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Linking allometric macrobenthic processes to hypoxia using the Peters mass balance model

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ABSTRACT

Eutrophication alters coastal aquatic food web structure and function through effects of organic enrichment and hypoxia. Macrobenthic communities provide good ecological indicators of impacts on ecosystem function induced by eutrophication. However, effective resource management requires validation of ecological indicators through mechanistic links to specific stressors of concern. Organic enrichment along with subsequent hypoxia typically engenders depauperate macrobenthic communities consisting of small short-lived surface-dwelling organisms. Metabolic ecology theory offers great promise for understanding the role of body size within the context of the trophic dynamics of aquatic ecosystems. Body size is a fundamental ecological trait because it underpins vital rates, which can be scaled up to the ecosystem level through allometric laws. Thus, body size-dependent mechanisms can potentially be used to link macrobenthic indicators to ecosystem consequences of organic enrichment and hypoxia. The Peters mass balance model (PMBM) [Peters, R.H., 1983. The Ecological Implications of Body Size. Cambridge University Press, New York, 329 pp.] provides a platform for projecting stress-induced changes in the distribution of biomass among discrete size classes of organisms. In the PMBM, changes in the distribution of biomass ensue over time from differences between gains due to ingestion and losses due to egestion, respiration, and mortality. Production of biomass results when ingestion exceeds combined losses. In the present study, the PMBM was modified to envisage an inhibitory effect of hypoxia on ingestion by: (1) using unique size-specific mortality coefficients in order to realize target inverted biomass-size distributions during model initialization; (2) linking ingestion rate to the hyperbolic relationship between oxygen consumption rate (OCR) and ambient dissolved oxygen (DO); (3) hypothesizing how oxygen regulation ability follows an allometric scaling rule relative to the supply of DO. If food is unlimited, the most straightforward hypothetical connection between the oxygen consumption rate and ingestion assumes a direct proportional relationship under oxygen limitation. Although small organisms can attain much higher mass-specific rates of oxygen consumption than large ones, previous limited research on oxygen consumption relative to oxygen supply implies that large organisms regulate oxygen intake better than small individuals as ambient DO declines. Counterintuitively, linked ingestion deficits would be accordingly higher for small organisms than large organisms under hypoxia. In pilot simulations incorporating hypothetical ingestion deficits due to oxygen limitation within a modified PMBM, contrasting scenarios emulated continuous DO and water temperature conditions experienced at two shallow subtidal sites for 32 d in summer. Resulting ingestion deficits reduced total biomass and favored large size classes. Effects were stronger under the more hypoxic scenario: total biomass was lower and the biomass-size distribution was more uneven among the size classes. Differences were even more evident in terms of an integrative macrobenthic process indicator, the normalized biomass-size spectrum (NBSS). A realistic hypoxia mass balance model (HMBM) must incorporate various allometric effects that are not necessarily expressed in the same direction relative to body size. A useful resource management tool for anticipating effects of environmental stress on aquatic ecosystem function could be developed from the quantification of trophic transfer potential within a refined PMBM.

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1. Introduction

The macrobenthic fauna plays an essential role in the maintenance of coastal ecosystem integrity by mediating exchanges and transformations of energy and materials, including nutrients, between the water column and the sediment (Hansen and Kristensen, 1997; Twilley et al., 1999). Furthermore, secondary production by the macrobenthos provides an important vehicle of trophic transfer within the coastal ecosystem (Diaz and Rosenberg, 1995). Therefore, stress-induced changes to the macrobenthos need to be understood well enough to apprehend consequential losses of attendant vital ecosystem services.

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Eutrophication adversely affects the benthic subsystem (Pearson and Rosenberg, 1978; Yingst and Rhoads, 1980; Dauer et al., 1992; Diaz, 2001; Levin, 2003). Resulting degraded sediment conditions include excessive organic enrichment, hypoxia, changes in sediment biogeochemistry, upward shifts in the redox discontinuity layer, and the release of toxic metabolic byproducts (Nilsson and Rosenberg, 2000; Rosenberg, 2001). Effects of these stressors are expressed through changes in macrobenthic community structure and function, as reflected by secondary production, diversity, and successional status (Diaz and Rosenberg, 1995; Gray et al., 2002). Ultimately, massive amounts of trophic potential are subverted into the microbial food chain under severe hypoxia and anoxia (Diaz and Rosenberg, 2008).

Organic enrichment and subsequent hypoxia typically engender depauperate macrobenthic communities mostly consisting of small short-lived surface-dwelling organisms. A classic conceptual model of organic enrichment effects, referred to by Gray et al. (2002) as the P-R model after Pearson and Rosenberg (1978), depicts marked responses by the macrobenthic community across an organic enrichment gradient in terms of concurrent changes in abundance, biomass, and species richness. Both taxonomic diversity and biomass increase with moderate organic enrichment. Peak abundance at high levels of enrichment coincides with markedly lower species richness and moderately high biomass, reflecting the occurrence of abundant short-lived small-bodied eurytolerant opportunists. All three faunal metrics fall off at extreme levels of organic enrichment, in connection with severe hypoxia and anoxia.

