Abstract

When two alleles carried by a heterozygote are transmitted unequally to the zygote at the time of fertilization, transmission ratio distortion occurs. The best studied example of this phenomenon in mammals is t-haplotype in mice. The mouse t-haplotype is a selfish variant region on chromosome 17, in nature transmitted as a unit. Male mice homozygous for t haplotype are sterile, but heterozygotes transmit the t haplotype up to 99% of their progeny. This is believed to be caused by motility differences between sperm carrying the t haplotype and wild-type sperm from the same heterozygous male. The concrete mechanism of the postulated sperm competition in favour of t haplotype carrying sperm was so far not fully illuminated.

During this project, we worked with the hypothesis that the differences in sperm motility putatively responsible for transmission ratio distortion are triggered, at least in part, by metabolic causes.

Our results from ATP and mitochondrial membrane potential (MMP) comparison indeed suggest that there are metabolic dissimilarities in sperm from the different genotypes of t (t/t, t/+, +/+). Specifically, our data show that there is significantly less ATP in t/t sperm when compared to the other two genotypes. Likewise, sperm from t/t mice also seem to have lower MMP, suggesting that their mitochondria are less functional.

A t-haplotype targeted qPCR assay was established in order to test if there is a correlation between metabolic characteristics of sperm and its t-haplotype in heterozygous samples. Since the established method requires further optimization, we could not conclude with certainty if subpopulations of sperm with different MMP are enriched in a specific haplotype. At any rate, our preliminary data do suggest that this is indeed the case.

Key words: transmission ratio distortion, non-mendelian inheritance, t-haplotype, sperm metabolism