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The Cardiovascular Effects of Mitochondrion-Toxic Agents

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DESCRIPTION

Mitochondrial-toxic agents can cause or worsen cardiovascular disease. Mitochondrion-toxic agents can be classified as having or not having a clinical effect, inducing cardiac disease only in humans, only in animals, or both, as prescribed drugs, illegal drugs, exotoxins, nutrients, affecting the heart exclusively or also other organs, being effective only in patients with a mitochondrial disorder or cardiac disease or also in healthy subjects, or being solid, liquid, or volatile agents. In humans, due to mitochondrial dysfunction, the cardiotoxic agents include anthracyclines (particularly doxorubicin), mitoxantrone, cyclophosphamide, fluorouracil, imatinib, cisplatin, bortezomib, trastuzumab, arsenic trioxide, cyclosporine-A, lamotrigine, glycosides, lidocain, isoproterenol, zidovudine, nitroprusside, pivalic acid, alcohol, cocaine, pesticides, cadmium, mycotoxins, cyanotoxins, meat meal, or carbon monoxide. Even more agents only cause cardiac abnormalities in animals or tissue due to mitochondrion toxicity [1-2].

The mitochondrion-toxic effect is caused by impairment of the respiratory chain, oxidative phosphorylation, the Krebs cycle, or oxidation, a decrease in mitochondrion-membrane potential, increased oxidative stress, decreased anti-oxidative capacity, or induction of apoptosis. Cardiomyopathy, myocarditis, coronary heart disease, arrhythmias, heart failure, and Takotsubo syndrome are examples of cardiac abnormalities caused by these mechanisms. In the vast majority of cases, discontinuing the cardiotoxic agent results in complete recovery. Antioxidants and nutrients may be beneficial in addition. Coenzyme-Q, riboflavin, vitamin-E, vitamin-C, L-carnitine, vitamin-D, thiamin, folic acid, omega-3 fatty acids, and D-ribose, in particular, have been shown to alleviate mitochondrial cardiotoxicity [3-4].

Pharmaceuticals, illicit drugs, exotoxins, and food ingredients are all examples of mitochondrion-toxic agents. Upregulation of certain genes may also be mitochondrion-related. Pharmaceuticals are the most common type of mitochondrion-toxic agent. The impact of mitochondrion-toxic drugs on cardiac function and disease is largely unknown, but cardiac side effects caused by mitochondrial toxicity are becoming more

common. Mitochondria are organelles found in all cells except erythrocytes that play important roles in cell metabolism, proliferation, cell division, and cell death. Depending on the function of the tissue, mitochondria may be more or less prominent within a cell type. Mitochondria take up about 45% of the myocardial volume in the heart. A review of the literature revealed that mitochondrion- and cardio-toxic agents harm the heart through a variety of mitochondrial mechanisms. These include reductions in Adenosine Tri Phosphate (ATP) production (imatinib, bortezomib, transtuzumab, cyclosporine-A, lidocain, malathion, cadmium, meat meal, carbon monoxide), reductions in anti-oxidative capacity (cyclophosphamide, glycosides), reductions in mitochondrial membrane potential (doxorubicin, 5-fluorouracil, imatinib) [5-6].

It is critical to recognize that an increasing number of compounds to which humans, animals, or tissue cultures are exposed cause cardiotoxicity *via* mitochondrial damage. Prescribed drugs and exogen stressors are the most well-known mitochondrion and cardio-toxic compounds. Chemotherapeutics are the most common, followed by immune suppressants, cardiac medication, and pro-drugs. Alcohol, cocaine, pesticides, and heavy metals are examples of common non-prescription compounds [7-8].

Mitochondria, or "powerhouses," are unique organelles in that they are surrounded by a double membrane and have their own small genome. They also divide by simple fission independently of the cell cycle. Mitochondrial division is stimulated by energy demand, so cells with higher energy demands have more of these organelles than cells with lower energy demands [9-10].

REFERENCES

1. Sayed-Ahmed MM, Salman TM, Gaballah HE, *et al*, *Pharmacol Res*, **2001,** 43(6):513-520.
2. Rossier MF, *Cell Calcium*, **2006,** 40(2):155-164.
3. Razavi BM, Hosseinzadeh H, Movassaghi AR, et al, *Chem Biol Interact*, **2013,** 203(3):547-555.
4. Ramirez-Ortega M, Zarco G, Maldonado V, et al, *Eur J Pharmacol*, **2007,** 566(1-3):34-42.
5. Qiu T, Xie P, Liu Y, et al, *Toxicology*, **2009,** 257(1-2):86-94.
6. Prince PS, Sathya B, *Eur J Pharmacol*, **2010,** 635(1-3):142-148.
7. Possamai FP, Fortunato JJ, Feier G, *Environ Toxicol Pharmacol*, **2007,** 23(2):198-204.
8. Pizzo P, Pozzan T, *Trends Cell Biol*, **2007,** 17(10):511-517.
9. Perrotta I, Tripepi S, *Micron*. **2012,** 43(2-3):183-191.
10. Pentassuglia L, Timolati F, Seifriz F, *Exp Cell Res*, **2007,** 313(8):1588-1601.