Gray et al. (2002) point out the descriptive nature of the P-R model, and note that it has seldom been rigorously tested. Moreover, they challenge a causal premise of the model, stating: "major effects on benthic fauna result from hypoxia rather than organic enrichment per se". Despite well-documented effects of organic enrichment and hypoxia on the macrobenthos, little has been done to elucidate macrobenthic community responses as a product of underlying mechanisms (Grall and Chauvaud, 2002). The development of an explanatory mechanistic model could help tease apart potentially disparate effects of organic enrichment and hypoxia on the macrobenthic community. A complete mechanistic model of macrobenthic responses to organic enrichment and hypoxia would be formidable due to multiple complex effects involving changes in food availability and quality, chronic physiological effects of low oxygen, acute mortality effects, biogeochemical interactions among multiple stressors, physiological respiratory adaptations, and faunal feedback effects. A metabolic ecology perspective predicated on allometric scaling laws (Brown et al., 2004; Kooijman et al., 2008; Sousa et al., 2008) could provide an organizing framework for relating such diverse phenomena occurring at multiple scales into a practical mechanistic model.

Recent advances toward a unified view of ecology ensue from the application of allometric rules for scaling metabolic processes up to the ecosystem level. Metabolic ecology theory offers great promise for understanding the role of body size within the context of the trophic dynamics of aquatic ecosystems (Brown et al., 2007; Cohen, 2007; Huryn and Benke, 2007), as well as a framework for considering effects of environmental stress on ecosystem function. Body size is a fundamental ecological trait because it underpins vital rates for many processes defined by allometric scaling rules (van der Meer, 2006; Persson and De Roos, 2007). Thus, body size-dependent mechanisms can potentially be used to link macrobenthic indicators to ecosystem consequences of organic enrichment and hypoxia.

The metabolic scope of aerobic aquatic metazoans may be constrained by the supply of dissolved oxygen (DO), given unlimited food availability (Forbes and Lopez, 1990). Furthermore, the solubility of oxygen decreases nonlinearly with increasing water temperature. Consequently, the metabolic scope for ectotherms can fall well below maximum or even standard metabolic rates at high temperatures as these two abiotic variables covary (Sokolova and Pörtner, 2003). As a principal determinant of the entire energy budget, food supply represents another potential constraint on the metabolic scope of ectothermic aquatic metazoans (Kooijman et al., 2008). Moreover, feeding rate and oxygen uptake constraints are both strongly linked to body size (Zeuthen, 1953; Ditadi et al., 1997; Kooijman et al., 2008). Hence, the difference between the metabolic scope and the standard metabolic requirement also scales allometrically. Because decomposition of organic matter consumes oxygen, opposing gradients in organic loading and oxygen supply act as interacting drivers of macrobenthic secondary production in marine and estuarine ecosystems (Forbes et al., 1994).

The Peters mass balance model (PMBM) (Peters, 1983) provides a platform for projecting stress-induced changes in the distribution of biomass among discrete size classes of organisms. In this simulation model, changes in the distribution of biomass over time result from differences between gains due to ingestion and losses due to egestion, respiration, and mortality. Production of biomass ensues when ingestion exceeds combined losses. The purpose of this study was to initiate the development of a Hypoxia Mass Balance Model (HMBM) based on modification of the PMBM and to demonstrate how the HMBM can be used to project changes in biomass-size distributions under different oxygen limitation scenarios. In the present study, the PMBM was modified to better envisage effects of hypoxia by: (1) incorporating unique size-specific mortality coefficients in order to realize inverted target biomass-size distributions during model initialization; (2) linking ingestion rate to the hyperbolic relationship between oxygen consumption rate (OCR) and ambient DO; (3) hypothesizing how oxygen regulation ability follows an allometric scaling rule relative to the supply of DO. Information about allometric scaling of mortality, oxygen consumption, and how ingestion is tied to oxygen consumption was obtained from existing data and the literature. Because the HMBM is size-structured and derived from autecological mechanisms, the output can be readily expressed in terms of macrobenthic process indicators involving body size as an underlying trait, including the biomass-size spectrum, secondary production, and the community turnover rate (Rakocinski and Zapfe, 2005; Rakocinski, 2007).

2. Materials and methods

2.1. Peters mass balance model (PMBM)

The PMBM simulates, "the movement of material through the ecosystem as a function of time and ecosystem size structure" (Peters, 1983) (Fig. 1). The size of each W_i compartment at each time step reflects the cumulative biomass for size class i at a particular daily increment. Model output represents biomass amounts within designated W_i compartments, which eventually reach a balanced equilibrium. The PMBM (*sensu* Griesbach et al., 1982; Peters, 1983) was initially implemented for this study using the simulation software package, Simile v4.4 (Simulistics Ltd©). Coefficients adopted from the Griesbach et al. (1982) version of the PMBM in accord with exponents based on the "¾ allometric scaling rule" express vital rates in g mass g^{-1} d^{-1}. The mass-specific form of the allometric exponent, −0.25, was used for model terms. Allometric vital rates represent ingestion (I_i), respiration (R_i), egestion (D_i), mortality (M_i) and resultant growth (G_i) (i.e., production), which accrues to compartment i biomass. PMBM simulations in the literature typically ensue over a two-year period, by which time equilibrium biomass-size distributions are often reached. Accordingly, trial HMBM simulations for this study were initialized for 700 d before imposing simulated oxygen limitation. Eight geometric scaled (octaves – base 2) size classes used for trial simulations ranged from 0.46 to 90 mg wet weight and matched the most predominant of 11 size classes observed within macrobenthic samples.

