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EGON T. DEGENS, JÓZEF KAŹMIERCZAK and VENUGOPALAN ITTEKKOT

CELLULAR RESPONSE TO Ca^{2+} STRESS AND ITS GEOLOGICAL IMPLICATIONS

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Knowledge on transport and regulation of free calcium in the living cell is used in support of the theory (Kaźmierczak *et al.* 1985) linking the onset of biocalcification at about the Precambrian/Cambrian boundary to a rise in Ca^{2+} concentrations in the shelf seas to levels toxic to biota. Following this event, fluctuating Ca^{2+} levels in the Phanerozoic seas are supposed to have challenged a variety of protists and invertebrates to respond by depositing no, thin, or thick skeletons respectively. Changes in type and extent of calcification, as observed in the stratigraphical record, are interpreted to reflect the pulsating flow of Ca^{2+} ions through crust, sea, and biota. Some implications of that theory to (i) the history of sea water, (ii) the global carbon cycle, (iii) stable carbon isotope geochemistry, and (iv) sedimentation of suspended clays, are briefly discussed.

K e y w o r d s: Cell physiology, biomineralization, calcification, calcium geochemistry, carbonate and clay sedimentation.

Egon T. Degens, Venugopalan Ittekkot: SCOPE/UNEP International Carbon Unit, University of Hamburg, Bundesstrasse 55, D-2000 Hamburg, Federal Republic of Germany; Józef Kaźmierczak, Zakład Paleobiologii, Polska Akademia Nauk, *Al. Żwirki i Wigury 93, 02-089 Warszawa, Poland.* *Received: February 1985.*

INTRODUCTION

Carbonates are extracted from modern ocean principally by the action of organisms. The way how they do it is still debatable. In 1962 it was said that "the nature of the local mechanism of calcification is one of the most important unresolved problems in biochemistry" (Urist 1962). In the years inbetween, research has advanced to a point that we understand by now the general pattern of calcification, but the local details are still missing (for reviews see: Degens 1976; Krampitz and Witt 1979; Westbroek and de Jong 1983).

Fossil products of biocalcification, i.e. shells, bones, teeth, are critical elements, for example, to reconstruct the path of organic evolution, to

outline paleogeographic settings, to determine type and dynamics of former ecosystems, or to time geologic events. Thus, geoscientists should be interested to know *why* organisms deposit carbonate skeletons and *what* cellular and environmental mechanisms drive organisms to deposit calcite, aragonite or Ca-phosphate (apatite). Such a knowledge could assist to unravel the mystery behind the first appearance of calcareous fossils at the end of the Precambrian era.

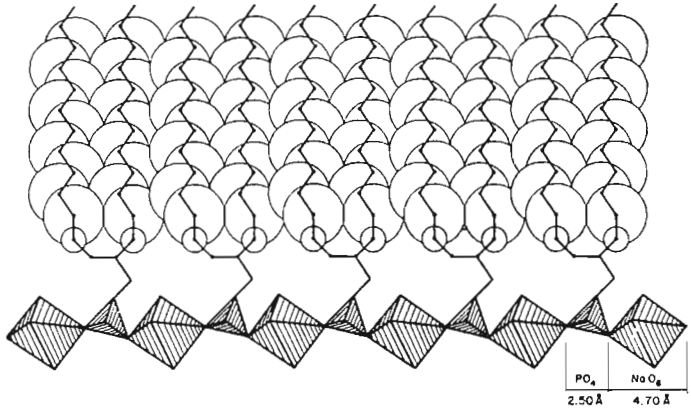
Ca^{2+} REGULATION

Eukaryotic calcification is under strict cellular control and part of the biological calcium cycle. Ca^{2+} is a ubiquitous ion at virtually all strategic sites of an organism. The question has often been asked why calcium was chosen in first place to occupy such a central position in the metabolism of the eukaryotic cell (Degens 1976; Williams 1976; Carafoli and Crompton 1978; Erulkar 1981). It is the structure of the calcium ion which can perfectly adjust to fit all kinds of physiological assignments. This is principally due to high affinity of calcium for oxygen ligands in complex biochemical molecules, a characteristic unsurpassed by any other of the common chemical elements. Work tasks for Ca^{2+} range from structural to functional and the essentials are briefly mentioned.

Structural aspects

The molecular geometry of phospholipid membranes bears striking resemblance to inorganic phosphate minerals in that in membranes and minerals corrugated layers of metal ion-phosphate coordination complexes exist. Fig. 1 (top graph) depicts a phospholipid membrane cross-section composed of double aggregates of hydrocarbons and oxygen polyhedra. To reveal the type of molecular pattern that is exposed at the ionic surface of phospholipid, the three-dimensional structure of two typical phosphate minerals is shown (fig. 1) exhibiting a sheet of phosphate-cation

