applicable to multiple physiological processes. Here, we compare them for the specific case of respiratory oxygen consumption in both zooplankton and bacteria.

Other than the conceptual physics—chemistry and mathematical differences described in the introduction, a major difference between the two models is the physiological scale of focus. The MTE focuses on explaining the difference in respiration between organisms of different size, for example a eucaryotic and a tintinnid. It finds that biomass, as in Kleiber’s (1932) law, serves as a good proxy for respiration over this scale of difference. If we examine this claim for zooplankton over a biomass range of 0.001–432 mg dry weight per animal and a respiration range of 0.004–183 $\mu$g O$_2$ h$^{-1}$ (animal)$^{-1}$, we find that Kleiber’s law and the MTE describe the data well over these five orders of magnitude (Figure 2). The equation is $R = 1.5 M^{0.76}$ with $M$ the dry weight per animal.
and cell protein in the culture. The kinetic constants were determined by optimization from ranges of values in the literature. (b) CO₂ contains mitochondria, and mitochondria contain the respiratory electron transport system. Because the respiratory ETS is a constitutive enzyme system, it correlates with cell biomass (Figure 2b) even better than does respiration (Figure 2a), the difference likely caused by differences in physiological or nutritional states. For resource availability, the EKM proposes that enzyme kinetics, via substrate-dependent regulation of the maximum rate capacity (V_{max}), can explain mechanistically the variations in respiration (Packard et al., 1996b, 2004). EKM argues that respiration is controlled by the levels of pyridine nucleotides available to the ETS and that the ETS controls the delivery rate of electrons to cytochrome oxidase and consequently the reduction of O₂ to H₂O. Accordingly, this implies that, for respiratory oxygen consumption, potential respiration, the V_{max} of the ETS is an appropriate predictor. For zooplankton, if in Figure 2a we replace dry weight with potential respiration (Φ), we find the correlation improved (Figure 4a) and, because the slope is so close to 1, we can eliminate the use of logarithms (Figure 4b). This figure shows that over more than five orders of magnitude, respiration and Φ are linearly related. Furthermore, the R–Φ ratio of 0.47 (Figure 4b) argues that the in vivo substrate level of these organisms (representing five phyla) was close to the Michaelis–Menten constant (K_{m}), i.e. the point at which the respiration rate is one half of the V_{max} or the potential rate (Figure 5).

In addition to using V_{max}, the EKM also uses the Michaelis–Menten rectangular hyperbolic expression, S/(K_{m}+S), as in
Equation (3) (Packard et al., 1996b, 2004). The parallel-decline in the pyruvate and the respiration rate between hours 10 and 13 in Figure 3 and the decline in the enzyme activity as the substrate for the enzyme falls in a Michaelis–Menten plot (Figure 5a) suggest that, as the pyruvate in the culture medium falls, so do the ETS reactants (Figure 5a and b) and the respiration rate (Figure 3). When the pyridine nucleotide substrates of the ETS are modelled as a time function (Figure 5b) and inserted in the bisubstrate version of Equation (2) along with the \( R \) time course in Figure 6, the EKM can accurately calculate respiratory O\(_2\) consumption during periods of both nutrient limitation and nutrient sufficiency (Figure 6). To our knowledge, no other model of

---

**Figure 2.** (a) Logarithmic plot of respiratory O\(_2\) consumption \([\mu L \text{O}_2 \text{ h}^{-1} \text{ (animal)}^{-1}]\) as a function of dry weight (mg animal\(^{-1}\)) in five phyla of zooplankton. *Tomopteris septentrionalis* represented Annelida; *Sagitta elegans*, *Chaetognatha*; and *Pleurobrachia bachei*, Ctenophora. *Arthropoda* was represented by eight species of crustaceans that included Amphipoda, Copepoda, Decapoda, and Euphausiacea. *Coelenterata* was represented by *Leuckartiara octona*, *Phialidium gregarium*, and *Stomotoca artra*. The power function \( R = 1.5 M^{0.76} \) is in close agreement with Kleiber's law (Hochachka and Somero, 2002; Whitfield, 2006). (b) Logarithmic plot of potential respiratory O\(_2\) consumption \([\mu L \text{O}_2 \text{ h}^{-1} \text{ (animal)}^{-1}]\) as a function of dry weight (mg animal\(^{-1}\)) in the same zooplankton. The data are recalculated and replotted from King and Packard (1975).

---

**Figure 3.** Respiration at different physiological states in the marine bacterium *Pseudomonas nautica* as in Figure 2(PnPy260593). Note the drop in the respiration (solid squares) after the pyruvate (solid circles) became limited. The MTE prediction of respiration from biomass according to Kleiber's law using the equation \( R = 0.76 M^{0.75} \) from Figure 2a, where \( M \) is the cell protein in the bacteria culture. Note that, as the nutrients become limiting after hour 10, the predicted \( R \) and the measured \( R \) diverge. Kleiber's law does not apply here.
respiration can predict the oxygen consumption rate at the level of accuracy shown in Figure 6.