2.2. Size-specific mortality

The PMBM is very sensitive to the mortality function. In the original form of the model, mortality was scaled to the relative standing biomass of any size class by the mortality coefficient, F, such that $M_i = (B_i^F/\sum B_i^F)$ $\sum I_i$ (where $\sum I_i$ = food requirements for the animal community)

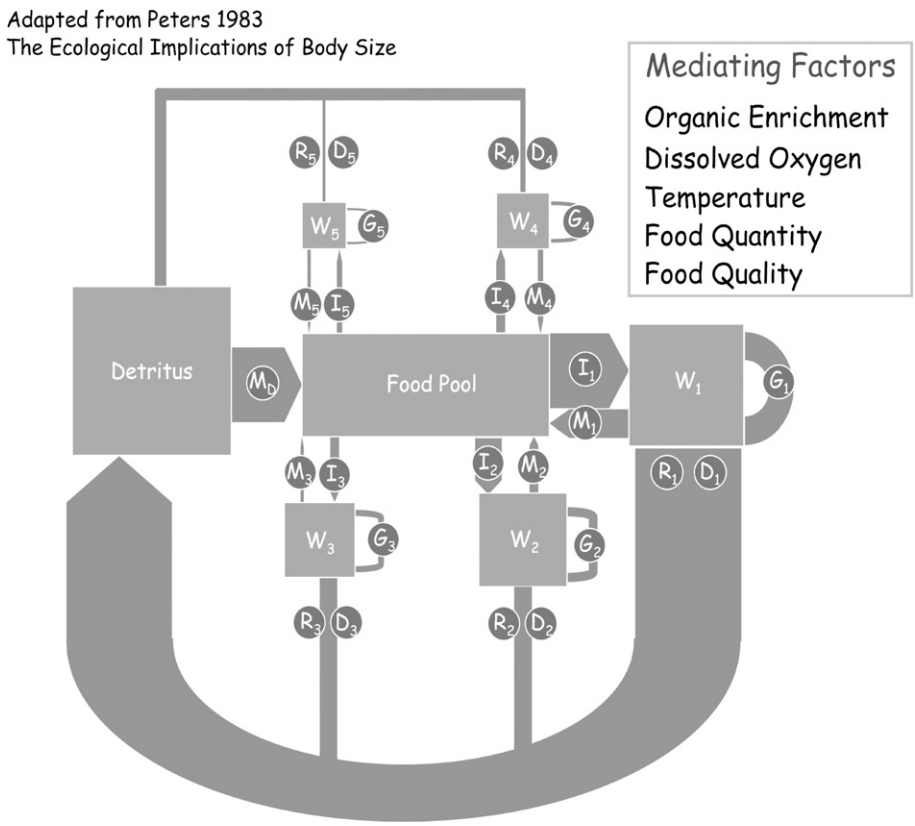

Fig. 1. Peters mass balance model (PMBM). The size of each W_i box represents the biomass of size class *i*, accumulated over time until an equilibrium biomass distribution is reached. I = ingestion; R = respiration; D = egestion; M = mortality; G = production.

(Peters, 1983). Although the value of *F* has a flexible effect on the distribution of biomass among the size classes, the biomass values of small size classes must always exceed those of larger organisms at equilibrium using this mortality formulation (Fig. 2A). However, the standing biomass of larger size classes is typically greater than that for small size classes in macrobenthic communities, reflecting the time-integrated accumulation of biomass (Rasmussen, 1993; Saiz-Salinas and Ramos, 1999). So, to enable proper initialization of the PMBM, a more flexible mortality function that can yield inverted macrobenthic biomass-size distributions is required.

Peters (1978) compared several modified forms of the PMBM that implement alternative mortality functions he referred to as: 'functional response', 'food selection', 'random stress', and 'size-specific' forms. Inverted biomass-size distributions are attainable using the size-specific mortality function, which limits the accumulation of biomass within each size class compartment through the allometric exponent, *d*: $W_j^d/\sum W_i^d$, where W_j represents the size class of concern, and *i* represents a counter for the size classes (Fig. 2B). Size-specific mortality causes the biomass-size distribution to remain dynamic over extended simulation periods (e.g., 25–100 years). As time progresses, smaller size classes consecutively die out until only the largest size class remains at equilibrium. This result resembles community succession. As with other vital rates, the mass-specific allometric exponent of −0.25 provides a good first approximation of *d*. However, possible target biomass-size distributions are still limited when the same mortality exponent is used for all size classes in the model.

To achieve the full flexibility needed for emulating observed target biomass-size distributions as a means of initializing the HMBM, unique size-specific *d* values are required: $W_j^d{}_j/\sum W_i^d{}_i$. Presumably, unique mortality exponents reflect an allometric component (i.e., the exponent, −0.25) in addition to specific vagaries for each size class which can accrue into substantive effects on cumulative biomass. For initialization, the PMBM can now be tuned to reach a specific target biomass-size

