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REVIEW

From Cellular Architecture to Regulation of Mitochondrial Function: Role of Vimentin in Ensuring Cellular Mitostasis

Roaa Deeb[1,2], Anton S. Shakhov[1,3], Aleksandra S. Churkina[1,3], Irina B. Alieva[1,3], and Alexander A. Minin[1,a]*

[1]*Institute of Protein Research, Russian Academy of Sciences, 119334 Moscow, Russia*

[2]*Moscow Institute of Physics and Technology (MIPT), 141701 Dolgoprudny, Moscow Region, Russia*

[3]*Belozersky Institute of Physico-Chemical Biology, Lomonosov Moscow State University, 119992 Moscow, Russia*

[a]*e-mail: alexminin@gmail.com*

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Abstract—Mitochondria play a central role in cell physiology, and in addition to performing their primary function as an energy source, they are involved in processes such as regulating intracellular calcium levels, generating reactive oxygen species, synthesizing many critical compounds, regulating apoptosis, and more. In this regard, maintaining them in a normal state is of great importance, ensuring their transport, intracellular distribution, timely biogenesis, and removal of damaged mitochondria from the cells. All of this is defined as cellular mitostasis, maintenance of which involves many cellular structures and, primarily, the cytoskeleton. This review summarizes the data on the role of one component of cytoskeleton, vimentin intermediate filaments, in these processes.

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INTRODUCTION. MITOCHONDRIA ARE POWERHOUSES IN CELLS BUT NOT JUST THAT

Classic notions on mitochondria as organelles responsible for supplying energy for the cell needs have been extended significantly in the recent half century. It was demonstrated in numerous studies that in addition to supplying energy, mitochondria also participate in a number of intracellular processes not directly associated with oxidation of organic compounds and generation of energy in form of ATP molecules (Fig. 1). Mitochondria are among the main intracellular depots of calcium ions [1], they play an important role in metabolism regulation, participate in synthesis of steroid hormones [2] and other compounds, and in regulation of apoptosis [3].

To effectively perform their functions, mitochondria require certain balance of intracellular factors ensuring, among other things, their correct mitostasis – maintenance and regulation of the distributed pool of healthy mitochondria during the entire lifespan of the cell [4]. The term ‘mitostasis’ is usually understood as combination of such processes as mitochondrial respiration, their fusion and fission (depending on the cell needs), mitochondrial transport, and mitochondria anchoring at the locations of their active functioning [5]. Furthermore, maintenance of mitochondria population in a healthy state is impossible without removal of damaged proteins and organelles.

* To whom correspondence should be addressed.