Fig. 1. (top) Schematic phospholipid membrane cross-section composed of double aggregates of hydrocarbons and oxygen polyhedra. The surface area of two hydrocarbon chains is about 40 $Å^2$ which is identical to the surface area occupied by a tetrahedron-octahedron unit. The 40 Å periodicity observed in biological membranes is a result of this structural design; (center) presentation of molecular structure of a sheet composed of phosphate-cation chains cross-linked into a planar pattern. The corrugated layers are simplified. In reality, Ca^{2+} is coordinated with 8 oxygens and not with 6; (bottom) molecular structure of vivianite $Fe_3(PO_4)_2 \cdot 8\,H_2O$. Phosphate tetrahedra link single and double octahedral groups. Note the space geometry of the open channels in center and bottom graph which is controlled by the type of metal ion and coordination number (from Matheja and Degens 1971).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NaO_6
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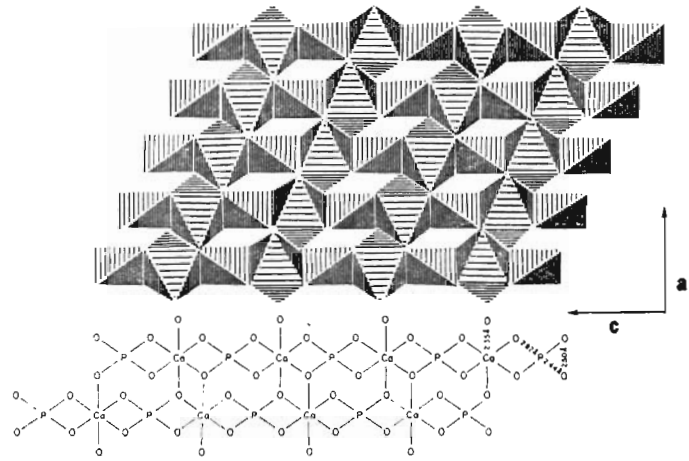
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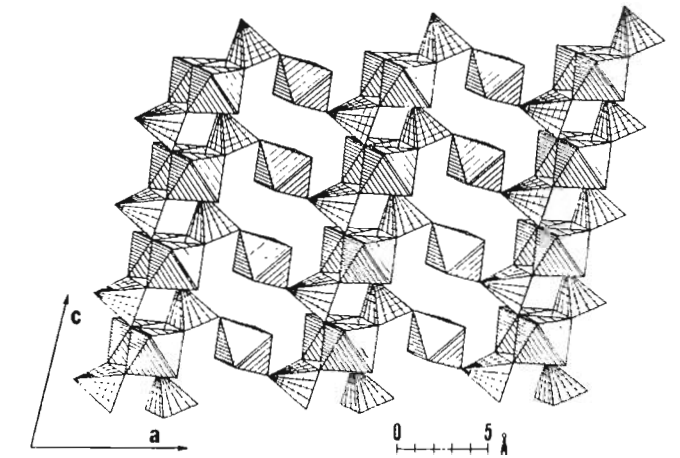
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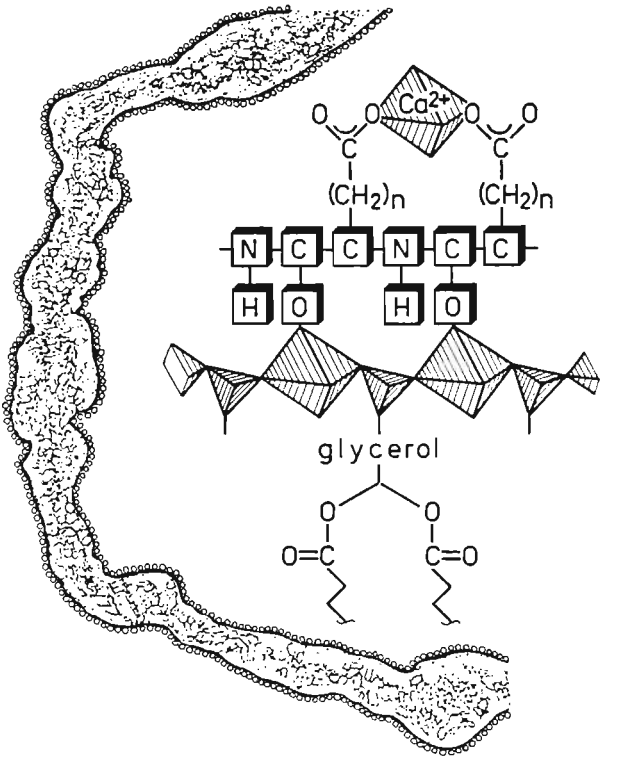


Fig. 2. Graph drawn after an electromicrograph of a beef-heart mitochondrial cross-section. Outer grains represent structural proteins. The black lines below the protein grains reveal the position of the phosphate-metal ion oxygen polyhedra which obey a 40 Å periodicity. Glycerol and double aggregates of hydrocarbons joined to the crystalline tetrahedron-octahedron surface occupy the space in between. Structural relationships between principal molecular units of a phospholipid membrane are depicted in the center of the figure. For graphical reasons only a cross-section through a single layer is shown and the hydrocarbon chains are only partly drawn. The way Ca^{2+} is fixed to carboxylate groups at the internal face of membranes is schematically presented.

chains crosslinked in a planar pattern. Note the open channels between the tetrahedra-octahedra assemblages. The ionic surface of phospholipid membranes is overlain by a protein layer which is coordinated by the sharing of oxygens to the ionic surface. Fig. 2 shows an electromicrograph of a beef heart mitochondrial cross-section next to which structural details of a phospholipid bilayer are schematically drawn.

It is by now well established that the dispositions and functions of membrane proteins are the critical element in calcium transport and regulation. Binding of Ca^{2+} ions to specific sites at the internal face of membranes alters conformation of some membrane constituents, thus allowing transfer or passage of hydrophilic substances across the lipid barrier. Membrane transport of a sugar carrier such as glucose is thus mediated by means of change of membrane permeability (Elbrink and

Bihler 1975). This transport is characterized by Michaelis-Menten saturation kinetics, chemical specificity and restructuring of substrate and membrane components rather than by simple diffusion processes. The cytoplasmatic calcium level is affected by Ca^{2+} and Na^{+} exchange in the endoplasmic reticulum and by excitation-contraction coupling in the sarcoplasmic reticulum system which supplies Ca^{2+} for muscle activities. Furthermore, rapid mitochondrial oxidation may deplete the regulatory Ca^{2+} pool which in turn is recharged by means of a feedback system from storage sites inside the cell or through some form of energy-dependent "ion pump" system (Takeichi and Okada 1972; Elbrink and Bihler 1975; Knudsen and Horwitz 1977; Rasmussen 1981; Llinas 1982). The close association of Na^{+} and Ca^{2+} in the "ion pump" system could be a consequence of similar ionic radii (close to 1.0 Å) and high affinity of both ions to oxygen in octahedral coordination.

The way Ca^{2+} is fixed to carboxylate groups exposed at the hydrophobic site of the membrane protein is schematically presented in fig. 2. Ca^{2+} can open or close off communication channels through the restructuring of the membrane fabric. The interchangeable nature of metal ions causes membranes to act as dynamic molecular sieves. Pore size and sieving qualities are variable and depend on metal ions regulated by enzymes. Assuming ATP traps a metal ion, such as Ca^{2+}, aperiodic pulsation of the lattice will take place. Such oscillations are known from mitochondria (Chance and Yoshioka 1965). The surface lattice of membranes acts on the phospholipid molecules in a manner similar to a zone melting and refining process. It can be considered a two-dimensional coding device for bonded proteins, whereby Ca^{2+} serves as a messenger. Permeability is achieved by placing the Ca^{2+} at opening gates of the membrane structure where it acts as modulating factor for material flux, energy transfer and excitability (Matheja and Degens 1971).

Functional aspects

Ca^{2+} serves as an important cellular regulator. Its message is relayed by a series of high-affinity calcium binding proteins of which calmodulin, troponin C (TN-C), and parvalbumin are three common species (e.g., Collins *et al.* 1973; Drabikowski *et al.* 1974; Kendrick-Jones 1974; Tufty and Kretsinger 1975; Kretsinger 1976; Cheung 1980, 1982).