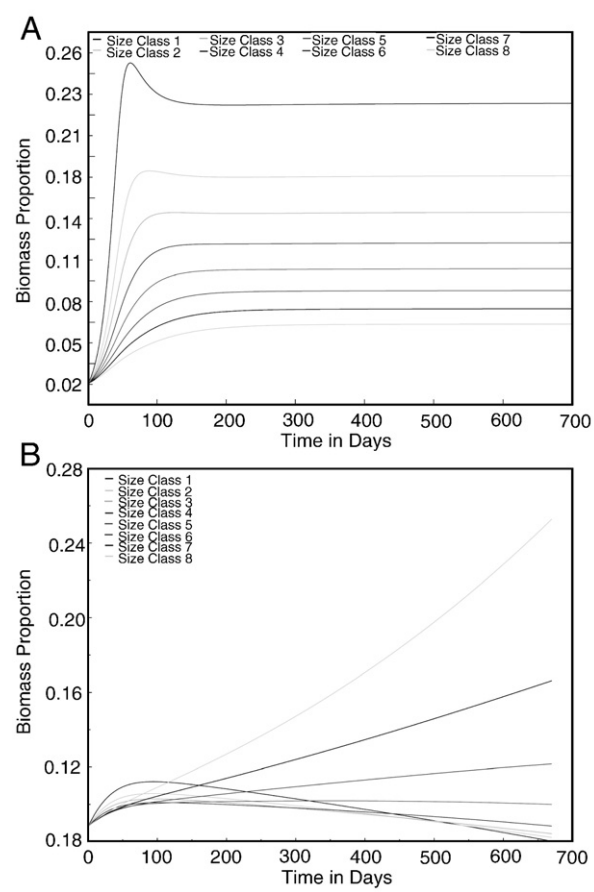

Fig. 2. A. Biomass-size distributions over 700 d based on the proportional mortality function, B. Biomass-size distributions over 700 d using unique size-specific mortality coefficients.

distribution within a specified time period (Fig. 2B). Feedback dependencies in the mass balances necessitate iterative solution to attain size-specific values (d_i) within a given tolerance needed to yield target biomass-size distributions matching empirical data. To accomplish this task, the PMBM along with an algorithm for obtaining size-specific mortality exponents was coded into VBASIC. An added advantage of this program is that it facilitates expansion of the model to incorporate additional factors related to oxygen limitation and other stressors. Accordingly, changes in biomass-size distributions under simulated hypoxic conditions can be projected subsequent to some designated time point when the distribution matches observed empirical data under normoxia.

2.3. Linking oxygen consumption and ingestion

Oxygen provides an essential reactant that directly limits aerobic metabolism, and hence indirectly limits metabolic capacity. Indeed, previous research reveals a link between oxygen limitation and reduced ingestion in deposit feeding polychaetes (Forbes and Lopez, 1990). This relationship reflects design and behavioral constraints because: (1) surface area:volume limits the amount of food that can be metabolically processed (Forbes, 1989); and (2) the need to ventilate takes precedence over feeding (Forbes et al., 1994). If food is unlimited, the most straightforward hypothetical connection between the oxygen consumption rate (OCR) and ingestion would be a direct proportional relationship with decreasing oxygen.

Oxygen limitation can be modeled analogously to light limitation in plants: OCR increases hyperbolically with ambient DO (Chen et al., 2001). Previous research demonstrates (1) that OCR is hyperbolic with increasing DO; (2) that OCR varies with body size in bivalves, gastropods, errant polychaetes, amphipods, and enteropneusts (Taylor and Brand, 1975; Prabhakara Rao and Prasada Rao, 1984; Suadicani et al., 1992; Hoback and Barnhart, 1996; Ditadi et al., 1997); and (3) that OCR curves vary interspecifically among bivalves (Bayne, 1971; Shumway and Koehn, 1982). Oxygen consumption rate can be referred to the ambient DO concentration in mg O_2 g^{-1} h^{-1} as $OCR = (a + b/DO)^{-1}$ (Fig. 3). The OCR parameter, a, physiologically represents several paths of diffusion and biochemical resistance for O_2.

A hypothetical connection between OCR and ingestion can be extended to subsequent effects on the mass balance dynamics of a particular size class through the ratio between OCR and OCR_{max} (i.e., $OCR_{max} = 1/a$). Resulting proportional deficits in ingestion in connection with oxygen limitation should differentially affect the terms in the mass balance model so as to maintain homeostasis. Accordingly, ingestion deficits should first be subtracted from production (i.e., growth and reproduction) and egestion is assumed proportionally less with ingestion (i.e., $D_i = 0.394 \times I_i$). Remaining deficits in mass would be expressed as degrowth (Forbes and Lopez, 1990) once production potential is exhausted, resulting in further reduction of biomass within the size class.

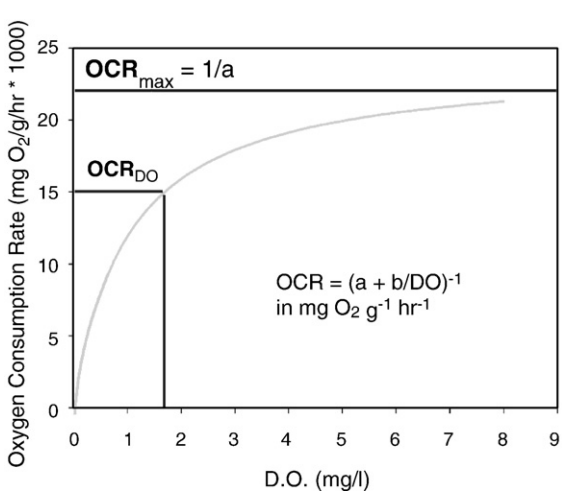

Fig. 3. Hyperbolic relationship between oxygen consumption rate (OCR) and ambient dissolved oxygen (DO).

2.4. Relating oxygen consumption and body size

2.4.1. Estimating asymptotic parameters in OCR curves

The asymptotic parameter for the OCR curve, a, can be approximated by *ad libitum* oxygen consumption (i.e., $OCR_{max} = 1/a$). Smaller organisms have higher asymptotic weight-specific OCR; and specific OCR curves pertain for any combination of water temperature and body size. To estimate provisional a values for trial simulations, an OCR response surface was fit relative to body size and temperature on a log–log scale using an extensive published dataset for three common estuarine bivalves (Kennedy and Mihursky, 1972). Data were digitized from published figures. Individuals ($n = 215$) spanning from 1.2 to 486.2 mg somatic dry mass were acclimated to one of three different temperatures (10°, 20°, or 30 ° C). The linear fitted surface in *ad libitum* OCR exhibited similar influences of temperature and body size on oxygen consumption rate: Log_{10} OCR = − 0.918 + 0.332 Log_2 Temp − 0.355 Log_{10} mg dw ($R^2 = 0.57$; $P < 0.001$) (partial $r = 0.72$ and 0.61, respectively).

