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The Effect of Ca Ions for Bacteria

Hüseyin Kahraman*

Department of Biology, Turkey

***Corresponding author:** Hüseyin Kahraman, Department of Biology, Faculty of Art and Sciences, Inonu University, Malatya 44280, Turkey

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ABSTRACT

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Calcium ions (Ca^{+2}) in eukaryotic cells have to; cell cycle, transport, mobility, gene expression and metabolism. Cells respond to various stimuli with transient changes in intracellular free Ca^{+2} concentrations. Indirect evidence indicates that Ca^{+2} also influence and such as spore formation, chemotaxis, heterosist differentiation, transport, and virulence. Many studies have shown that bacteria can maintain intracellular Ca^{+2} homeostasis. In addition, Ca^{+2}; nitrogen starvation, environmental stress and metabolites of carbohydrate metabolism. According to studies with *Escherichia coli*, *Bacillus subtilis* and *Pseudomonas aeruginosa*, it has been shown that expression of hundreds of genes is regulated by extracellular Ca^{+2} changes. Processes affected by these changes include, for example, swarming, type III secretion, polysaccharide production, iron uptake, quinolone signaling, and general stress responses. Many studies demonstrate that Ca^{+2} play a regulatory role in the physiology of prokaryotes. The mutation analysis of EF-el protein (EfhP) from *P. aeruginosa* appears to be necessary for the maintenance of intracellular Ca^{+2} homeostasis. Specifically, adenylate kinase and fructose-bisphosphate-aldolase have also been detected in association with Ca^{+2} regulations in eukaryotic organisms.

Bacteria Ca^{+2} are; membrane-bound structures. Agrobacterium tumefaciens and Rhodospiriullum rubrum are very similar to the acidoccalcisomas first described in eukaryotic organisms. Asidocalcisom on; other elements such as Ca^{+2}, Na^{+}, K^{+}, Mg^{+2}, Zn^{+2} and phosphorus (P) are acidic organelles that serve as the main storage for Ca^{+2} in pyrophosphate (PPi) and polyphosphate (poly P) form. In eukaryotic microbes, acidocalcisomes function in P metabolism, Ca^{+2} homeostasis, maintenance of intracellular pH and osmo regulation. Prokaryotic acidocalcisomes are very similar to eukaryotic structures that contain H^{+}-ATPase carriers and vacuum proton translocated pyrophosphatase responsible for their acidification. The ability of *R. rubrum* acidocalcisomes to store Ca^{+2} could be demonstrated by X-ray microanalysis when bacterial cells were grown at 100 mM $CaCl_2$. As a result, it has been suggested that acidocalcisomes may be involved in Ca^{+2} homeostasis in some bacteria. In Gram (-) bacteria, this region includes the region between the periplasmic space, the outer membrane and the cytoplasmic membrane.

The presence of this compartment was also detected in Gram (+) bacteria. X-ray mapping and electron loss spectroscopy showed that high concentrations of Ca^{+2} were related to the cellular structure of *E. coli*. The periplasma contains oligosaccharides and anionic proteins that may play a role in the storage of Ca^{+2} in this compartment. According to a study, the outer membrane and periplasm first serve as a barrier to Ca^{+2} entries. Secondly, it is thought to play a very important role by buffering and storing Ca^{+2}. By targeting the photoprotein aequorin using the N-terminal OmpT signal sequence, the periplasma has been shown to store Ca^{+2} in the periplasm of living *E. coli* cells ranging from 3-6-fold relative to the external environment. These results show us that bacterial cells; it supports our idea that it may have the ability to regulate Ca^{+2} concentrations by using different mechanisms within cellular separate compartments. However, the answer to this question is unknown today; to what extent do micro-regions in bacteria contribute to Ca^{+2} homeostasis? [1] this is one of the pending questions.

Ca^{+2} are a well-known signal molecule that regulates a number of basic processes in eukaryotes. Abnormalities in cellular Ca^{+2} regulations have been associated with bacterial infections such