It was suggested that many proteins involved in calcium transport and regulation are homologous and would contain one or several "EF-hands". The ubiquity of EF-hands may simply reflect a thermodynamically preferable conformation. Fig. 3 illustrates the basic structural elements of the EF-hand. Comparison of EF-hand amino acid sequences in several high-affinity calcium binding proteins reveals the significance of the

carboxylate groups for coordination purposes. In the case of troponin C (TN-C), 17 of 24 oxygens needed for the four Ca^{2+} octahedra that can become part of one TN-C molecule come from carboxylate groups. The predominance of aspartic acid is noteworthy (Collins *et al.* 1973; Tufty and Kretsinger 1975).

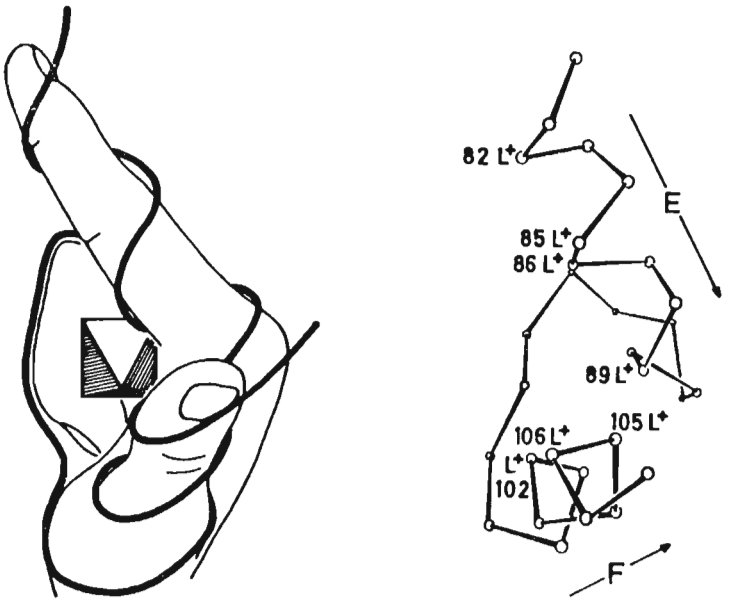


Fig. 3. The symbolic EF hand on the left represents helix E (forefinger), the calcium binding loop (middle finger enclosing a $Ca^{2+}O_6$ octahedron) and helix F (thumb). On the right the carbon skeletal model of a high-affinity Ca^{2+} binding parvalbumin is depicted (from Tufty and Kretsinger 1975).

In conclusion, the presence of carboxylate groups at specific coordination sites appears to be the determining control mechanism for the selective and reversible binding of Ca^{2+} to proteinaceous membrane surfaces or to calcium binding carrier molecules. Free calcium, by itself, has no regulating power. Only the moment Ca^{2+} interacts with the protein will an active complex be generated.

Excretion

Such an elaborate system, where Ca^{2+} has to be present at the right spot, in the correct concentration, and at the proper time requires, to prevent Ca^{2+} stress, a valve system for a fast removal of calcium ions in case a critical level is exceeded. The Golgi apparatus serves in this capacity. The Golgi complex is in a constant state of flux. Cisternal membranes arising from the rough endoplasmic reticulum change to smooth endoplasmic reticulum and become the Golgi cisternae (North-

cote 1971). Those cisternae then break down to vesicles which can fuse with and extend the plasmalemma. Therefore, the Golgi apparatus may be viewed as a membrane transformer from the endoplasmic reticulum to the cell membrane. During this irreversible process proteins are sorted, refined, and delivered to multiple destinations.

Among the numerous functions the Golgi exercises in the eukaryotic cell (Rothmann 1981) its responsibility for excretion, including the removal of excess Ca^{2+} from within the cell to the outside environment, has a direct bearing on the mechanism of calcification. A high-affinity calcium binding protein serves as a carrier for surplus Ca^{2+}. The protein becomes solubilized by the addition of glucuronic acid or a similar acting sugar (Degens 1976). The newly assembled glycoprotein complex is now ready for shipment (excretion) to the outside environment.

CALCIFICATION

Everything would be alright, if the aquatic habitat would follow the concept: "The solution to pollution is dilution". Yet, in a wide variety of invertebrates, protein sheets positioned at or close to the cell walls interact with the Ca^{2+}-charged complex and carbonate deposition is initiated. The mechanism is as follows.

The outer protein sheet of a silk-type nature develops hydrogen bonding to the hydrophilic side of the glycoprotein complex following the removal of the glucuronic acid by means of a protease. The now freely exposed hydrophobic side composed principally of carboxylate groups derived from aspartic acid will atract additional calcium which becomes structured in the form of $Ca^{2+}O_6$ or $Ca^{2+}O_9$ coordination polyhedra. $CaCO_3$ deposition is mediated by carbonic anhydrase which functions as a kind of "chemical homeostat" by releasing calcium from cellular depots and by activating carbonate from the aqueous medium. The role of carbonic anhydrase in calcification does *not* rest on its ability to hydrate CO_2, but to remove carbonic acid from the site of calcification (Goreau 1959):

$$Ca^{2+} + 2\,HCO_3^- \rightarrow Ca(HCO)_3 \rightarrow CaCO_3 + H_2CO_3.$$

Expressed differently, the substrate of carbonic anhydrase is the neutral H_2CO_3 molecule (Koenig and Brown 1972). As long as calcium is not the limiting factor, the rate of formation of calcium carbonate will depend on the rate by which carbonic acid is carried off from the calcification site. Since the overall crystallisation of $CaCO_3$ will liberate 2—4 kcal/mole, there is no energy problem in $CaCO_3$ deposition. In the presence of carbonic anhydrase inhibitors, carbonate formation slows down and eventually ceases (Goreau 1963).

The structural relationships that arise during calcification are briefly described. The attachment of the Ca^{2+} carrier to a protein template will activate the mineralizing substrate and calcium carbonate crystals are laid down in epitaxial order. Crystals are nucleated on stereochemically analogous faces when proteins are adsorbed onto a rigid substrate (Addadi and Weiner 1985). Crystal growth terminates upon secretion of a newly formed Ca^{2+} carrier protein on the growing crystal surface, whereby a kind of organic blanket is formed. Subsequently, the protein template becomes attached to the hydrophilic side of the Ca^{2+} carrier and a sandwich structure is produced (fig. 4). The cycle can start again. Thousands of layers can be generated during the life time of an organism (e.g. in a mollusc shell). Fig. 5 depicts a vertical cross-section of alternating layers of the two types of proteins.