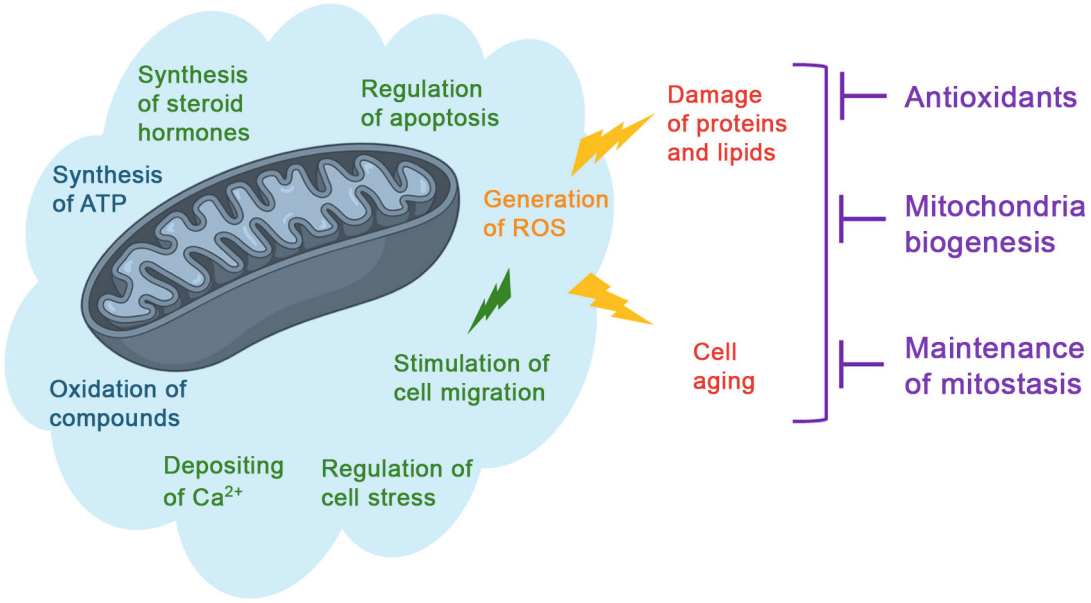

Fig. 1. Functions of mitochondria in cells and their effects on different processes. The best-known classical functions of mitochondria (shown in blue color) are primarily associated with ATP synthesis and oxidation of organic compounds. However, direct participation of mitochondria in numerous other processes (shown in green color) has been demonstrated in recent years. Among those: synthesis of steroid hormones, regulation of apoptosis and cell stress, serving as an intracellular depot of calcium ions. Reactive oxygen species (ROS) generated in mitochondria participate in regulation of cell migration and, at the same time, negatively affect various cellular structures both inside mitochondria and outside of them; in particular, they could cause damage in various proteins and lipids, as well as they could induce cell aging. Many studies have been devoted to investigation of factors preventing negative effects of ROS; among those antioxidants have been investigated in most detail. Moreover, it is known that biogenesis of mitochondria and maintenance of normal mitostasis also facilitate slowdown of aging.

This process includes degradation of individual mitochondrial proteins and whole mitochondria via mitophagy and macroautophagy [6].

Quality and number of mitochondria decrease with aging, which results in the enhanced production of ROS that facilitate cell aging [7, 8]. Under conditions of enhanced production of ROS, oxidative reactions are induced, which damage nucleic acids, proteins, and membrane lipids. This, in turn, disrupts mitochondria functioning and subcellular localization of different important components, thus causing cellular pathologies [9, 10]. Intracellular levels of ROS are controlled by a number of enzymes with antioxidant activity. The available experimental data indicate a close relationship between the mitochondria biogenesis and antioxidant activity [11].

That is why the cellular components capable of mitostasis maintenance or of its normalization in the case of disruption under the effect of negative factors attract particular interest of the researchers. One of the probable candidates for this role is the cell cytoskeleton consisting of three fibrillar components – microtubules, actin microfilaments, and intermediate filaments (IF). We believe that IFs (vimentin filaments, in particular) are the most likely candidates for this role, and in this review, we intend to justify this idea.

CELL CYTOSKELETON AS A PLATFORM FOR DISTRIBUTION AND MODULATION OF CELULAR ORGANELLES

Cytoskeleton is a network of microtubules, actin filaments, and IF, it plays both the role of a rather rigid carcass of the cell, and the role of dynamic regulator of the cell organization. This comprehensive network of protein filaments exhibits a remarkable ability for rearrangement in response to various internal and external signals, which facilitates immediate adaptation of the cells to the changing conditions. Cytoskeleton is a key player in many processes including cell division, cell motility, maintenance of the cell shape, intracellular transport, and distribution of different organelles. Interactions of the cytoskeleton structures with mitochondria, which mediate their transport and distribution are of particular interest [12].

While the microtubules mediate long-distance transport of mitochondria, actin filaments facilitate their local transport [13]. The transport is realized with the help of motor proteins, kinesins and dyneins, in the case of microtubules, and with the help of myosins in the case of actin filaments [14]. Direction of transport along microtubules is determined by their polarity, as well as by the features of motor proteins,

which move along them only in one direction: almost all kinesins – towards the so-called plus-end, and dyneins – towards the minus end. The actin-dependent transport is realized via the action of myosin motor proteins, which also differ in the direction of walk along microfilaments. In particular, the myosin V transports cargo towards the fast growing plus ends of microfilaments, while the myosin VI mediates transport towards the minus ends [15]. Furthermore, movement of some organelles could occur as a result of forces generated by the polymerizing actin [16]. This complex transport system ensures timely delivery of mitochondria and other organelles to various, sometimes rather distant parts of the cell. In addition to transport, the cytoskeleton structures play roles in maintaining position of the delivered organelles in the location of their functioning.

Videomicroscopy analysis (time-lapse microscopy) of the mitochondria behavior in the fibroblasts revealed that majority of these organelles are in the stationary state, and only minor fractions are in the process of transport along microtubules [17-21]. It was found that motility of mitochondria is under regulatory control. In particular, one of the growth factors, lysophosphatidic acid, acting through the small GTPase RhoA and mDia1 protein associated with it, inhibits mitochondria movement and causes their anchoring at the cell periphery [20]. This effect is associated with induction of actin polymerization, because latrunculin B, which F- disrupts actin, completely blocks it, and, *vice versa*, increases motility of mitochondria [19, 20]. It was shown in further studies that the vimentin IFs play a role of a mediator between the interacting mitochondria and cytoskeleton actin structures [22].

