Cell Calcium xxx (2014) xxx–xxx

Contents lists available at ScienceDirect

Cell Calcium

journal homepage: www.elsevier.com/locate/ceca

Review

The impact of mitochondrial endosymbiosis on the evolution of calcium signaling

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ARTICLE INFO

Article history:
Available online xxx

Keywords:
Calcium
Eukaryogenesis
Evolutionary conflict
Metabolic state
Mitochondria
Proto-mitochondria
Reactive oxygen species

ABSTRACT

At high concentrations, calcium has detrimental effects on biological systems. Life likely arose in a low calcium environment, and the first cells evolved mechanisms to maintain this environment internally. Bursts of calcium influx followed by efflux or sequestration thus developed in a functional context. For example, in proto-cells with exterior energy-converting membranes, such bursts could be used to depolarize the membrane. In this way, proto-cells could maintain maximal phosphorylation (metabolic state 3) and moderate levels of reactive oxygen species (ROS), while avoiding the resting state (metabolic state 4) and high levels of ROS. This trait is likely a shared primitive characteristic of prokaryotes. When eukaryotes evolved, the α-proteobacteria that gave rise to proto-mitochondria inhabited a novel environment, the interior of the proto-eukaryote that had a low calcium concentration. In this environment, metabolic homeostasis was difficult to maintain, and there were inherent risks from ROS, yet depolarizing the proto-mitochondrial membrane by calcium influx was challenging. To maintain metabolic state 3, proto-mitochondria were required to congregate near calcium influx points in the proto-eukaryotic membrane. This behavior, resulting in embryonic forms of calcium signaling, may have occurred immediately after the initiation of the endosymbiosis. Along with ROS, calcium may have served as one of the key forms of crosstalk among the community of prokaryotes that led to the eukaryotic cell.

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1. Introduction

Calcium is a highly versatile signal that regulates many different cellular functions [1]. The basis for calcium signaling is a shared feature of all cells: the intracellular concentration of calcium is maintained at a much lower level than that of the extracellular environment. For instance, in the well-studied mammals, the concentration within a cell approaches 100 nmol l^{-1}, as compared to the extracellular concentration of 1 mmol l^{-1} [2]. Various pumps, channels, exchangers, and binding proteins maintain the low intracellular concentration [3,4]. Against this low intracellular background, influx from the extracellular environment or intracellular stores results in pulses or waves of Ca^{2+}. These pulses can be tuned in an amazing number of ways, rendering the calcium ion a key second messenger.

As with all other examples of adaptation, the evolution of calcium signaling is based partly on function and partly on history. Indeed, there is a consensus that calcium signaling provides important information regarding the history of life [5–10]. Free calcium is toxic to life in a number of ways, e.g., causing chromatin condensation, precipitation of phosphate and protein, and activation of degradative enzymes [5,7,8]. To the degree that early life shared these features with extant life, the origin of life must have occurred in an environment with very low calcium concentrations. In seawater, calcium and carbonate ions combine to precipitate calcium carbonate abiotically. To deplete the calcium in seawater, high levels of carbonate are needed and hence high alkalinity. At low pH, however, the equilibrium favors bicarbonate over carbonate, so both high pH and alkalinity are needed to deplete calcium by abiotic precipitation of calcium carbonate. If the ocean exhibited these features globally [8] or locally [11], then calcium concentrations suitable for early life would have been achieved. As life dispersed from havens of high pH and alkalinity or as the chemistry of the entire ocean changed, or both, calcium levels surrounding proto-cells would have increased. To survive, the proto-cells had to evolve mechanisms to maintain low intracellular calcium concentrations. Calcium signaling emerged as a by-product of these low intracellular concentrations, and likely did so before the divergence of archaeans and bacteria. Mechanisms of calcium signaling may thus be shared derived characteristics of LUCA, the last universal common ancestor from which all living organisms are descended. Considerable insight regarding the

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http://dx.doi.org/10.1016/j.ceca.2014.11.006
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Please cite this article in press as: N.W. Blackstone, The impact of mitochondrial endosymbiosis on the evolution of calcium signaling, Cell Calcium (2014), http://dx.doi.org/10.1016/j.ceca.2014.11.006

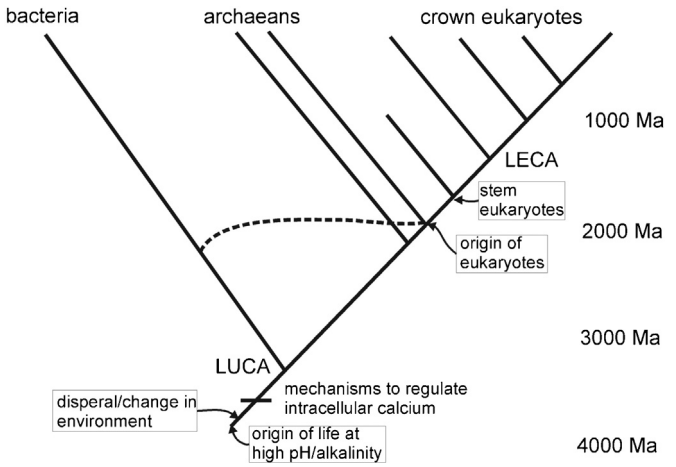

Fig. 1. Schemata illustrating the history of life and the evolution of calcium signaling. Life originated in seawater at high pH and alkalinity, which leads to precipitation of calcium carbonate. The biochemistry of life thus requires low [Ca^{2+}]. As life dispersed and ocean chemistry changed, life evolved mechanisms to regulate intracellular [Ca^{2+}] prior to LUCA, the last universal common ancestor. These mechanisms may constitute a shared derived character for all extant life. Calcium signaling emerged as a by-product of the low intracellular [Ca^{2+}] compared to the extracellular environment. Considerably later in the history of life during the origin of eukaryotes [20], proto-mitochondria, descended from α-proteobacteria (dashed line), began an endosymbiosis with an archaean host [21]. A number of features were derived by stem eukaryotes, which include all of the extinct eukaryotes from their origin to LECA, the last eukaryotic common ancestor. One of the earliest features derived by stem eukaryotes was likely a new form of calcium signaling. Since they inhabited the low-calcium interior of another cell, proto-mitochondria could not carry out calcium signaling in the usual prokaryotic fashion by using an influx of extracellular calcium. Rather, proto-mitochondria congregated near influx channels in the host membrane. Eukaryotic calcium signaling was thus initiated and served to mediate conflicts early in this evolutionary transition (timescale is approximate).