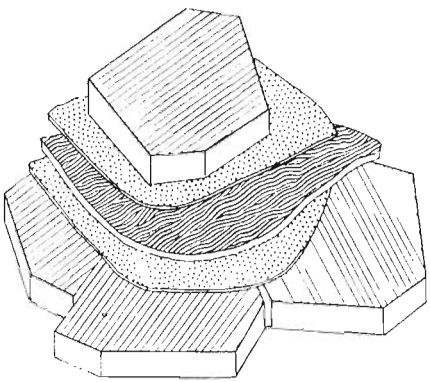


Fig. 4. Schematic representation of structural relationship between layers of silk-type template (banded), high-affinity Ca^{2+} binding protein (dotted) and $CaCO_3$ (block-shaped) (from Degens 1976).

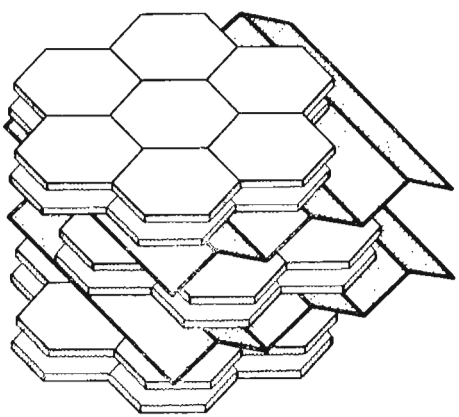


Fig. 5. Schematic representation of vertical cross-section showing alternating layers of high affinity Ca^{2+} binding proteins (hexagons) and silk-type template (pleated sheet conformation); the $CaCO_3$ has been omitted for graphical reasons (from Degens 1976).

The key role Ca^{2+} exercises in cellular regulation is in the last instance the reason behind calcification. Biocalcification has been probably forced onto the evolving eukaryotic cell as a consequence of the build-up of Ca^{2+} in the ancient sea (Kaźmierczak *et al.* 1985). It was principally invented not for storage of calcium, protection against predators or environmental hazards, but is a byproduct of excretion of excess Ca^{2+} from within the cell. That the organisms took later *advantage* of it, is a different story.

BIOGEOCHEMICAL IMPLICATIONS

$CaCO_3$ forms principally along two routes: (i) a chemical one:

$$Ca^{2+} + 2HCO_3^- \rightleftharpoons CaCO_3 + CO_2 + H_2O$$

and (ii) an enzymic one which instead of CO_2 and H_2O yields H_2CO_3

$$Ca^{2+} + 2\,HCO_3^- \rightarrow Ca(HCO_3)_2 \rightarrow CaCO_3 + H_2CO_3.$$

The carbonic anhydrase mediated $CaCO_3$ deposition is restricted to the eukaryotic cell and came into existance with the onset of biological calcification at about the Precambrian/Cambrian boundary. It is emphasized that microbially induced carbonate deposition of the type associated, for example, with stromatolites of Precambrian or Phanerozoic age is strictly chemically controlled. This has interesting consequences for carbon isotope geochemistry (Degens *et al.* in press) because a chemically precipitated $CaCO_3$ will form in isotopic equilibrium making the solid phase 4 per mil heavier than the starting bicarbonate. Expressed differently, a common $\delta\ ^{13}C$ value of -1 per mil for Precambrian limestones (Schidlowski 1982) would signal a -5 per mil for the former marine bicarbonate pool. In contrast, the majority of skeletal carbonates have a $\delta\ ^{13}C$ identical to its sea water bicarbonate source. Only a few species exist where some contribution of isotopically light respiratory CO_2 is indicated. The approximately 2 per mil drop in $\delta\ ^{13}C$ at about the Vendian time could principally be related to a switch from chemical to enzymatic carbonate deposition.

As mentioned above, the first calcification events during Vendian are considered the response of the eukaryotic cell to higher Ca^{2+} levels in the ancient sea. The question comes up why has the Ca^{2+} pressure not been felt by the organisms earlier in time? We believe that the answer rests in the "peculiar" chemistry of the Precambrian sea. It was suggested (Degens *et al.* 1984; Kempe and Degens 1985) that not rock salt but soda was the major solute in the early ocean. A general chemistry similar to that presently found in lakes from volcanic regions is envisioned. Lake Van, the largest soda lake on Earth is regarded the prototype of

a primordial sea (Kempe 1977). Soda lakes contain little Ca^{2+}, because residence time of calcium is extremely short, due to its fast removal from the water column in the form of aragonite, calcite or dolomite depending on Mg/Ca ratio. Soda lakes are highly alkaline with a pH in the range of 9.5 to 10.5. This has interesting consequence for the fate of CO_2 in the early atmosphere. In a matter of less than 100 million years, all carbon in the crust, if released in the form of CO_2, would be consumed by weathering and could readily be accomodated as dissolved carbonates in a soda ocean (Kempe and Degens 1985). Only a moderate amount of CO_2 would remain airborn and PCO_2 would be at most a few times higher than today.

In the course of the Precambrian, the initial alkaline sea became titrated with acid volatiles and pH dropped down to about 8 (fig. 6).