Discussion

The final difference in the two models is the temperature term. The MTE uses the Boltzmann factor \(e^{E_a/kT}\) from statistical mechanics with the atomic-scale Boltzmann constant, the absolute temperature, and atomic-scale Arrhenius energy of activation in electron volts (eV), according to Gillooly et al. (2001). The atomic scale is used by atomic, nuclear, and particle physicists, because it is convenient (Feynman, 1998), but normally it is not used by other scientific communities. On the other hand, the EKM works at the molar level using Arrhenius’ theory and equation, \(R = \frac{Ae^{E_a/RgT}}{2Ea} \) relating absolute temperature to biological, biochemical, and chemical rates according to more than a century of research following Arrhenius’s seminal paper in 1889 (Arrhenius, 1889, 1915; Seiwell, 1937; Sizer, 1942; Moore, 1955; Segel, 1976; Ikeda et al., 2000; Hochachka and Somero, 2002; Angelova et al., 2007). It calculates the \(F_1\) at \(T_1\) from the \(F_0\) measured at \(T_0\) by exploiting the fact that \(A\) is constant for the reaction. Thus, the temperature expression in the EKM [Equation (2)] is \(e^{E_a(1/T_1 – 1/T_0)}\) rather than \(e^{E_a/kT}\) as in the MTE algorithm [Equation (1)]. The scale difference in the two models is large, i.e. Avogadro’s number (6.022 \(\times 10^{23}\) atoms mol\(^{-1}\)). However, if one does not mix scales and calculates \(e^{E_a/kT}\) using molar-scale units of J mol\(^{-1}\), J mol\(^{-1}\) K\(^{-1}\), and K\(^{-1}\) and calculates...
of oxygen. Here, ETS complex I, the complex that oxidizes the

From there, we can continue to focus down into the mitochondrial
somes, endoplasmic reticulum, and vacuoles to the mitochondria.

organism, the organs and tissues, down past the non-respiring ribo-

why we do not focus our attention down through the biomass of an

ory biochemistry yields so much more understanding of respiration

20th century, Warburg, Keilin, Szent-Györgyi, Krebs, Lehninger,

problems. We argue that, by thinking about causality, one can construct

margin of error will handicap the MTE in resolving fine-scale pro-

as much as 20 orders of magnitude in body mass, the uncertainty in

dictor of respiration and, although respiration can be predicted over

laureate physicist, argued against (Feynman, 1997). For these

Community. Electron volt is a unit that even Richard Feynman, the Nobel

in it should describe respiratory temperature depen-

Conclusions

(i) The MTE and Kleiber’s law describe respiration over a large

range of body sizes, but do not describe respiration on short

time-scales or in different physiological states (Figures 2a and

(ii) The Arrhenius equation describes the impact of temperature on the organism’s respiration within the normal temperature range of an organism’s habitat.

(iii) The EKM argues that predictions of respiration could be improved, as in Figure 6, if the respiration model were based on potential respiration and not an organism’s biomass.

(iv) Finally, the EKM states that, at any temperature, respiration is the product of potential respiration and the relative availability of the substrates that modulate the potential respiration [Equation (3)]. On this basis, the model can explain and accurately predict respiration on short time-scales (Figure 6).

Acknowledgements

We thank Santiago Torres Curbelo, Miguel Alcaraz Medrano, and Elisa Berdalet for their help, support, and encouragement. Luis Valdez encouraged us to contribute our work to this symposium volume; we thank him for catalyzing the crystallization of our

Figure 6. Potential respiration (as $\Phi/4$), measured respiration
(solid squares), and the EKM predicted respiration (solid circles) in
the marine bacterium, Pseudomonas nautica as in Figure 1a
(PnPhy26593). Note that $\Phi$ does not drop with the respiration after
pyruvate was exhausted, but from $\Phi$, with the reduced substrates
(Figure 5b), and using Equation (2), the EKM predicts low respiration
after pyruvate was exhausted. Note also the parallelism between the
measured respiration and the EKM-predicted respiration through the
entire 27 h time course of the experiment.

$e^{-E_r/RT}$ using the atomic-scale units of $eV$, $eV K^{-1}$, and $K^{-1}$, then $e^{-E_r/RkT}$ will equal $e^{-E_r/RT}$. However, the great body of biological,

biochemical, and chemical literature over the last century dealing
with energy of activation and temperature-dependence of biological
rate processes is in molar units. Switching to $eV$ units, which are not
mainstream SI units, will confuse and fractionate the biological com-

munity. Electron volt is a unit that even Richard Feynman, the Nobel

laureate physicist, argued against (Feynman, 1997). For these
reasons, the EKM uses the Arrhenius equation, embedded in
Equation (2), to calculate respiration.

