ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 01, pp.44956-44957, January, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

REVIEW ARTICLE

"INTRACELLULAR DRUG TARGETING": SPECIFIC BINDING OF DRUGS TO THE OUTER MEMBRANE OF MITOCHONDRIA

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ARTICLE INFO

Article History:
Received 20th October, 2016
Received in revised form
22nd November, 2016
Accepted 17th December, 2016
Published online 31st January, 2017

Key words:

Intracellular Drug Targeting,
Outer Mitochondrial Membrane.

ABSTRACT

This short communication is a proposal. Experimental data are not yet available. Mitochondria in eukaryotic cells are not involved in the processes of membrane flow and membrane transformation between cellular organelles. Therefore, a drug intended to remain inside a cell can be expected to stay there when it is bound to mitochondria. A prerequisite for binding is that the drug is equipped with a component that is suited to specifically bind to a kind of protein that is exposed at the surface of the outer mitochondrial membrane ("intracellular drug targeting").

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Citation: Frank Mayer. 2017. ""Intracellular drug targeting": specific binding of drugs to the outer membrane of mitochondria", *International Journal of Current Research*, 9, (01), 44956-44957.

INTRODUCTION

Membrane flow and membrane transformation are typical features of eukaryotic cells (Fig.1). An example: Golgi vesicles in a dictyosome are closely related to other vesicles, to the endoplasmic reticulum, but also to the cytoplasmic membrane of the cell and to the membrane envelope of the cell nucleus. When it is intended that a drug present inside a cell does remain inside the cell, this would not work when the drug is bound to one of these organelles. Mitochondria are not involved in these processes. This can be explained by the endosymbiont theory. Hence, for immobilization of drugs that are intended to remain inside the cell, mitochondria can be expected to be their target of choice. Specifically, it should be a protein that is exposed at the outer surface of the outer mitochondrial membrane, independent of how the drug had achieved the passage through the cytoplasmic membrane and its arrival in the cellular cytosol. One point should be kept in mind: it should be made sure that the uptake of the drug into the cell should not be by endocytosis. Uptake by endocytosis would result in the final location of the taken-up drug not in the cytosol, but in one of the organelles involved in membrane flow and membrane transformation. Its degradation would be unavoidable. Immobilization of the drug could either be permanent (depending on its intended function, e.g. as a kind of catalyst for a reaction inside the cell), or it could be transient, i.e. if a "depot" of the drug, with occasional release, is the goal.

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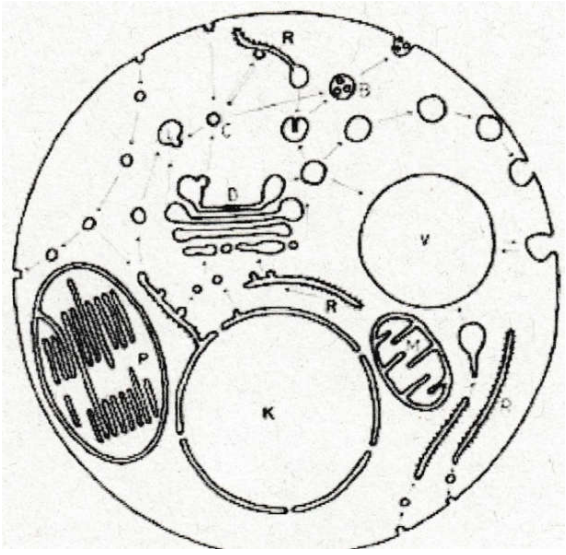

Diagrammatic view of an idealized eukaryotic cell, exhibiting membrane-enclosed compartments and their interaction by membrane flow and membrane transformation. Note: mitochondria and chloroplasts are not involved in these dynamic processes.(From Mayer et al 2006). Abbreviations: B, C, L, vesicles, D dictyosome, K nucleus, M mitochondrium, P chloroplast, R endoplasmic reticulum, V vacuole

Fig. 1. Structural organization of eukaryotic cells

It is obvious that a number of questions have to be answered, e.g. identification of a protein exposed at the outer membrane of the mitochondrion, suited as a binding site for intracellular

drug targeting (Benda *et al*., 2002 describe an example), search for a chemical group that reacts with this protein and does not hinder the protein´s natural function, design of a complex consisting of the drug and this chemical group, mode of permeation of this drug-chemical group complex through the cytoplasmic membrane (Torchilin 2008), mechanism of release of the drug from the mitochondrial membrane in the case of transient immobilization, etc. Intracellular drug targeting, applied along the outlines described above, could have a most welcome positive consequence: the loss of substantial amounts of drugs could be avoided by such an intracellular drug targeting, both in cases where permanent or transient presence of the drug inside a cell is needed. The amount of negative side effects of drug application would be expected to be reduced. After all, the drug doses would probably be drastically reduced because the drug would not be lost or degraded.

REFERENCES

Binda, C., Newton-Vinson, P., Hubalek, F., Edmonson, D.F. and Mattevi, A. 2002. Structure of human monoamine oxidase B, a drug target for the treatment of neurological disorder. *Nature Struc Biol.,* 9 22-26

Mayer, F., Wheatley, D. and Hoppert, M. 2006. Some properties of interfacial water: determinants for cell architecture and function? In: Pollack G et al. (eds) Water and the Cell, pp. 253-271 (Chapter 12). Springer

Torchilin, V.P. 2008. Cell-penetrating peptide-modified pharmaceutical nanocarriers for intracellular drug and gene delivery. *Biopolymers* 90,Nr. 5 604-610

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