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Critical Writing Assignment #3

Mitochondrial DNA replacement therapy (mtDNA therapy) should be considered for clinical use because it gives families affected by severe mitochondrial diseases an option that currently doesn't exist for them. These conditions come from mutations in the small mitochondrial genome, which is passed down only through the maternal line, and even a relatively low proportion of mutant mtDNA can cause major health problems (Mitalipov and Wolf 2014). For women with high levels of mutated mtDNA, the usual alternatives—egg donation, adoption, or choosing not to pursue a biological child—don't address the specific hope of having a genetically related child who is not at risk for a serious inherited disorder. mtDNA replacement is meant to fill that gap.

The basic idea behind the therapy is straightforward. Researchers move the parents' nuclear DNA into a donor egg containing healthy mitochondria. Two main approaches, spindle transfer (ST) and pronuclear transfer (PNT), have been tested in animal studies and in early human embryo research (Mitalipov and Wolf 2014). ST, for example, has produced healthy macaques with only donor mtDNA, and both ST and PNT have supported normal development to the blastocyst stage in human embryos. One of the main concerns is how much mutated mtDNA carries over into the donor egg, but current data show that these levels tend to stay low—often below 2% and well under the threshold associated with disease (Mitalipov and Wolf 2014). While research is ongoing, the results so far suggest the technique can function without interfering with nuclear genes or causing large-scale chromosomal problems.

A lot of the ethical discussion centers on the fact that the change is heritable. Because mtDNA is passed to future generations, the therapy falls under germline modification and requires strict oversight (Mitalipov and Wolf 2014). There is also the "three-parent child" idea that comes up often, but it doesn't reflect what's actually happening. Mitochondria make up only about 0.1% of a person's DNA and do not affect traits like appearance or personality (Mitalipov and Wolf 2014). The donor simply provides working mitochondria so normal energy production can occur. Groups that have reviewed the issue, such as the Nuffield Council on Bioethics, generally conclude that if safety continues to hold, the benefits to affected families are significant (Mitalipov and Wolf 2014).

Some critics emphasize that families do have other options, and that is true. But these alternatives don't meet the specific goal of preventing transmission while keeping a genetic connection to both parents. Because mtDNA therapy replaces only cytoplasmic DNA and leaves the nuclear genome intact, it does not move into the territory of enhancement or designer traits (Mitalipov and Wolf 2014). It is focused on preventing a well-understood medical condition.

Given the severity of many mitochondrial disorders and the encouraging results from early ST and PNT studies, mtDNA replacement therapy should be allowed within a regulated clinical framework. It offers families a meaningful option they currently lack, while still keeping safety, oversight, and ethical review at the center of its use.

References

Mitalipov, S., & Wolf, D. P. (2014). Clinical and ethical implications of mitochondrial gene transfer. *Trends in Endocrinology & Metabolism, 25*(1), 5–7.