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Review

Modulation of mitochondrial functions by xenobiotic-induced microRNA: From environmental sentinel organisms to mammals

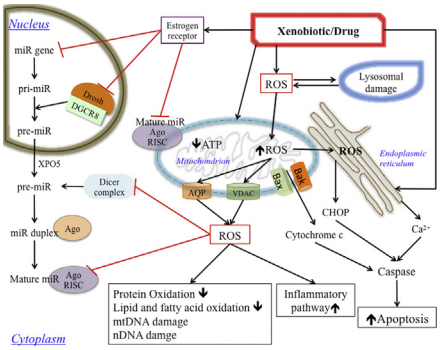
Mario Alberto Burgos-Aceves [a], Amit Cohen [b], Gaetana Paolella [a], Marilena Lepretti [a], Yoav Smith [b], Caterina Faggio [c,*], Lillà Lionetti [a]

[a] Department of Chemistry and Biology, University of Salerno, via Giovanni Paolo II, 132, 84084 Fisciano, SA, Italy
[b] Genomic Data Analysis Unit, The Hebrew University of Jerusalem-Hadassah Medical School, P.O. Box 12272, Jerusalem 91120, Israel
[c] Department of Chemical, Biological, Pharmaceutical, and Environmental Sciences, University of Messina, Viale F. Stagno d'Alcontres, 31, 98166 Messina, Italy

HIGHLIGHTS

- This review summarizes the current knowledge of miRNA role in condition of xenobiotic exposure.
- miRNA in regulating mitochondria and the effect of xenobiotic on mitochondrial function
- Modulation of miRNA levels may provide a new therapeutic approach.

GRAPHICAL ABSTRACT



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ABSTRACT

Mitochondria play a crucial role in energetic metabolism, signaling pathways, and overall cell viability. They are in the first line in facing cellular energy requirements in stress conditions, such as in response to xenobiotic exposure. Recently, a novel regulatory key role of microRNAs (miRNAs) in important signaling pathways in mitochondria has been proposed. Consequently, alteration in miRNAs expression by xenobiotics could outcome into mitochondrial dysfunction, reactive oxygen species overexpression, and liberation of apoptosis or necrosis activating proteins. The aim of this review is to show the highlights about mitochondria-associated miRNAs in cellular processes exposed to xenobiotic stress in different cell types involved in detoxification processes or sensitive to environmental hazards in marine sentinel organisms and mammals.

* Corresponding author.
E-mail address: cfaggio@unime.it (C. Faggio).

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Contents

1. Introduction

In eukaryotic cells, mitochondria are the main source of adenosine triphosphate (ATP) production as well as reactive oxygen (ROS) and nitrogen species (RNS) (Heise et al., 2003; Donaghy et al., 2012; Putti et al., 2015; Rivera-Ingraham et al., 2016). Reviews by Donaghy et al. (2015) and Zeeshan et al. (2016) point to the endoplasmic reticulum (ER) as another source of ROS. The synthesis of mitochondrial ROS as well as other essential biological processes seems to be regulated by microRNAs (miRNAs, miRs), both in mammals and non-mammalian organisms (Kren et al., 2009; Christian and Su, 2014; Duarte et al., 2014; Burgos-Aceves et al., 2018a). The miRNAs are a group of small endogenous noncoding segments of RNA, ~18–25 nucleotide (nt) long that play a critical role in modulating gene expression (Filipowicz et al., 2008). To date, there is growing evidence that miRNAs are also present in or associated with other organelles (Fig. 1) such as mitochondria (Sripada et al., 2012; Tomasetti et al., 2014), ER (Li et al., 2013; Montgomery and Ruvkun, 2013; Axtell, 2017), processing bodies (P-bodies), stress granules, multivesicular bodies, and exosomes (Nguyen et al., 2014). Further, it has been suggested that cytosolic miRNAs can be transferred within the mitochondria (Li et al., 2012) or generated within it (Latronico and Condorelli, 2012; Sripada et al., 2012; Bandiera et al., 2013), and modulate genes expression and regulate important signaling pathways (Li et al., 2012). Thus, deregulation of miRNAs biosynthesis can be associated with mitochondrial dysfunction (Wang et al., 2017).

