Part 1

According to the Sudden Cardiac Arrest Foundation, cardiac arrests have been considered a public health crisis in the United States. 90% of cardiac arrests each year will result in fatality (Newman, 2024, p.1). In 2022, there were an estimated 356,461 cardiac arrests outside of hospitals. This means that less than 1 percent of the population will suffer from a cardiac arrest in 2022. However, they are still extremely deadly considering that based on the statistics, 320,815 of the arrests resulted in death. John Hopkins Medical Center says that fatigue, dizziness, shortness of breath, nausea, chest pain, heart palpitations and loss of consciousness are all symptoms of cardiac arrest that could be indicators for potential witnesses. They also say that you are at risk for cardiac arrest if you have a history of drug and alcohol abuse or smoking. Also, if you are obese, have heart disease, high blood pressure, high cholesterol or low potassium or magnesium. A family history of heart disease can also raise your risk factor for going into cardiac arrest (Chrispin, 2024, p. 1).

Ischemia is defined by the Cleveland Clinic as "a less-than-normal amount of blood flow to part of your body." This means that you have less oxygen cells being distributed throughout your body. Ischemia-Reperfusion is the process of attempting to restore blood flow to ischemic tissues, but can often result in the death or dysfunction of the cells that are being circulated. Reperfusion can also cause the organ to fail overall (Cowled & Fitridge, 2011, p. 1). The failure of the organs, and the death of the cells is known as Ischemia-Reperfusion Injury.

Intercellular Mitochondrial Transfer is the relocation of the mitochondria by methods of tunneling nanotubes (TNTs), extracellular vesicles (EVs), and gap junction channels (GJCs). This process also increases the mtDNA within the cells that are receiving and helps the cell survive by improving its ability to respirate (Liu, Qi, Sun, Cao & Ding, 2022, p. 1). This simply means that using mitochondrial transfer, healthy mitochondria are transferred into already dying cells to hopefully save the cells. This suggests that this process could be beneficial to even cancer cells, where we could slow or stop the decay of cells within the body. This method has been used on humans still at the clinical level using injections like an epidural, and it is continuing to be studied in humans and many mammals. Mitochondrial transplants could be the best way to fight otherwise thought incurable diseases or hard to treat diseases by saving healthy cells from the very beginning.

Part 2

Artificial Mitochondrial Transplantation, also known as AMT, is a medical method that has been around for over forty years at this point. This method is performed by extracting healthy mitochondria from functioning cells and transferring it to cells that are actively dying or are malfunctioning (Fairley, Grimm, Eckert, 2022). AMT seems to be possible anywhere in the body, but has shown recently to be increasingly effective in relation to brain injuries. It has been promising as well as a form of therapy for patients with extreme brain disorders. One of the strengths of AMT is that it can address many forms of mitochondrial dysfunction at the same time, since the dysfunction is hard to pin-point in the first place. This study done by Fairley,

Grimm and Eckert shows that it is possible to increase neuron survival through mitochondrial transfer in figure 2.

Cardiac arrest occurs when the heart stops pumping, which can lead to the brain being deprived of oxygen and blood flow for long periods of time. This can be detrimental to the survival or quality of life of a given patient. Based on a study by the Feinstein Institutes for Medical Research, when transplanting new mitochondrial material into rats (both frozen and fresh) post cardiac episode of some kind, the fresh mitochondria transplant increased rates of survival from 55 percent to 99 percent. While this does on animals, researchers believe that it is possible for it to be effective in humans as well. Another study, published in the Journal of Translational Medicine by BMC explains that both blood lactate levels and neuron function increased after a transplant of mitochondrial matter after cardiac arrest. The transplant increased brain function by increasing levels of ATP production in neurons, which then improved cognitive abilities and neurological function following a cardiac episode. Also, by helping reduce the buildup of lactate, normal metabolic rates are restored in the body. It can also alleviate neuroinflammation which leads to improved cerebral blood flow.

A study done and published in the National Library of Medicine, explains that the mitochondrial content can live in the body for around 6 months. However, the researchers digress that it is very difficult to find an accurate vitality age of mitochondria because of how many components it contains and how widespread it is. Long living mitochondria though can have lots of beneficial effects on the body and its energy production by increasing the production of the electron transport chain. Also, shown in a graphical abstract it is clear that while we might not be able to age mitochondria on their own, we do know that proteins inside the mitochondria can be short or long lived. The longer lived proteins are found to be much more stabilizing. These proteins also are related to the protection of mitochondrial respiration after the decline of transcription factors in RNA and DNA.

In conclusion, Mitochondrial Transfer (and synthetic processes like AMT) are incredibly effective in increasing rates of survival after both cardiac arrest and brain injuries. That being said, there is a difference between the transplantation of fresh mitochondrial content and frozen mitochondrial content. We saw this above with the mice in the experiment by the Feinstein Institute, that fresh mitochondrial content transfer is much more effective that frozen, at least in mice. This is because the freezing and dethawing process effectively destroys any sort of viable material. This means that it is not possible to see the same results when using frozen mitochondrial content instead of fresh. The freezing process damages the section of the mitochondria that produces energy, which is the point of having a transfer in the first place.