2.4.2. Allometric scaling of oxygen regulation capacity

Owing to design considerations related to high surface:volume (i.e., uptake) and less branching of transport networks, small aquatic organisms can attain much higher mass-specific OCR than large organisms (Fig. 4A). Intuitively by extension, small organisms might

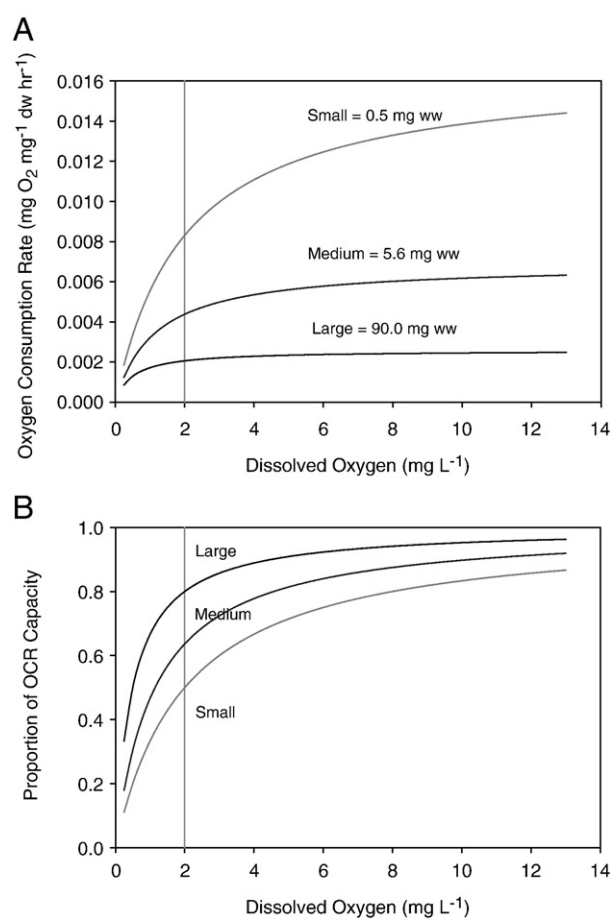

Fig. 4. A. Hypothetical body-size differences in OCR curves, B. Corresponding relative size differences in OCR capacity. Vertical reference line at 2 mg L^{-1} denotes hypoxic threshold.

also be able to regulate oxygen intake better than large organisms as oxygen supply declines. However, previous research on oxygen consumption of crustaceans and bivalves relative to oxygen supply implies that large organisms can regulate oxygen intake better than small individuals as ambient DO declines (Bayne, 1971; Taylor and Brand, 1975; Bridges and Brand, 1980). Apparently, because it is difficult for small organisms to sustain such high weight-specific OCR levels as DO declines, OCR curves of small organisms start falling off at higher ambient DO levels than OCR curves of large organisms. Consequently, relative OCR capacity would also be lower for small organisms as ambient DO declines (Fig. 4B).

The ratio of OCR curve parameters, b/a, also conveys useful information about the metabolic capacity for regulating OCR (Bayne and Livingstone, 1977). A greater capacity to sustain OCR is conferred by a lower b/a ratio (i.e., lowest $b/a \rightarrow 1/a$) (Chen et al., 2001). This ratio seems to increase in association with body size in marine crustaceans (Bridges and Brand, 1980). The ratio, b/a, can also be hypothesized to follow an allometric scaling rule: ratios of parameters for OCR curves fit to data for three body sizes of *Galathea strigosa* from Bridges and Brand (1980) were consistent with the ¾ allometric scaling rule. If such an allometric scaling rule is assumed and a is known, the OCR parameter, b, can be approximated in a relative sense for organisms of different sizes. A ratio of 1 for b/a implies a transitional capacity for regulating OCR. By scaling the ratio of b/a internally and symmetrically from 2 to 0.5 across eight geometric size classes of concern, a relationship fitting an allometric scaling rule was obtained for trial simulations: the slope of the log body mass vs. b/a relationship was −0.285 (i.e., intermediate between hypothetical ¾ fractal based (branching transport networks) and 2/3 surface area–volume based scaling rules). Following this rationale, size-related differences in OCR capacity were obtained for trial simulations. Although these OCR parameter values are provisional, implicit scale invariance (Schmid and Schmid-Araya, 2007) suggested that the incorporation of these allometric effects of oxygen limitation should be reasonable for trial simulations.

3. Results

3.1. Incorporation of DO and water temperature profiles

For trial HMBM simulations, continuous DO and water temperature data measured with YSI multi-parameter sondes in summer 2005 at two shallow subtidal sites in East Bay (Pensacola, FL), PB5 and PB1, provided a template for depicting contrasting oxygen limitation scenarios. Although the scenarios were partly fabricated, realistic profiles representing 32 d of fluctuations were obtained using existing data (Fig. 5). Missing portions of the series were filled in from adjacent data so that implicit trends and cycles were preserved and complete representative series were obtained. Although the PB5 DO profile exhibits wide daily fluctuations and many low DO values, it seldom dips into hypoxia. In contrast, the PB1 DO profile remains hypoxic most of the time and depicts four marked weekly cycles in DO levels. Observations measured at 15 min intervals were aggregated into hourly values for model calculations.