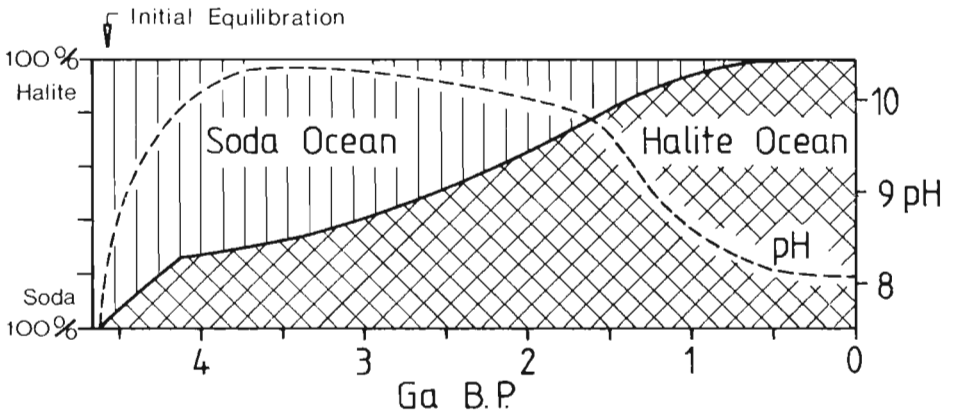
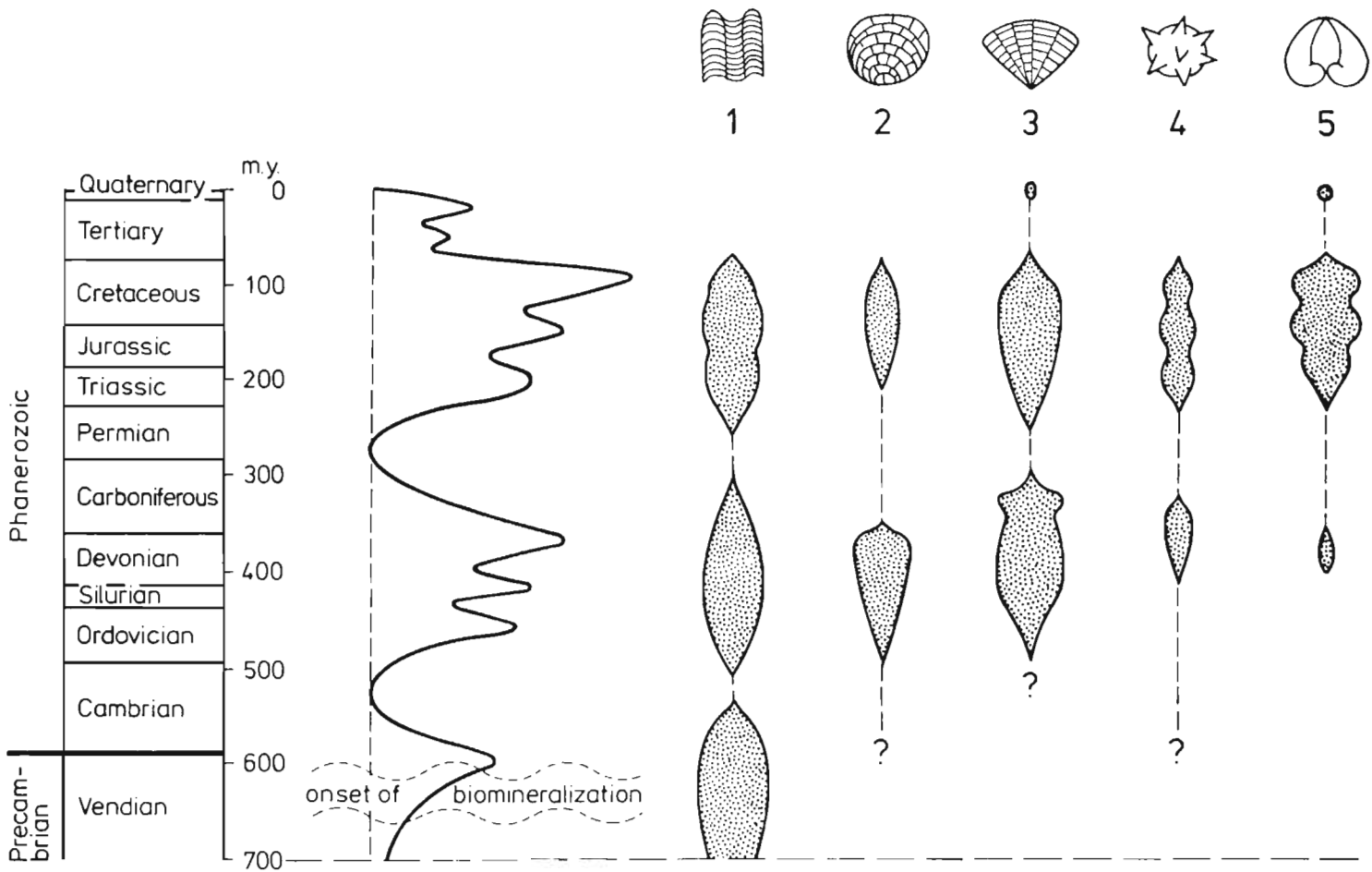


Fig. 6. Evolution of ocean chemistry during geologic time (pH-curve deduced from total alkanity and pH-relation of present-day soda lakes (from Kempe and Degens 1985).

This marked the time when water chemistry shifted from soda to sodium chloride dominance, and the dissolved carbonate system changed from sodium to calcium-magnesium regulation. Ca^{2+} started to increase and when a critical point was reached where the eukaryotic cell had to switch on the Golgi for the excretion of excess Ca^{2+}, skeletal minerals

Fig. 7. Curve of relative abundance of marine carbonate sediments during the Phanerozoic (compiled from various sources) compared with stratigraphic distribution and frequency of selected groups of organisms and biosedimentary structures considered as good indicators of a calcium intoxicated environment. 1 — calcareous stromatolites in general, 2 — stromatoporoid stromatolites: subtidal, in situ calcified coccoid cyanobacterial mats typical particularly for lower Palaeozoic carbonate platforms, 3 — sclerosponges: siliceous sponges (Demospongiae) with additional basal calcareous skeleton; typical fossil representatives are favositids and chaetetids, 4 — calcispheric structures: mostly post mortem calcified phytoplankters like acritarchs or volvocacean algae or calcified resting cysts of dinoflagellates abundant in protected areas of past carbonate platforms, 5 — thick-shelled molluscs (e.g., megalodontid and rudist bivalves or nerineid gastropods) (from Kaźmierczak *et al.* 1985)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Tertiary
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Triassic
Permian
Carboniferous
Devonian
Silurian
Ordovician
Cambrian
Vendian
Phanerozoic
Precam-brian
onset of biomineralization
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started to form and an almost spasmodic evolution of the skeletonized biota began.

Massive marine limestone deposits observed at certain times during the Phanerozoic are in all likelihood linked to an effective recharge of Ca^{2+} to the sea through riverine and hydrothermal channels and its concentration along shelf margins (Kaźmierczak *et al.* 1985). This caused a number of shallow water organisms to calcify (which otherwise didn't) and others to lay down thicker skeletal deposits. In fig. 7, the relative abundance of five fossil groups indicative for Ca^{2+} stressed marine environments are illustrated: (1) calcareous stromatolites, (2) stromatoporoid stromatolites, (3) sclerosponges, (4) calcispheres, and (5) thick-shelled molluscs. All exhibit the same distribution pattern and good correlation with maximal extensions of Phanerozoic carbonate facies. The Phanerozoic limestone facies developed mostly as so-called carbonate platforms or ramps which are considered comparatively stable off-shore shallow marine habitats in an epicontinental sea comparing with on-shore settings. Of particular interest are calcispheres, a heterogenous group of phytoplankters (volvocaceans, acritarchs, dinoflagellates) mineralized not *in vivo* but only *post mortem* (Kaźmierczak 1976). They occurred at Ca^{2+} peaks and concentrated excessive calcium in their extracellular excretes (chelants) which after cell death calcified immediately close the sediment/water interface. Fig. 8 depicts the same forms of acritarchs in an unmineralized state from calcium depleted black shale facies and in $CaCO_3$ encrusted "calcispherized" state from carbonate facies.