The aim of this review is to summarize the current knowledge on the role played by miRNA in regulation of mitochondrial function in condition of xenobiotic exposure. In the first part of the review, we introduced the role played by miRNA in regulating mitochondria and the effect of xenobiotic on mitochondrial function. In the second part, we summarized current knowledge on xenobiotic effect on mitochondrial associated-miRNA in cells from both hazards sentinel organisms and

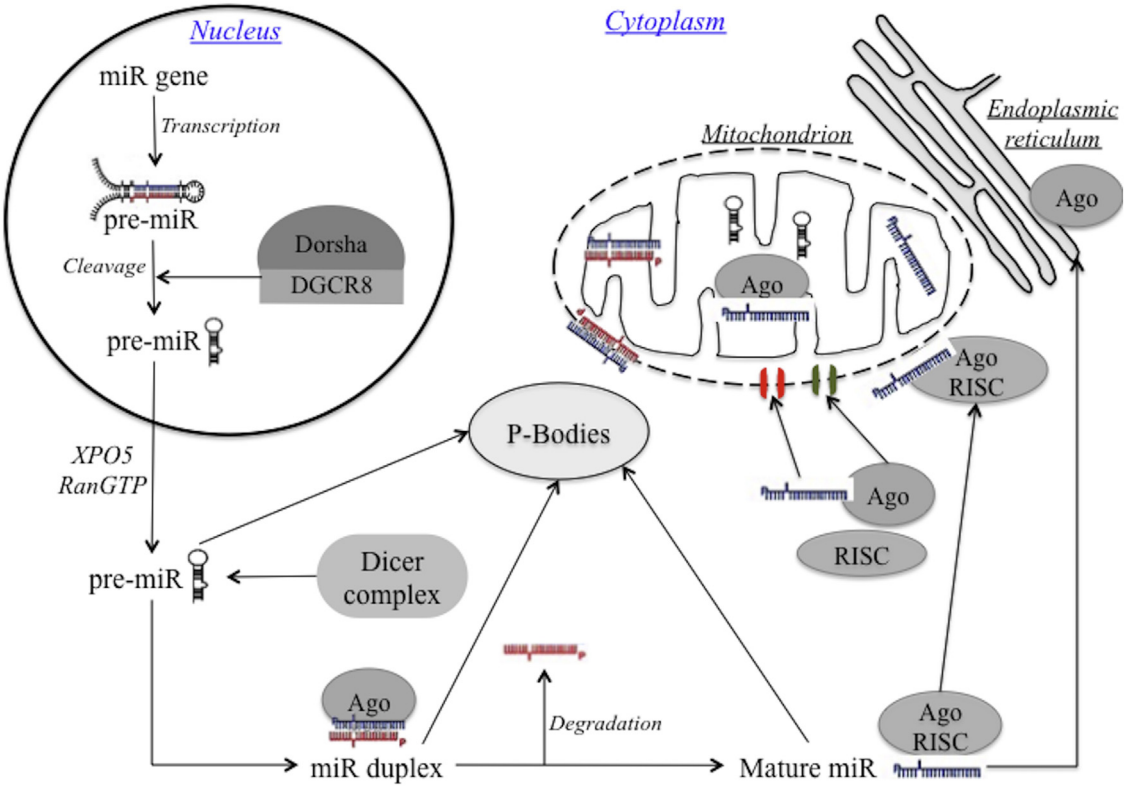

Fig. 1. Schematic microRNAs (miRNAs, miRs) translocation to mitochondrion. Once the biogenesis process (canonical) of miRNAs has been carried out, both pre-miRNAs and mature miRNAs can be translocated to various subcellular locations as nucleus, mitochondria, endoplasmic reticulum, P-bodies, etc. Mitochondrial outer membrane may itself serve as a novel platform for the miRNA transport or assembly, and the presence of pre-miRNAs also in mitochondria, suggesting that mitochondria may provide a miRNAs assembly platform. miRNAs have been suggested to augment translocation under specific circumstances through a separate importation of miRNAs and argonaute (Ago) protein by a yet unidentified protein import complexes located in the mitochondrial intermembrane space (channels in red and green). Abbreviations: RISC, RNA-induced silencing complex; Exportin, 5 XPO5; DGCR8, DiGeorge syndrome critical region gene 8. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)