3.2. Trial simulations

To initialize trial HMBM simulations, a target biomass-size distribution was generated comprising eight $\log_2$ scaled size classes and matching that observed at PB1 in late spring 2004 before the onset of seasonal hypoxia. Model initialization used the pre-determined set of size-specific mortality coefficients yielding the target biomass-size distribution in 700 d. Simulated organisms experienced unconstrained ingestion for 700 d prior to 32 d of ingestion deficits associated with oxygen limitation. A reference biomass-size distribution representing 732 d of unconstrained ingestion was also generated for comparison with distributions from simulations of oxygen limitation (Fig. 6). Thus, constrained biomass-size distributions reflected 32 d of ingestion deficits at the end of the 732 d simulation period (i.e., 700 d with 100% ingestion followed by 32 d of constrained ingestion), whereas reference distributions represented the same point in time without any oxygen limitation (i.e., 732 d with 100% ingestion). Ingestion deficits linked proportionately to DO consumption accounted for the differences between constrained and reference biomass-size distributions.

Biomass-size distributions were markedly different between constrained and reference simulations for both PB5 and PB1 scenarios (Fig. 6). Overall, total biomass was reduced and large size classes were relatively better represented under oxygen limitation. Generally, constrained distributions shifted progressively downward with decreasing size relative to the reference distribution. Effects were visibly stronger for the more hypoxic PB1 scenario than for the PB5 scenario. Total biomass was more reduced and distributed less equitably among the size classes in the PB1 scenario than in the PB5 scenario. All of the size classes shifted downward to a greater extent with decreasing body size under the PB1 scenario. Biomass within the largest size class actually shifted slightly upward in the PB5 scenario relative to the reference distribution. Differences in the biomass-size distributions were especially noticeable when expressed as normalized biomass-size spectra (NBSS) (Platt and Denman, 1977; Schwinghamer, 1988), wherein the biomass within each logarithmic size interval is normalized by the width of the size interval (Duplesia and Kerr, 1995) (Fig. 6). Consequently, the downward shift in NBSS values with decreasing body size was very evident, as shown by slopes and intercepts of NBSS.

4. Discussion

The trial HMBM simulations demonstrated how the PMBM can be used as a platform for projecting effects of different DO scenarios on macrobenthic biomass-size distributions. Projected biomass-size distributions were visibly altered by the influence of only 32 d of fluctuations in DO and water temperature on ingestion deficits in association with OCR. Trial simulations only reflect a single hypothetical chronic effect of hypoxia. Hypoxia imposes multiple direct and indirect effects on the growth and survival of macrobenthic organisms (Diaz and Rosenberg, 1995). For example, macrobenthic organisms experience direct lethal effects from severe hypoxia (Hoback and Barnhart, 1996). Consequently, severe hypoxia favors the prevalence of abundant opportunistic small-bodied short-lived taxa reflecting earlier stages of macrobenthic succession (Forbes et al., 1994; Gray et al., 2002). Behavioral effects such as increased energetic costs of heightened ventilation (Forbes and Lopez, 1990), or effects of H_2S on feeding activity (Fuller, 1994) can also be important.

A complete HMBM should consider complex macrobenthic responses within the context of both organic enrichment and hypoxia, which are inextricably linked (Gray et al., 2002). For example, hypoxia may be exerted upon macrobenthic communities that have already shifted towards small body sizes in response to organic enrichment. One conceptual model considers the interaction of opposing gradients in oxygen and food availability as primary drivers of macrobenthic production (Forbes et al., 1994). Consequently, macrobenthic production would be expected to be highest at intermediate levels of both food and oxygen; such a relationship is borne out in macrobenthic samples (Rakocinski, 2007). For example, tidal marsh sites that are subject to high organic loading and cyclic DO are typified by very productive macrobenthic communities consisting of numerous small opportunistic organisms. Consequences of organic enrichment should be tractable within the HMBM because the size of the food pool is specified in the original PMBM, and food quality (e.g., C:N) could also be further specified. Related allometric vital rates could then be incorporated into the HMBM.

The PMBM is compatible with metabolic ecology because both approaches are founded upon the idea of scaling up from the individual

