## Dinoflagellate toxins stimulate coral calcification and cause bleaching

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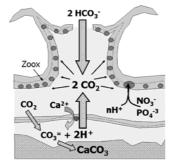
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*Symbiodinium*, the dinoflagellate symbiont of reef corals, makes potent activators of cellular calcium influx called zooxanthellatoxins. Scenarios describe how algal toxins may stimulate coral calcification and promote algal invasion and exit from the host.

The coral's calcifying ectoderm appears particularly susceptible to zooxanthellatoxins because of its abundant calcium transporters. Ectodermal cells compensate for the enhanced calcium influx by pumping calcium toward the skeleton, in exchange for protons. That causes calcification. The coral secretes the protons from calcification into its internal coelenteron cavity. That promotes photosynthesis and nutrient acquisition, by converting bicarbonate to carbon dioxide, and by stimulating mechanisms such as proton-

nutrient co-transport. Toxin production schedules likely contribute to faster coral calcification during the daytime and during nutrient shortages.

Zooxanthellae may also use toxins to modulate host cell phagocytosis and/or exocytosis, assisting their entry into or exit from host corals. Dinoflagellates



produce more of some toxins when nutrient shortages retard their progression through the G1 phase of the cell cycle, which can last for months in nutrient deficient corals. High toxin levels may then induce the coral to evict the algae, or cause infected endodermal cells to lose adhesion and slough away. Either way, algal toxins help the algae to disperse to new hosts with better access to nutrients. Nutrient shortages correlate with high sea surface temperatures, contributing to the correlations between temperature and coral bleaching.

Such putative inter-species uses for dinoflagellate toxins may have evolved from internal uses involving calcium modulation of chromatin conformation and activity.

## Interaction of mineral surfaces with oligopeptides as organic templates: An insight into biomineralization

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Calcite is a rhombohedral polymorph of calcium carbonate  $(CaCO_3)$ . The lowest energy faces of pure calcite are the {104} family of faces. The mineral(calcite) deposition on the exoskeleton of the organism can be related to its DNA pattern, and hence, to the protein sequence that forms the hard body exoskeleton. As a precursor of biomineralization, we are studying the interaction of oligomers/polypeptide chains on (104) calcite surfaces. Our main objective is to find suitable orientations of amino-acid residues in these peptide chains, where these peptide chains align themselves parallel to the calcite surface or parallel to a step on these surfaces. Various sequences of small-chain (3-aminoacid) peptide residues on both polar and non-polar surface steps on calcite (104) faces have been studied. The residue with the highest adsorption energy of the ones that we studied is Phe-leu-lys<sup>4-</sup> with total adsorption energy of -1.071eV (non-polar calcite surface step) or -0.3571 eV/residue. The pH during the calculations is assumed to be high such that the carboxylic groups are completely deprotonated. For the interaction of a 12-amino acid long peptide chain in alkaline conditions with polar steps, we calculate an adsorption energy of -0.09824 eV/amino acid residue and -0.1978 eV/amino acid residue when the peptide residue is neutral (acidic condition and non-polar step). Peptide residues that contain negatively charged amino acids (high pH, alkaline condition) are more stable along the  $Ca^{2+}$ polar step edge. The 12-amino acid long peptide chain with alternating glycine and alanine shows better parallel allignment.

Studies of mineral surface interactions with onedimensional and two-dimensional peptides indicate the dependence of structural matching and parallel alignment as principle criterion for inorganic nucleation on organic template. In order to understand the nucleating mechanism of inorganic mineral surfaces on organic templates during biomineralization, we are studying the growth and nucleation of calcium carbonates on three-dimensional peptide-chain networks.

## Reference

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