Using targeted mutagenesis, we were able to identify the site at the N-terminus of the vimentin molecule responsible for mitochondria binding [22] that exhibits properties of signaling sequence for mitochondria localization [23]. Considering that the mitochondrial proteins with such signaling sequences interact with the complexes of mitochondrial translocases [24], it could be suggested that vimentin also interacts with them. These data attracted significant attention from many researchers, because it was known that the functions of mitochondria in many cells are disrupted in the case of damage of the IF network [25, 26]. Large amounts of data have been accumulated on various pathological states associated with disruption of different types of IFs and, as a consequence, with defects in mitochondria functioning [27-30]. Many authors emphasized the probable role of IFs in regulation of the properties of mitochondria and of the whole cells [31-33], however, at present there are no sufficient data on connections of these organelles and on mechanisms underlying the observed cellular defects to make definite conclusions.

VIMENTIN INTERMEDIATE FILAMENTS ENSURE CELLULAR MITOSTASIS

Unlike in the case of microtubules and actin microfilaments, which are composed from a limited number of isoforms of tubulin and actin, respectively, and are practically identical in all eukaryotes, IFs are represented by one of the numerous protein families [34]. In the human genome, more than 70 genes encoding various IF proteins were identified [35], which are expressed depending on the tissue type and stage of differentiation. Vimentin is unique among the other IF proteins: in addition to mesenchymal cells where it is the only representative of this family, its expression was found in neurons, epitheliocytes, muscle cells, and some other [35], where it appeared under certain conditions. And, although, as has been mentioned above, disruption of the functions of mitochondria has been observed in different cell types with damaged IFs of different composition, the issue on their ability to bind mitochondria directly remains open to discussion. At present, direct association with mitochondria was demonstrated in our study only for vimentin [36] and desmin IFs [37], no data are available for other IFs.

How the vimentin IFs control properties of mitochondria? In addition to limiting motility of mitochondria on their binding to vimentin IFs, this interaction results in the increase of the mitochondrial membrane potential [38]. Interestingly, increase of the level of membrane potential was observed not only during restoration of vimentin IFs in the knockout cells, but also in the case of expression of the vimentin fragment containing the mitochondria-binding site [37]. Association of vimentin IFs with mitochondria is under regulatory control. In particular, it was shown in our study [39] that activation of the small GTPase Rac1 increases motility mobility of mitochondria in the mouse fibroblasts 2-fold. It was found that one of its effectors, protein kinase PAK1, could participate in vimentin phosphorylation at the Ser55 residue, and this disrupts its association with mitochondria. In addition to the increase of motility of mitochondria upon activation of the GTPase Rac1 and protein kinase PAK1, a decrease in the mitochondrial membrane potential was observed. Interestingly, replacement of Ser with Ala at this position of vimentin molecule blocked the effect of GTPase Rac1 on the properties of mitochondria [39]. It is likely that there are other mechanisms regulating this interaction. In particular, analysis of recombinant vimentin binding with mitochondria isolated from the rat liver cells

demonstrates that the protein could be subjected to partial hydrolysis, which is catalyzed by mitochondrial proteases [36]. Proteolysis of the N-terminal part of the vimentin molecules was observed during incubation in the absence of inhibitors of cysteine proteases, and their association with mitochondria was disrupted. The results of inhibitory analysis showed that the mitochondrial atypical Ca^{2+}-dependent calpain protease is the enzyme responsible for vimentin degradation. Considering that mitochondrial matrix contains main fraction of the intracellular Ca^{2+}, and its concentration in the intermembrane space of mitochondria and in cytosol is very low, it could be suggested that proteolysis is initiated by the decrease of mitochondrial potential, when Ca^{2+} is excreted [36]. It is likely that the association of mitochondria with vimentin IFs is controlled by the mitochondrial potential.