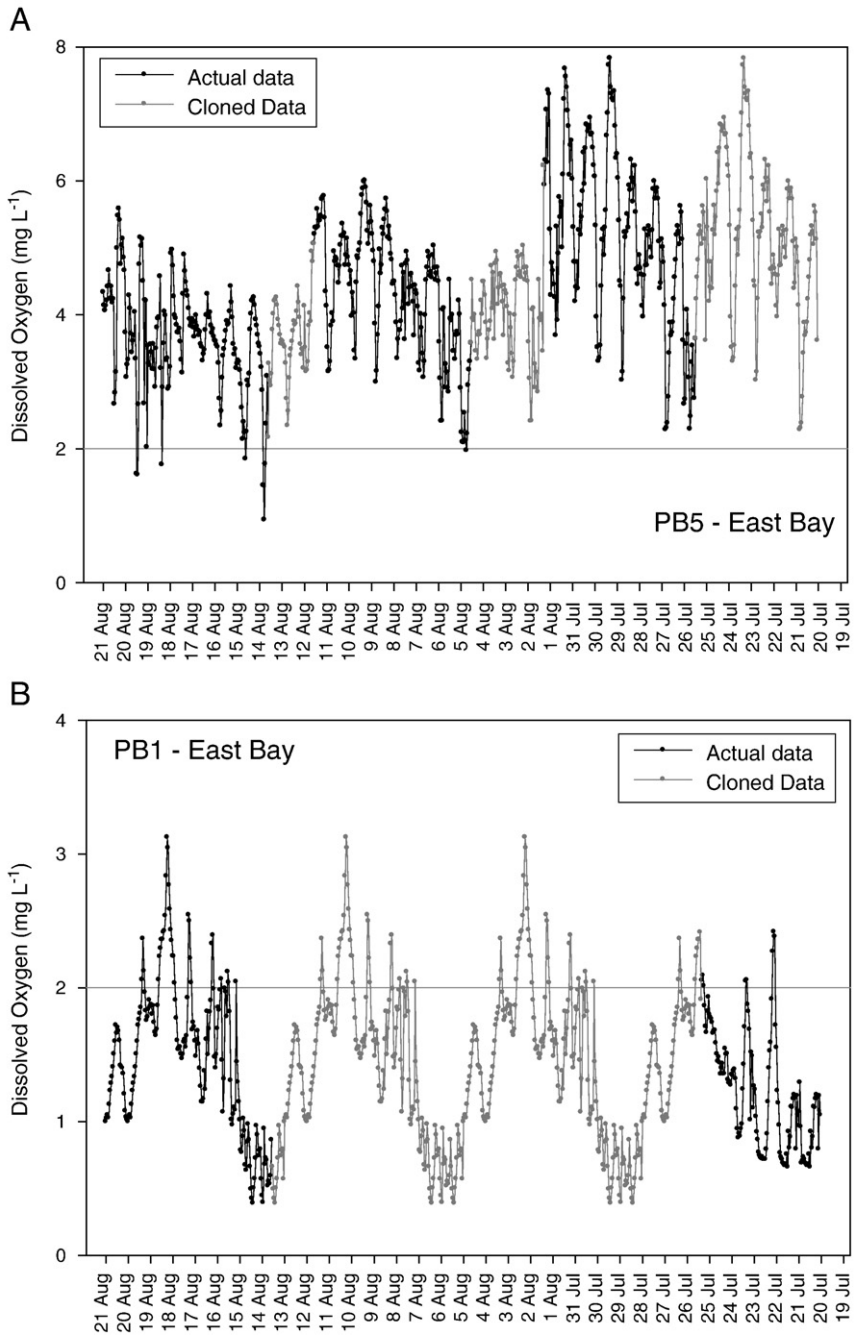

Fig. 5. Dissolved oxygen profiles for contrasting trial HMBM simulations. The scenarios emulate patterns in continuous DO measured at two shallow subtidal sites in East Bay, FL.

level to higher levels of organization via allometric vital rates (Brown et al., 2004; Kooijman et al., 2008; Sousa et al., 2008). In addition, metabolic ecology embodies a myriad of concepts, including many that should be considered in a refined PMBM. Moreover, model terms within the PMBM need to be clearly defined in ways that are consistent with parameters and quantities in metabolic ecology. For example, a refined PMBM should account for various overhead costs such as those associated with assimilation, digestion and absorption of food. In addition, any parameter involving uncertainty should be treated as probabilistic within a refined PMBM (Constable, 1999). Reconciliation of the PMBM with metabolic theory will help identify quantities and parameters for future experiments designed to calibrate the HMBM.

Body size provides an organizing principle for relating a variety of responses to hypoxia within the HMBM. Many design constraints vary with body size. For example, respiration capacity for small organisms may be driven more by surface area:volume limitations (Graham, 1988) (e.g., integumentary respiration; 2/3 scaling); whereas capacity for large organisms may depend more on transport constraints (e.g., branching networks; 3/4 scaling). It seems counterintuitive that the hypothetical effect of hypoxia on ingestion should favor large organisms in light of many studies of hypoxia that find shifts to small organisms in macrobenthic communities (Pearson and Rosenberg, 1978; Diaz and Rosenberg, 2008). Clearly, the allometric relationship in metabolic capacity for regulating OCR used in trial simulations is based on limited data from the literature and still needs to be tested for generality. Some laboratory experiments found that the growth of small polychaetes was less limited by low DO compared to large worms (Forbes and Lopez, 1990). Clearly, many allometric effects of hypoxia which may not all differ in the same direction with body size are still unaccounted for in the HMBM model. However, trial