early history of life is thus provided [5–10]. The power of calcium signaling to illuminate the history of life, however, is not limited to life's early stages (Fig. 1). Here, the focus will be on a later but equally crucial period in the history of life, extending from the origin of eukaryotes to LECA, the last eukaryotic common ancestor from which all living eukaryotes are descended. During this time period the endosymbiotic origin of mitochondria and the formation of the eukaryotic cell occurred concurrently.

In the context of calcium signaling in living cells, mitochondria have been perceived as everything from irrelevant to central [12,13]. The findings in the 1970s that the affinity of the mitochondrial uniporter for calcium was weak suggested the former. More recently, recognition that Ca^{2+} microdomains exist within the cell argued decisively for the latter [14]. Thus when for instance inositol triphosphate binds to the inositol triphosphate receptor, the massive pulse of calcium being released by the endoplasmic reticulum easily activates mitochondria that are nearby. Further data revealed that not only do mitochondria congregate in the appropriate areas, they also form a specialized junction with the endoplasmic reticulum: the mitochondria-associated ER membrane (MAM) [2,15,16].

The physical and functional coupling of MAM allows the mitochondrial metabolic state to be telegraphed in calcium waves. For instance, in mitochondria that are provided with sufficient substrate, entry of moderate amounts of calcium will lower the membrane potential and cause an immediate activation of oxidation of substrate to rebuild this potential [13,17–19]. Several mitochondrial enzymes used in processing substrate are activated by calcium, including the pyruvate dehydrogenase complex [19]. These activated enzymes can then maintain the flow of electrons to the electron transport chain, which can continue to build or rebuild membrane potential. Using this potential, ATP can energize the nascent calcium wave. On the other hand, such calcium signaling will proceed differently when for example mitochondria are deprived of substrate. Such mitochondria will be unable to rebuild their membrane potential or convert ADP to ATP. Under these circumstances, the cellular calcium signal could be stillborn.

Table 1
Characteristics of stem eukaryotes relevant to conflict mediation and the hypothesized timing of their origin.

Derived earlier	Derived later
Metabolic association	Nucleus
Endosymbiosis	Other organelles
Chimeric proto-nuclear genome	Specialized carrier proteins
Size increase	Translocase outer membrane
Aerobic metabolism	Translocase inner membrane
Mitochondria	Gene/genome loss
Reactive oxygen signaling	Other forms of signaling
Calcium signaling	Eukaryotic sex

These and other features of modern cells can provide clues regarding the history of the interactions that led to the formation of the eukaryotic cell. Consider this modern interaction between mitochondria and the cytosol in the context of a two-billion-year-old endosymbiosis. Mitochondria are α-proteobacteria that became endosymbionts within an archaean host roughly 2 Ga [20,21]. While the modern interaction is cooperative, in any interaction between biological units, cooperation is not automatic and must evolve [22]. Indeed, evolutionary theory predicts that conflictual stages occurred during the early history of endosymbiosis. These conflicts likely destroyed many budding proto-eukaryotes and go a long way to explain why eukaryotes only evolved once [23,24]. To survive, the proto-eukaryote had to evolve mechanisms of conflict mediation [22]. Typically, these mechanisms increase variation among higher-level units (the proto-eukaryotes) and decrease variation among lower-level units (the proto-mitochondria). In the former case, unfit combinations can be weeded out by selection; in the latter, lower level units that favor their own replication at a cost to the group are less likely to evolve. Here calcium signaling will be examined with regard to the evolution of mechanisms of conflict mediation and their impact on eukaryogenesis.

2. Evolutionary context

Mitochondrial endosymbiosis appears to be intimately related to the formation of the eukaryotic cell [25,26]. Given the phylogenetic evidence [21,27], the most likely partners were α-proteobacteria (the proto-mitochondria) and an archaean (the host). With some assumptions, the common ancestor of all extant eukaryotes can be termed LECA (last eukaryotic common ancestor). The crown eukaryotes include LECA and all of its descendents including all extant eukaryotes and many now extinct ones as well (Fig. 1). Stem eukaryotes include all of the extinct taxa from the origin of eukaryotes to LECA. During the evolution of stem eukaryotes, many of the shared features that characterize eukaryotes were derived. Some of these shared derived characters were relevant to conflict mediation. Given the lack of fossil evidence, the timing of the derivation of these characteristics remains a matter of conjecture. A simple premise can be useful here: features of eukaryotes that were co-opted from prokaryotes are considered more likely to have evolved earlier, while features of eukaryotes that evolved de novo are considered more likely to have evolved later. As described below, a number of these features (Table 1) may thus have been derived earlier (concomitant with the origin of eukaryotes), while others were derived later (chronologically closer to LECA).

A number of metabolic associations could have led to eukaryotes [28]. Such metabolic associations may have had a number of consequences, perhaps including increased size of the archaean partner

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