as cystic fibrosis (CF) and endocarditic. All evidence; shows that intracellular and extracellular Ca^{+2} balances in a host can be a clue to opportunistic pathogenic bacteria and trigger their virulence. In addition, Ca^{+2} in prokaryotes; spore formation, mobility, cell differentiation, transport and virulence. It has also been shown that Ca^{+2} regulate bacterial gene expression and its regulatory role in prokaryotes. There is also increasing evidence that Ca^{+2} play a signaling role in the regulation of cellular Ca^{+2} in prokaryotes. The intracellular Ca^{+2} of various bacteria such as *E. coli, Propionibacterium acnes, Streptococcus pneumoniae, B. subtilis* and *Cyanobacteria* were maintained at μM levels and the Ca^{+2} effect was shown in response to environmental and physiological conditions. Such responses; Ca^{+2} regulated bacteria may play a key role in physiology and virulence. Some studies show that bacteria control [Ca^{+2}] using various mechanisms to transport or chelate Ca^{+2}. Some types of Ca^{+2} transport systems have been described in prokaryotes:

a) Gradient driven Ca^{+2} heat exchangers,

b) ATPase

c) Non-protein polyhydroxybutyratepolyphosphate (PHB-PP) channels.

It is also thought that Ca^{+2} modifiers are identified in some bacterial strains and serve as a main mechanism for Ca^{+2} transports in prokaryotes. These are low affinity Ca^{+2} carriers that used the stored energy of the electrochemical gradient of ions. They can work in both directions depending on the gradient. *P. aeruginosa* is an opportunistic human pathogen and is known to be one of the main causes of nosocomial infections and severe chronic infections in endocarditis and CF patients. Previously, growth in high Ca^{+2} has been shown to increase the formation of *P. aeruginosa* biofilm and induce biosynthesis of many secreted virulence factors including alginate, extracellular proteases and pyocyanin [2]. Ca^{+2} levels include hydrolytic modulation of the hydrolytic enzyme in *Pectobacterium carotovorum* produced in *V. cholerae*. In addition, Ca^{+2} in animal and plant hosts; as a secondary messenger, it regulates defense responses based on regulatory systems. Calcium, magnesium and iron are known to be involved in the process of *X. fastidiosa* infection.

However, it has also been suggested that in some cases (e.g. Ca^{+2} and Mg^{+2}), these elements are non-specific and act as bridges of adhesion between negatively charged bacterial cells and xylem vessels. The presence of Ca^{+2}; increases biofilm formation, cell binding and mobility under *in vitro* conditions. Ca^{+2} levels also affect biofilm production. Bivalent cations containing Ca^{+2} and Mg^{+2} have previously been shown to play a role in the formation of biofilm by different bacteria. In *Erwinia carotovora*, high Ca^{+2} levels suppress the expression of PehA, an endopoliglaklacronase, one of the major virulence determinants. The opposite effect, ie increase in virulence, was observed in *P. aeruginosa*. Here, with the addition of Ca^{+2}, increased production of extracellular proteases and increased expression of alginate biosynthetic genes, which are the main components of extracellular matrix, also affected the increase of biofilm thickness. *X. fastidiosa* has been shown to play a role in the regulation of biofilm formation, its effect on the cell surface and twitch mobility. Some studies; it also shows that the addition of Ca^{+2} significantly increases the surface binding strength of the cells. In addition to the "bridging" effect that may occur with Ca^{+2}, it has been shown that in addition to the effect of Ca^{+2} on biofilm and movement, a metabolic dependent effect may be responsible [3].

In addition to their role in biofilm matrix stability, Ca^{+2} can also affect bacterial gene expression. In eukaryotic cells, Ca^{+2} is an important signaling molecule and can play a regulatory role in bacteria. Many bacteria; *P. aeruginosa* PA4107 also includes genes for calmodulin-like proteins with characteristic EF hand motifs, including PlcR, possibly shown to bind Ca^{+2}. The secretion and stability of some extracellular proteins in *P. aeruginosa* are influenced by Ca^{+2}. The toxins secreted by the Type III secretion system are suppressed by Ca^{+2}. The amounts of extracellular elastase (LasB) and LasA secreted by Type II secretion increase in the presence of additional Ca^{+2}. It is also known that Ca^{+2} affect the biofilm structure. Interestingly, added Ca^{+2} has been shown to cause biofilms of mucoid *P. aeruginosa* FRD1, which is 10 to 20-fold thicker than non-Ca^{+2} added biofilms. The amounts of extracellular proteases were increased in Ca^{+2} modified biofilms and it was also found that the proteases were housed in the Ca^{+2} enhanced alginate matrix [4]. To date, salt and glucose have always been studied and studied in health-related diets. As a result of this information, it should be remembered that Ca^{+2} diets may also be effective in understanding and reducing the effects of microorganisms.

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Conflict of Interest

No conflict of interest.

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Hüseyin Kahraman. Biomed J Sci & Tech Res

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