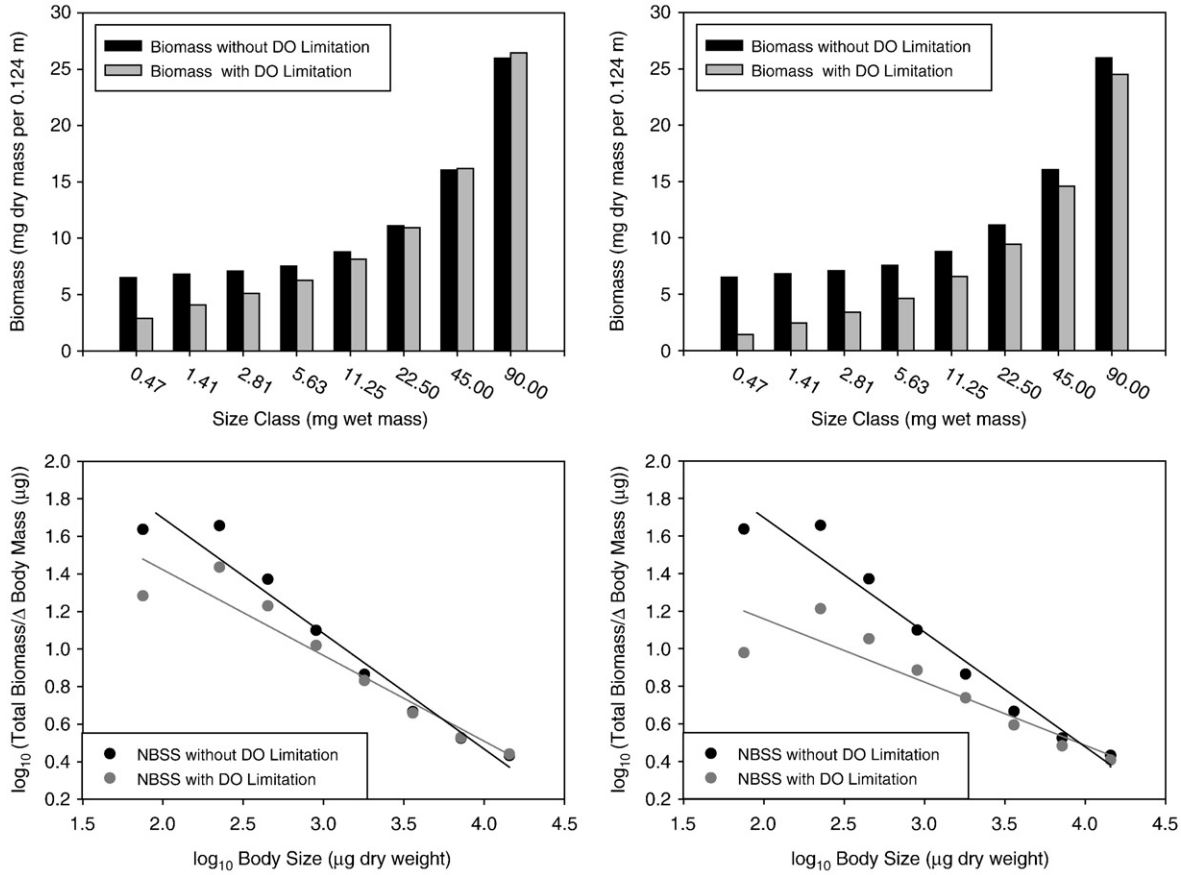

Fig. 6. Output from two trial HMBM simulations. Constrained biomass-size distributions reflect 32 d of ingestion deficits at the end of the 732 d simulation period. Upper panels show biomass-size distributions resulting from constrained vs. reference simulations; Lower panels depict the same output as upper panels in terms of normalized biomass-size spectra (NBSS). Left hand panels depict the PB5 scenario; right hand panels depict the PB1 scenario.

HMBM simulations illustrate how a single mechanism could be expressed within the context of a complex suite of potential effects.

Mortality is paramount within the HMBM model for several reasons. First, HMBM output is very sensitive to the mortality function. Although mortality was treated as deterministic in trial simulations, mortality potentially represents the most probabilistic driven influence on the HMBM. Total mortality comprises multiple causes. Accordingly, total mortality represents the joint probability distribution resulting from the product of mortality constituents, each of which can be expressed by its own probability distribution. Some abiotic causes of mortality would include physical disturbance and lethal effects of anoxia as well as biogeochemical products of hypoxia, such as H_2S (Shumway et al., 1983). Biotic causes of mortality would include death from metabolic deficits and predation (Sagasti et al., 2001). Some mortality constituents would be expected to be independent of body size whereas many others could be related to body size (Forbes et al., 1994).

Identification of different mortality constituents would facilitate separation of trophic transfer and mineralization ecosystem functions within the HMBM. Massive amounts of trophic potential are ultimately subverted into the microbial food chain, including the foraminifera, by severe hypoxia and anoxia (Woulds et al., 2007; Diaz and Rosenberg, 2008). This portion of total mortality would be directed to decomposition and mineralization, rather than being transferred to higher trophic levels. On the other hand, predation mortality provides a key to quantifying trophic transfer potential within the HMBM (Kerr and Dickie, 2001). Total trophic value representing the composite of predation-related trophic transfers for all size classes in the HMBM in turn could be used as a common currency for ecosystem management goals.

A refined PMBM can potentially serve as a science-based ecosystem resource management tool for investigating the implications of different hypoxia scenarios. Metabolic ecology provides a theoretical basis for linking environmental stress and macrobenthic function. In combination with the explanatory power of metabolic ecology, the PMBM offers an ideal platform for testing the influence of ecological factors on macrobenthic processes. For example, the PMBM provides a mechanistic approach for understanding how and why macrobenthic process indicators vary in the face of eutrophication (Rakocinski and Zapfe, 2005; Rakocinski, 2007). The ability to explain shifts in biomass-size distributions in terms of allometric vital rates offers a key to understanding food web dynamics and trophic transfer potential in aquatic ecosystems. Furthermore, many features of the PMBM are commensurate with the most general and widely used ecosystem level trophic model, Ecopath (Pauly et al., 2000; Christensen and Walters, 2004). Recent applications of this ecosystem mass balance model use quantities that are clearly accessible within the PMBM, including terms for 'lost production' (respiration, mineralization), biomass values for the 'trophic groups', production:biomass ratios, consumption:biomass ratios, accumulation, and ecotrophic efficiency (= fraction of production not remineralized) (Arreguín-Sánchez et al., 2002; Pinkerton et al., 2008). The PMBM is clearly amenable to the calculation of these quantities and could also serve as a bridge model to Ecopath. Finally, analogous models that integrate other stressors can be derived similarly to the HMBM. In conclusion, the PMBM approach offers a general framework for investigating aquatic ecosystem stress within an era of rapid climate change and global warming.

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