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A model explaining the presence of zones of secondary change and repetition in some bryozoan colonies

Although the colonialism surely developed independently in Graptolithoidea and Bryozoa, both groups share similar patterns of astogeny. One of the common features is the presence of morphological gradients. Many attempts at its explanation were made for more than a half of the century. This paper discusses a new model of the late astogeny in some bryozoan colonies, showing a cyclic reappearance of secondary zones of astogenetic change and astogenetic repetition that cannot be explained by the single morphogen gradient theory.

Recent paper by Urbanek (Urbanek, 2004), discusses all the theories concerning the astogeny (development of colony) of two groups of clonal organisms: Graptolithoidea (a class composed of recent Pterobranchia and extinct Graptolithina, see Mierzejewski & Kulicki, 2002), and Bryozoa. In both groups the first individual, an oozooid (named ancestrula in bryozoans and sicula in the case graptolites) is a sexually produced founder of colony. In some colonies of both groups a morphogenetic gradient can be observed, that can be explained by the morphogen gradient theory (see Urbanek, 2004). In short, this model states that the founder individual produced a morphogen , that by diffusing through the colony, controls the genes expression and thus produce a morphological gradient. This model however does not explain the presence in some bryozoan colonies cyclical zones of change and repetition (Urbanek, 2004). The suggested explanations assumes the cyclicity of the environmental change or cyclity in colony physiology, but the evidence for both seems inadequate. Some authors proposed that during the astogeny new individuals with the abilities of secreting the morphogen were secondarily emerging (see Urbanek, 2004 for references), thus suggesting that the ancestrula was not the only zooid capable of producing the morphogen. However, the question why those secondary zooids, called "pseudoancestrulae" were appearing, remains open.

I propose here a model of astogeny in some bryozoan colony that is based on morphogen theory and explains the proposed presence of "pseudoancestrulae". It can be assumed that the ancestrula posseses the ability to produce a morphogen (let's call it morphogen A), that controls the gradient throughout the colony. Along the colony the amount of A is decreasing, until the threshold level, in which the morphological gradient disappears and a zone of astogenetic repetition begins. But the amount of A is still decreasing, and when it deops to a second threshold level, it activates another morphogen (morphogen B), that again begins to control genes expression thus providing a second zone of astogenetic change. Of course along the colony, the portion of B also decreases, causing a second zone of repetition to appear, still decreasing it may finally activate another morphogen (C) and the process goes on (**see diagram**). This succession of morphogens can be presented as A->B->C->… More parsimoniously would be to assume that only two (A->B->A->…) or even only one morphogen (A->A->…) with a number of thresholds were involved in this process. The last variant would be preferable from the standpoint of the economy of thinking.

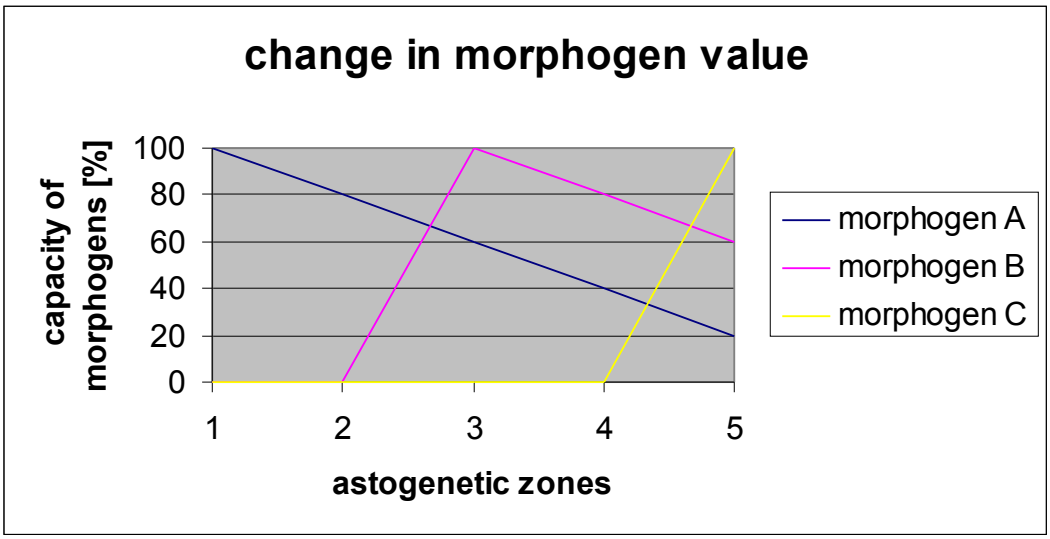

Diagram No. 1: Change in morphogen value along the bryozoan colony. Phases: **1.** - Morphogen "A" enables a primary zone of change. **2.** - Zone of repetition. Decrease of "A" value. Activation of morphogen "B". **3.** – Zone of secondary change enabled by morphogen "B". **4.** – Zone of repetition. "B" value decreasing. Activation of morphogen "C". **5.** – Zone of secondary change enabled by morphogen "C".

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