Ca^{2+} stress exercised by the environment on the evolving organisms eventually reached a stage that even unicellular eukaryotes started to calcify. The emergence of calcareous nannophytoplankton and of planktic foraminifers during the Jurassic may be viewed as a response to this challenge. In the Cenozoic, areal reduction of carbonate platforms left the burden of Ca^{2+} removal to the pelagic plankton community. This is precisely the situation we are left today.

The fact that even today many normally calcifying organisms stop $CaCO_3$ deposition at times of environmental stress (e.g. Lutz and Rhoads 1977) or during active stages when Ca^{2+} is in high demand (e.g. Hemleben *et al.* 1979) may serve as a reminder that calcification ranks in low priority for the metabolism of the organism and is just the cellular response to an environmentally induced Ca^{2+} stress.

NON-CALCIFYING ORGANISMS

One aspect of cellular Ca^{2+} regulation has not been mentioned so far: What do non-calcifying organisms do when subjected to a Ca^{2+} stress? The answer is: the same! They secrete glycoproteins, coordinate Ca^{2+}

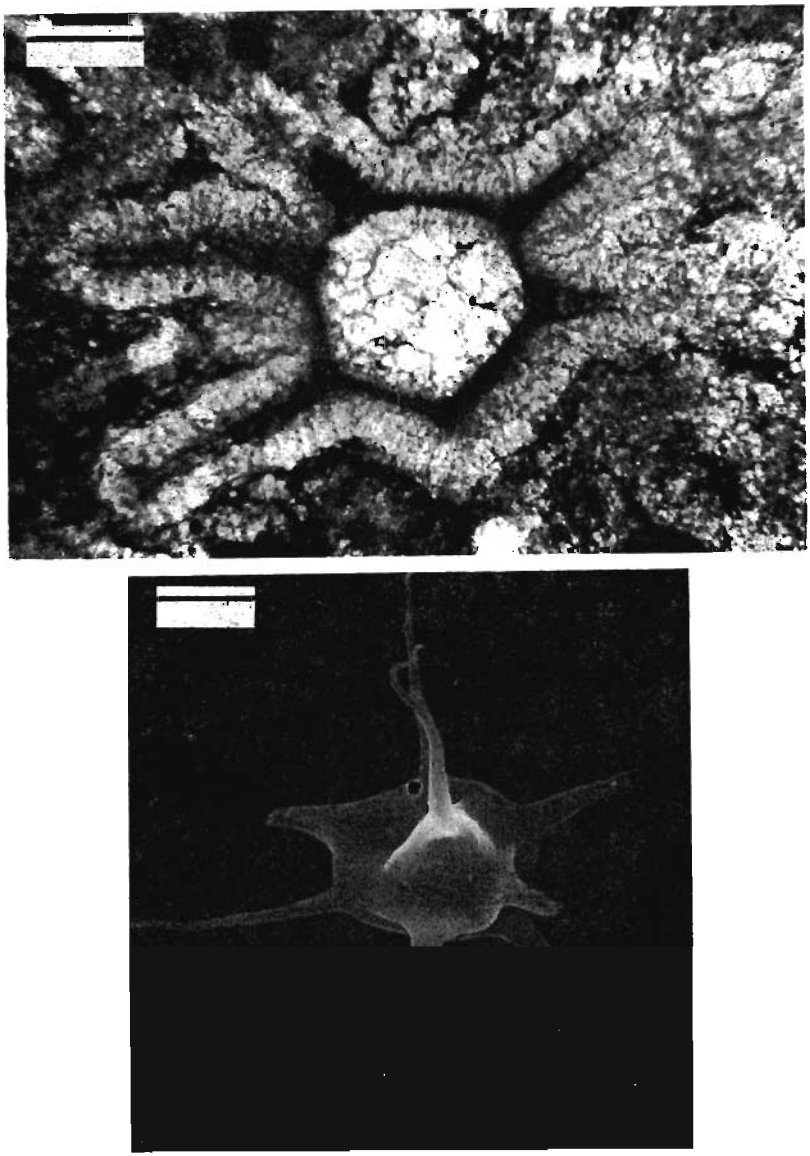

Fig. 8. Examples of variously preserved acritarch phytoplankters (*Baltisphaeridium* group) depending on basically different calcium availability in their life and burial environment; (top) organic-walled specimen from calcium depleted environment (Chagrin Shale, Upper Devonian, Ohio; courtesy Dr. E. R. Wicander); bar scale = 10 μm; (bottom) heavily calcified specimen with poorly preserved organic wall from calcium stress environment (Frasnian carbonate platform, Upper Devonian, southern Poland); bar scale = 50 μm.

and excrete a high affinity Ca-binding protein, except that there is no proteinaceous cell wall template to hold back the dissolved complex.

In a city lake, the Hamburg Alster, an about 90-fold supersaturation with respect to calcite existed at the time of sampling. We examined

the molecular nature of the excretion products released to the environment during a non-calcareous algal bloom (Michaelis *et al.* 1976). Of the total proteinaceous material excreted by the plankton about 90% consisted of aspartic acid attesting to the universiality of this amino acid for Ca^{2+}-fixation. In such a situation roughly half of the daily photosynthesized organic matter ended up as dissolved organic carbon which because of its high functionality would readily combine with clay minerals forming macroflocs a few hundred microns in size. These settled almost instantaneously to the sediment-water interface, yielding an organic-rich mud.

Similar interaction occur in the open sea and a variety of organic excretion products have been reported (Anderson and Zeutschel 1970; Thomas 1971; Hellebust 1974; Williams 1975; Fogg 1983). It is noteworthy that clay minerals on their own would almost never reach the sea floor should they only rely on Stoke's Law. The physical settling velocities of clays are in the order of a few cm per day. Convection in the euphotic zone measuring a few meter per day would thus keep clays in suspension. However, when aggregated to macroflocs (Kranck and Milligan 1980; Honjo 1982), or packaged in the form of fecal pellets, after being eaten by zooplankters (Schrader 1971; Honjo 1976; Roth *et al.* 1975; Honjo and Roman 1978), clay aggregates will settle at rates of a few hundred meters per day (fig. 9). Material collected in sediment traps in mid-water at depths of several thousend meters demonstrate that clay-sized fractions are removed from the surface layer via macroflocs or fecal pellets. There are strong seasonal and interannual variations which are directly related to the pulses of planktic activity in the sea (Deuser and Ross 1980; Deuser *et al.* 1981, 1983; Honjo 1982).

