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Phosphorus Excretion and Body Size in Marine Animals: Microzooplankton and Nutrient Regeneration

Abstract. *In marine animals the rate of excretion of dissolved phosphorus per unit weight increases as body weight decreases. As a consequence microzooplankton may play a major role in planktonic nutrient regeneration.*

Animal excretions are a major source of plant nutrients in the sea (*1*). The animals most often studied in this regard are those captured in plankton nets. The smaller species which are not retained by plankton nets, the microzooplankton, are often overlooked by marine ecologists, and their role in nutrient cycles has never been evaluated.

Examinations of unfiltered water samples reveal that microzooplankton may often constitute a considerable fraction of the total animal biomass. Lohmann (*2*), for example, found that protozoa and very small metazoa, including *Rotifera*, copepod eggs, and certain invertebrate larvae, constituted an annual average of over 50 percent of the total zooplankton biomass in the waters off Kiel. A number of investigators have described the abundance of colorless flagellates (*3*) and ciliates (*4*) in the plankton.

It is well known that the smaller the animal the greater the metabolic rate per unit weight. The rate of nutrient excretion of microzooplankton should therefore be higher than that of net zooplankton per unit weight. Accordingly, it seemed worthwhile to attempt to evaluate the relative importance of animals of different sizes in the production of dissolved phosphorus.

For marine animals larger than 1 mg (dry weight), phosphorus excretion rates were determined by measuring spectrophotometrically the total phosphorus content of the animals and their soluble excretions (*5*). Excretion rates of animals weighing less than 1 mg were determined with the radioisotope P^{32} (*6*). Excretion rates are reported here as the time it takes an animal to release an amount of dissolved phosphorus equal to its total phosphorus content. This will be referred to as the body-equivalent excretion time (BEET).

A marked decrease in the body-equivalent excretion time (Fig. 1) is associated with decreasing animal size. The method of least squares leads to the following linear regression equations: For the upper line, log BEET (hours) is equal to 0.67 log dry weight (g) plus 3.2 ($r = 0.96$). For the lower line, log BEET = 0.33 log dry weight plus 2.6 ($r = 0.98$). Both correlations and the difference between the two slopes are significant at the 1 percent level. An analysis of the phosphorus excretion rates of 24 lots of differently sized (0.05 to 0.1 g without shells) mussels, *Modiolus demissus* (*7*), produced the regression equation: log BEET = 0.51 log dry weight plus 3.5 ($r = 0.53$). There was no significant difference between the slope of this regression line and the upper line in Fig. 1.

Whereas a 12-g lamellibranch released an amount of phosphorus equal to its total phosphorus content every 438 days, the body-equivalent excretion time of a 0.6-mg amphipod was 31 hours, and that of an 0.4×10^{-3} μg ciliate was 14 minutes. Excretion-time of an animal the size of a 1-μ^3 phagotrophic microflagellate is estimated to be about 2 minutes (*7*), based on extrapolation of the lower regression line in Fig. 1 (*8*). It is difficult, if not impossible, to determine the excretion rates of these fragile forms directly.

When the data used in Fig. 1 were compared with data on oxygen consumption as related to body weight in marine animals (*9*) it was found that the ratio of oxygen consumed to phosphorus excreted decreases markedly with decreasing animal size. An animal weighing 1 μg releases approximately 50 times as much phosphorus per unit weight as a 100-mg animal, while the smaller animal consumes only 5 to 8 times as much oxygen per unit weight. Two other workers (*10*) have likewise noted that the "O/P ratio" was significantly lower for mixed zooplankton species than for several larger benthic invertebrates. The explanation of the marked lack of parallelism between these two physiological processes deserves investigation.

A recent example serves to demonstrate the importance of considering size distribution of fauna when computing excretion rates of faunal communities. Rigler (*11*) calculated the rate of release of dissolved phosphorus for lake zooplankton, assuming that rotifers excrete the same amount of phosphorus per unit weight as cladocerans 1000 times heavier. He acknowledged the possibility of error arising from this assumption. My results suggest that this error is indeed significant. On the basis of the lower regression line in Fig. 1 the rotifers would be expected to excrete dissolved phosphorus about ten times as fast as the cladocerans per unit weight.

Figure 1 can be used to demonstrate the relative importance of microzooplankton and macrozooplankton for

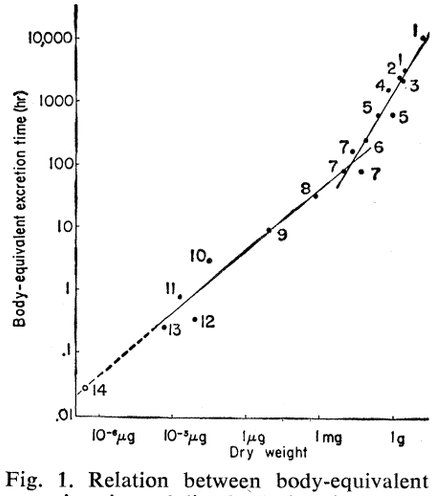

Fig. 1. Relation between body-equivalent excretion time of dissolved phosphorus and body weight of marine animals. 1, *Tridacna crocea*; 2, *Penaeus setiferus*; 3, *Crassostrea virginica*; 4, *Modiolus demissus*; 5, *Uca pugnax*; 6, *Salpa fusiformis*; 7, *Littorina irrorata*; 8, *Lembos intermedius*; 9, *Artemia salina* (nauplii); 10, *Euplotes crassus*; 11, *Euplotes trisulcatus*; 12, *Euplotes vannus*; 13, *Uronema* sp.(?); 14, hypothetical 1-μ^3 (2.5×10^{-7} μg dry wt.) phagotrophic flagellate.