The MTE, based on Kleiber’s law, uses mass as its primary pre-
dictor of respiration and, although respiration can be predicted over
as much as 20 orders of magnitude in body mass, the uncertainty in
the prediction may be as much as several orders of magnitude
(Hochachka and Somero, 2002; Whitfield, 2006). Such a large
margin of error will handicap the MTE in resolving fine-scale pro-
blems. We argue that, by thinking about causality, one can construct
a model based on dynamic biochemistry that can yield more under-
standing of respiration than can a model based on biomass. In the
20th century, Warburg, Keilin, Szent-Györgyi, Krebs, Lehninger,
Chance, Green, Slater, Boyer, and Mitchell explained the role in res-
piration of the cell-free extracts, oxidative enzymes, the cyto-

chromes, the mitochondria, ATP, the electron transfer chain,
oxidative phosphorylation, and proton motive force (Fruton and
Simmonds, 1958; Keilin, 1966; Mahler and Cordes, 1971;
Lehninger et al., 1993; Nelson and Cox, 2005). They do not
invoke biomass in any of their explanations. Knowing this, respira-
tory biochemistry yields so much more understanding of respiration
than can be attributed to biomass alone that it is hard to understand
why we do not focus our attention down through the biomass of an
organism, the organs and tissues, down past the non-respiring ribo-
somes, endoplasmic reticulum, and vacuoles to the mitochondria.

From there, we can continue to focus down into the mitochondrial
inner membrane where the five complexes of the ETS control the
oxidation of the reduced pyridine nucleotides (produced in carbon
oxidation), the production of ATP, and the consumption of oxygen. Here, ETS complex I, the complex that oxidizes the

pyridine nucleotides, regulates the electron flux to O$_2$ and hence
the rate of O$_2$ consumption. This flux sets the respiratory potential,
the $V_{max}$ of respiration. Using this $V_{max}$ as a predictor of respiration,
rather than biomass of the organism ($M$), alone yields a mechanistic
understanding of respiration. However, the predictive capability
becomes even greater upon realizing that, because $V_{max}$ is a reaction
rate, one can apply the theories of both Michaelis–Menten, to
resource limitation (substrates), and Arrhenius, to predict tempera-
ture dependence. With this understanding, it becomes possible to
write a first-principle-based model of respiration as in Equation (2).

Therefore, is Equation (2) a good first-principles model of zoo-
plankton respiration from potential respiration, enzyme kinetics,
and the Arrhenius equation? Because the dominant part of the
EKM is the relationship between the respiration and the potential
respiration and because this relationship, $R = f(V_{max}) = 0.466\times V_{max} -
1.560 \ (r^2 = 0.9846)$ or $R = 0.54^\times V_{max}^{0.9414} \ (r^2 = 0.9811)$, describes
the respiration rate in zooplankton so well over five orders of mag-
nitude, the EKM has a good chance of being successful. Add to
this, the probability that the successful use of Michaelis–Menten
kinetics to describe the variations of respiration during different
physiological states in bacteria (Figure 6) can be repeated in zoo-
plankton, and the chance of the EKM being successful becomes
even greater. Furthermore, Sizer (1942), Moore (1955), Pauling
(1958), Eggers et al. (1964), Fruton and Simmonds (1958),
Giese (1963), Prosser and Brown (1961), Mahler and Cordes
(1971), Segel (1976), Lehninger et al. (1993), and Hochachka
and Somero (2002) have explained to generations of students
how, via the Arrhenius equation, temperature determines the rate of chemical reactions, biochemical reactions, enzymatic re-
actions, and the physiological processes built on these reactions.
Consequently, the use of Equation (2) with the Arrhenius equation
embedded in it should describe respiratory temperature depen-
dence well.

Conclusions

(i) The MTE and Kleiber’s law describe respiration over a large
range of body sizes, but do not describe respiration on short

time-scales or in different physiological states (Figures 2a and

(ii) The Arrhenius equation describes the impact of temperature on the organism’s respiration within the normal temperature range of an organism’s habitat.

(iii) The EKM argues that predictions of respiration could be improved, as in Figure 6, if the respiration model were based on potential respiration and not an organism’s biomass.

(iv) Finally, the EKM states that, at any temperature, respiration is the product of potential respiration and the relative avail-
ability of the substrates that modulate the potential respiration [Equation (3)]. On this basis, the model can explain and accurately predict respiration on short time-scales (Figure 6).

Acknowledgements

We thank Santiago Torres Curbelo, Miguel Alcaraz Medrano, and
Elisa Berdalet for their help, support, and encouragement. Luis
Valdez encouraged us to contribute our work to this symposium
volume; we thank him for catalyzing the crystallization of our
thoughts here. Financial support was provided by the University of Las Palmas de Gran Canaria (ULPCC), the Spanish Ministry of Education and Science, the Graduate Programme in Oceanography at the ULPCC, the Institut Ciències del Mar (CSIC) in Barcelona, and the research grants MICROROL (CICYTLTM 2004-02575/MAR), MODIVUS (CTM2005-04 795/MAR), and CONAFRICA (CTM 2004-02319/MAR). This is contribution #200709 from the Bigelow Laboratory for Ocean Sciences.

References


doi:10.1093/icesjms/fsn003