Interestingly, this transport mechanism affects not only the vertical transport of particles. Horizontally transported particles in mid-water are also found to be translated into a vertical flux (Honjo 1982). It has also been suggested that sinking macroaggregates will scavange particles suspended in the water column (Deuser *et al.* 1983) increasing the abiogenic material flux to deep ocean (Honjo *et al.* 1982).

In ancient environments devoid of such mechanisms before the advent of zooplankton and organic matter secreting planktic algae, sedimentation processes must have been significantly different and more in agreement with Stoke's Law. Higher residence times for particles will be the result and as a consequence, more intense particle-seawater interactions should occur. In such an ocean, dissolution or ion-exchange of silicate minerals with seawater column (Tsunogai *et al.* 1973) would have been an efficient source for dissolved Ca^{2+}. Conditions similar to this were probably repeated also in the Phanerozoic during periods of large scale extinctions of marine planktic biota. At those times, the terrestrial detritus content in the ocean would have been enormous

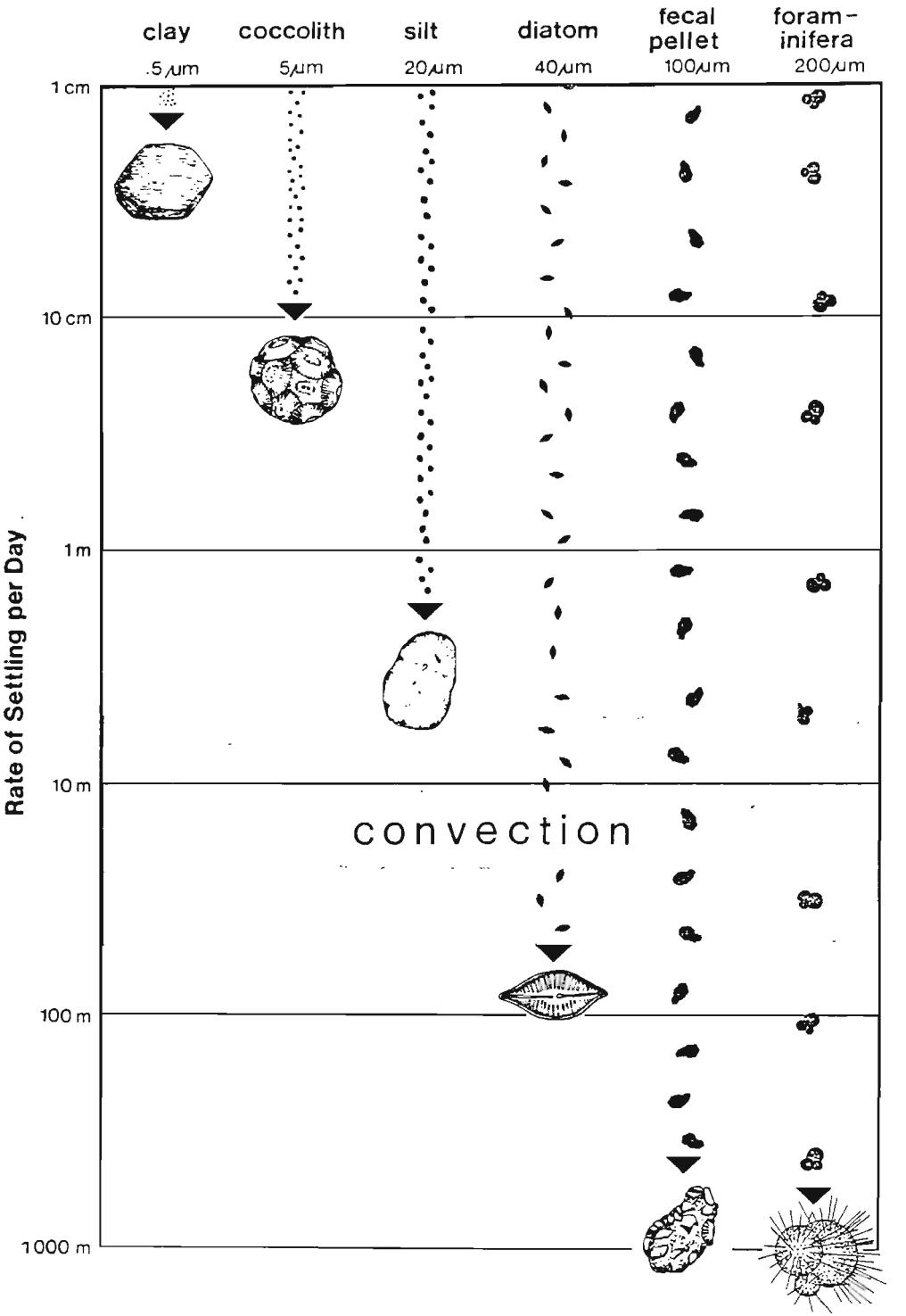

Fig. 9. Settling rates per day of materials in various size fraction in the marine environment (from Degens and Ittekkot 1984).

because of the absence of efficient and rapid transfer mechanisms such as fecal pellets and macroaggregates. Only particles with high specific density, like volcanic and cosmic dust may be expected to sink rapidly towards the seabottom leading to geochemical anomalies.

Macroaggregate formation induced by calcium specific organic matter will also lead to the clustering of cells. It has been found that polysaccharide-rich excretion products of coccolithophorids are significant in the formation of cell macroaggregates (Ittekkot *et al.* 1984). It is conceivable that such macroaggregate formation could have been instrumental in the early steps of multicellular evolution (Kempe *et al.* 1986).

CONCLUSIONS

Ca^{2+} in the sea is derived from the principal sources: (i) riverine run-off, and (ii) hydrothermal solutions emanating from the oceanic crust. For most of the Precambrian removal of Ca^{2+} was principally accomplished by carbonate precipitation in tidal and supratidal environments (e.g., in the form of stromatolites, dolostones) or in the vicinity of rifts. Seawater chemistry at that time, that is a "soda ocean", forced Ca^{2+} to precipitate close its marine inlets.