Mitochondrial membrane potential could be maintained via two pathways: a) operation of mitochondrial electron-transport chain of mitochondria; b) as a result of ATP hydrolysis in the reverse reaction catalyzed by H-ATPase. Hence, the effect of vimentin IFs on the level of membrane potential observed in our study could be caused both by stimulation of activity of the mitochondrial respiratory chain, and through the independent pathway involving acceleration of ATP hydrolysis. In order to find out which of these mechanisms underlie the effect of vimentin, we used the fibroblast cell line lacking mitochondrial DNA, and, as a consequence, lacking several important protein components of the mitochondrial respiratory chain. Potential in these cells is maintained through hydrolysis of ATP formed in the process of glycolysis. We demonstrated that restoration of vimentin IFs in these cells did not result in the increase of potential. And, on the contrary, suppression of vimentin expression in the cells with disrupted respiratory chain with the help of RNA interference did not decrease potential as observed in the initial cells [38]. Hence, the effect of vimentin IFs on the mitochondrial membrane potential is observed only in cells with a fully functional respiratory chain. How could vimentin affect the operation of the mitochondrial respiratory chain?

It was shown previously that mitochondria have low respiratory activity, increased level of ATP, and disrupted morphology in the fibroblasts isolated from the mice with knockout of the vimentin gene [33]. The authors of this study suggested that the main cause of disruption of mitochondrial functions is oxidative stress, and vimentin protects mitochondria against ROS [33]. However, the causes of the increase of ROS levels in the mitochondria remain poorly understood.

It was demonstrated recently in the collaborative study of the laboratories of Russo and Markovitz that knockout of the vimentin gene in mice causes significant changes in the expression of the genes of many proteins in neutrophils [40]. They found an increase of expression of 108 genes and decrease of expression of 416 genes in the cells lacking vimentin in comparison with the wild type neutrophils. Significant changes were observed in the expression of the genes participating in mitochondria functioning. In particular, expression of 55 genes encoding proteins of all five complexes of mitochondrial respiratory chains was reduced. Hence, vimentin IFs in neutrophils control protein composition of mitochondria, and, consequently, their properties. Furthermore, it was shown that deletion of vimentin results in the decrease of expression of the proteins SOD1 and SOD2 that play roles of antioxidants [40]. The authors suggested that exactly these changes were the causes of noticeable increase of ROS levels in the neutrophils.

It should be mentioned that ROS, which cause different damages and pathologies, also perform a number of regulatory functions, and for this, their concentration should be maintained at a certain level. Several sources of ROS exist in the cells including NADPH-oxidase integrated into the plasma membrane, xanthine oxidase, and mitochondria [41], as well as the system of antioxidant enzymes. Thus, our data [42] indicate that peroxide formed in mitochondria stimulates migration of fibroblasts. Hence, it is likely that vimentin IFs not only decrease the level of ROS but also participate in stabilization of its level. On the other hand, vimentin ensures enhanced resistance of mitochondria to oxidative stress [43] and to action of anti-cancer preparations doxorubicin and vincristine [44]. This protective effect could indicate not only decrease of the ROS level but also changes in the mitochondria properties caused by interaction with vimentin. This suggestion is supported by the fact that expression of the vimentin mutant with disrupted ability to bind mitochondria did not provide protective effect in the cells [42, 43]. Hence, the protective effect of vimentin depends on its ability to bind mitochondria. However, binding of vimentin IFs with mitochondria has not been investigated in sufficient detail, although there are data indicating that regulation of the mitochondria interaction with vimentin IFs could be realized both directly and indirectly. The ITPRIPL2 protein co-localized with vimentin could play a role of a mediator [45]; knockdown of this protein disrupts processing of vimentin and formation of IFs and plectin 1b [46]. It was also shown that the microRNA miR-124 regulates vimentin expression and indirectly controls motility of mitochondria [47].

Based on the data obtained in our study [22], the site of vimentin molecule responsible for interaction is located in the N-terminal part of the molecule, and its composition is similar to the localization