phosphorus regeneration in a given population. For example: if we take a hypothetical zooplankton population composed of 10 percent by weight of ciliates weighing 1×10^{-2} μg each and 90 percent crustaceans the size of *Calanus finmarchicus*, weighing about 0.3 mg each (*12*), the ciliates would contribute over 70 percent of the total dissolved phosphorus released by the population. The substitution of phagotrophic flagellates for ciliates would shift the balance further in favor of the domination of nannozooplankton in nutrient release processes, while the substitution of smaller crustaceans, such as *Acartia clausii*, for *Calanus finmarchicus* would increase the dissolved phosphorus contribution of the net zooplankton.

Thus the contribution of microzooplankton to phosphorus regeneration is much greater than their contribution to total zooplankton biomass. Their participation in other ecologically important processes (for example, regeneration of other nutrients, food consumption) must likewise exceed the importance of their biomass.

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Genetic Regulatory Mechanisms at the Population Level in Man

Abstract. *The distribution of the Rhesus and ABO blood groups, thalassemia, and glucose-6-phosphate dehydrogenase deficiency in Europe and in Jewish populations argues for a regulatory mechanism at the population level which adjusts gene frequencies so that the overall genetic load is not excessive, since gene frequencies giving a high genetic load at one locus are often associated with those giving low genetic loads at other loci.*

Genetic polymorphisms are common in man. The occurrence of polymorphism implies a balanced genetic load (*1*) made up of components derived from the segregation of genotypes less fit than the optimum, and incompatibility. If each polymorphic locus is completely independent of every other, the genetic load would perhaps be so great that it could not be tolerated, especially in a species such as man with a low reproductive potential. However, the development of fitness interactions between genes at different loci may lead to the total load over all loci becoming less than directly cumulative (*2*). An example in man is the ABO-Rh incompatibility interaction (*3*).

A second, but not entirely distinct, method of compensation may be by the control of gene frequencies such that, if there is a high load associated with one polymorphic locus, the gene frequencies at other loci might be adjusted to give relatively low genetic loads. There may, therefore, be a regulatory mechanism at the population level controlling gene frequencies.

In Sardinia, there is a high frequency of thalassemia (*4*) and glucose-6-phosphate dehydrogenase (G6PD) deficiency (*5*). Genes for these conditions may be maintained at a high frequency in the population as a consequence of malaria. Thalassemia, in particular, implies a high genetic load as homozygotes are relatively inviable. Genes for both these conditions are also frequent in the Ferrara region of the Po Valley, and in regions of southern Italy. A map by Morganti (*6*) shows that the frequency of Rh-negative individuals is extremely low in Sardinia, and, furthermore, Ceppellini (*4*) has presented evidence suggesting that within Sardinia the frequency of Rh-negative individuals is low in the malarial regions where the gene frequencies for thalassemia and G6PD deficiency are high, whereas in the mountainous nonmalarial districts, where the gene frequencies for thalassemia and G6PD deficiency are lower, the frequency of Rh-negative individuals is higher. Similarly, Ferrara is a region of low Rh-negative gene frequency, and on the whole there are fewer Rh-negative individuals in southern than in northern Italy. Thus low Rh-negative frequencies, implying a low Rh-incompatibility load, are associated with a high load due to thalassemia and presumably G6PD deficiency.

Morganti (*6*) gives maps of the A, B, O, and Rh gene frequencies for 90 Italian provinces. Let the gene frequencies of the A, B, and O alleles be p, q, and r, respectively, such that $p + q + r = 1$, and let d_A and d_B be the probabilities of death due to incompatibility based on A and B sperm, respectively. The ABO incompatibility load is then (*7*)

$$d_A p(1 - p)^2 + d_B q(1 - q)^2.$$

As q does not vary much in Italy, variations in the ABO incompatibility load are due mainly to variations in p. Maximum A load occurs at $p = 1/3$. The lowest values of p (0.15 to 0.25) occur in Sardinia (*6*). In Ferrara, p is lower than in the neighboring districts, and in southern Italy p is lower than in northern Italy (*6*), but nowhere is p greater than 1/3. Thus low ABO and Rh incompatibility loads occur in regions where the genetic load due to thalassemia and G6PD deficiency is high.

The Rh incompatibility load (*2*) is

$$d_B l m^2,$$

where d_B represents the probability of death due to Rhesus incompatibility, and l and m are the frequencies of the R and r alleles respectively, such that $l + m = 1$. With the assumption that $d_A = d_B$, which is reasonable since