With the advent of a NaCl dominated ocean, somewhere during the Proterozoic, Ca^{2+} level in sea water could rise and the dissolved carbonate chemistry became governed by $Ca^{2+} - Mg^{2+}$ mineral equilibria. Organisms responded to this Ca^{2+} challenge later in the Precambrian by the formation of multicellular aggregates and eventually in Vendian time through skeletonization.

The biocalcification event changed carbonate deposition from a chemical to a predominantly enzymic process. In accordance, the extraction of Ca^{2+} from the Phanerozoic sea became the domain of organisms.

Changes in scale of calcification as seen in weight and morphology of shell structures are in all likelihood intimately connected with tectonic events which supplied Ca^{2+} directly through hydrothermal channels or indirectly via erosion to the marine environment.

Non-calcifying marine protists, e.g. the nannoplankton, respond to Ca^{2+} and other metal ion stress by secretion of complexing molecules such as polysaccharides or glycoproteins. Such complexing agents interact with suspended particles and lead to macrofloc formation. Sedimentation of clay-sized suspensions in the open marine habitat is thus mediated by the activity of planktic organisms. Thus, in the final analysis, marine limestones and shales are products of organic-inorganic interactions.

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EGON T. DEGENS, JÓZEF KAŹMIERCZAK, VENUGOPALAN ITTEKKOT

GEOLOGICZNE KONSEKWENCJE REAKCJI KOMÓRKI NA STRES WAPNIOWY

Streszczenie

Ca^{2+} jest jednym z najważniejszych jonów w procesach życiowych, pełniącym, między innymi, różnorakie funkcje jako regulator wewnątrzkomórkowy. Przeciętne stężenie Ca^{2+} w cytoplazmie wynosi 10^{-8}—10^{-7} M i jest zwykle o 4—5 rzę-

dów wielkości niższe od stężeń zewnątrzkomórkowych. Wysokie stężenia jonów wapnia w sąsiedztwie komórki powodują zwiększony napływ Ca^{2+} do cytoplazmy, zagrażający normalnemu funkcjonowaniu organizmu. Do usuwania toksycznego nadmiaru Ca^{2+} z komórki służą różnego rodzaju „pompy jonowe" oraz nośniki białkowe (tzw. kalcyproteiny). Biokalcyfikację można rozpatrywać jako jedną z form detoksykacji wapniowej komórek. Różni się ona od innych sposobów usuwania nadmiaru Ca^{2+} z komórek tym, że w tym przypadku wyprowadzany na nośniku białkowym (glikoproteinowym) wapń jest przy współudziale enzymów (węglanowej anhydrazy lub alkalicznej fosfatazy) neutralizowany do postaci słabo rozpuszczalnej w wodzie soli, której ostateczna forma mineralogiczna zależy od specyficzności wzornika (template) białkowego, na którym epitaksjalnie narastają kryształy.

Omówione w pracy dokładniej mechanizmy transportu i regulacji Ca^{2+} w komórce posłużyły do poparcia hipotezy Kaźmierczaka, Ittekkota i Degensa (1985), łączącej powstanie pierwszych struktur szkieletowych na przełomie prekambru i kambru ze wzrostem stężeń Ca^{2+} w szelfowych środowiskach morskich do poziomu toksycznego dla zasiedlających je organizmów. Zgodnie z tą hipotezą, w ciągu fanerozoiku koncentracja Ca^{2+} w morzach szelfowych ulegała wielkoskalowym fluktuacjom, odpowiadającym cyklom regresywno-transgresywnym, sterowanym aktywnością stref ryftowych. Rezultatem tych fluktuacji byłoby okresowe, masowe pojawianie się w prawie wszystkich grupach organizmów morskich form opatrzonych szczególnie masywnymi, wapiennymi lub Ca-fosforanowymi strukturami szkieletowymi.

Ca^{2+} w dzisiejszych morzach pochodzi z dwóch głównych źródeł: (1) produktów wietrzenia lądowego, znoszonych wodami rzecznymi i (2) roztworów hydrotermalnych, wydobywających się ze skorupy oceanicznej w strefach ryftowych. Przez większość prekambru usuwanie Ca^{2+} ze środowiska morskiego następowało przede wszystkim przez chemiczne wytrącanie węglanów w strefach pływowych i nadpływowych (np. w formie stromatolitów), lub w pobliżu ryftów. Skład chemiczny wody morskiej w tym czasie zbliżony był do składu dzisiejszych przyryftowych i wulkanicznych jezior sodowych, których wysokie pH (>10) pozwala utrzymać w roztworze tylko znikome ilości Ca^{2+}. Z przejściem od oceanu sodowego do chlorkowego (halitowego), co miało miejsce w ciągu proterozoiku, poziom Ca^{2+} w wodzie morskiej stopniowo wzrastał, szczególnie w zbiornikach epikontynentalnych, zasilanych wraz z postępującą kratonizacją litosfery coraz większymi ilościami Ca^{2+} pochodzącego z wietrzenia kontynentów. Reakcją organizmów na wzrastający stres wapniowy była prawie jednoczesna biokalcyfikacja wielu grup bezkręgowców w wendzie i wczesnym kambrze. „Wymuszenie" przez środowisko wapiennych struktur szkieletowych na organizmach nie było procesem ograniczonym do pogranicza prekambru i kambru. W ciągu fanerozoiku szereg grup organizmów bezszkieletowych odpowiedziało na stres wapniowy swoich środowisk życia wykształceniem wapiennych szkieletów (np. w dewonie otwornice bentosowe, zaś na przełomie triasu i jury planktonowe otwornice i część nannofitoplankterów). Biokalcyfikacja zmieniła generalnie charakter wytrącania węglanów w morzu

z chemicznego na enzymatyczny, a ekstrakcja Ca^{2+} w morzach fanerozoicznych stała się prawie wyłącznie domeną organizmów.

Wiele pierwotniaków i glonów planktonowych reaguje na stres wapniowy środowiska obfitym wydzielaniem wielocukrów czy glikoprotein kompleksujących Ca^{2+}. Obecność tych substancji w wodzie prowadzi z kolei do makroflokulacji drobnych cząstek osadu zawieszonych w wodzie, które bez łączenia się w większe agregaty nie mogłyby opadać na dno. Sedymentacja zawiesiny ilastej jest więc związana z intensywnością wydalniczą planktonu, zależną bezpośrednio od stężenia jonów wapnia (a także innych metali) w środowisku.

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