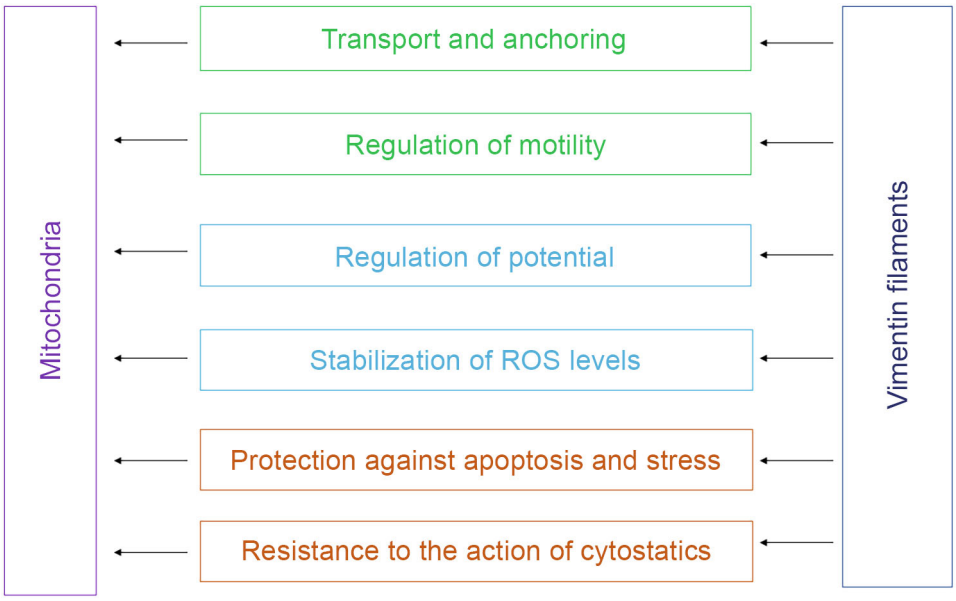

Fig. 2. Effects of vimentin IFs on mitochondria that maintain their homeostasis: binding of mitochondria to the network of IFs limits their motility and determines their intracellular distribution (shown in green color); maintenance of high mitochondrial membrane potential and decrease of ROS concentration (shown in blue color); increase of resistance of mitochondria to oxidative stress and to the action of cytostatic agents, and protection against apoptosis (shown in orange color).

signal typical of many mitochondrial proteins. Hence, vimentin could bind to the mitochondrial translocases TOM/TIM responsible for import of proteins containing these sequences. However, accessibility of the N-terminal domain in vimentin could be limited. According to the recent data reported by the Medalia research group obtained with the help of cryo-electron microscopy and tomography [48], the vimentin IFs comprise a cylindrical structure formed by five protofibrils, each composed of 40 alpha-helices of the vimentin central domain. The non-structured C-terminal parts are located outside and stabilize bonds between the protofibrils, while the N-terminal domains are in the inner cavity of the filament and form a rather compact bundle also creating additional bonds between the individual components. Hence, the N-terminal domain site responsible for mitochondria binding turns out to be hidden inside the filament. It can be assumed that mitochondria bind to this part of the vimentin molecule at the terminal parts of the filaments, where the cylindrical structure is not fully formed. Otherwise, significant rearrangements of the vimentin IFs would be required to allow external exposure of the N-terminal sites.

CONCLUSIONS

In conclusion, it could be stated that among the three components of cytoskeleton, the vimentin IFs are the most suitable candidates capable of maintaining mitostasis. They could play a role of organizers of intracellular space, and this organization is vital – abundant evidence exists on different pathological states associated with disruptions of the IF structure, which cause defects in the mitochondria functioning.

The data on participation of vimentin in regulation of the properties of mitochondria provide grounds to assumptions that the vimentin IF could contribute to mitostasis maintenance. A number of factors, both structural and functional, indicate their involvement in realization and maintenance of normal functioning of mitochondria. In particular, vimentin filaments interact with mitochondria; this interaction could be direct or through the protein mediators. Vimentin filaments regulate motility of mitochondria; they are capable of stabilizing mitochondria and protecting them against apoptosis and stress. And, finally, vimentin filaments could affect the mitochondrial potential (Fig. 2).

By regulating mitochondrial functions, vimentin IFs could significantly affect properties of the whole cells: the cells expressing vimentin, unlike other cells, have the ability to migrate over significant distances, they exhibit increased resistance to the action of cytostatics, and are less susceptible to apoptosis under unfavorable conditions. From this point of view, vimentin filaments could be considered as a target for normalization of mitostasis in the case of its disruption under the action of external or internal negative factors. This fundamentally novel approach could offer a new solution to the problem of treating numerous human diseases associated with disruptions of mitochondria functions.

Abbreviations

IF	intermediate filaments
ROS	reactive oxygen species

Contributions

I. B. Alieva and A. A. Minin – concept and supervision of the study, discussion of the results; R. Deeb, A. S. Churkina, I. B. Alieva, A. S. Shakhov, and A. A. Minin – writing text of the paper; A. S. Shakhov and A. S. Churkina – preparation of illustrations; I. B. Alieva and A. A. Minin – editing text of the paper.

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Ethics approval and consent to participate

This work does not contain any studies involving human and animal subjects.

Conflict of interest

The authors of this work declare that they have no conflicts of interest.

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