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Vitamin K2, as electron carrier molecule, holds potential as novel therapeutic strategy in Parkinsons' disease

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In a genetic screen for modifiers of Parkinson disease (PD) in fruit flies we isolated Heix, the Drosophila orthologue of human UBIAD1. This enzyme localizes to mitochondria and converts vitamin K1 to vitamin K2. Vitamin K2 is best known as a cofactor in blood coagulation, but in bacteria it is a membrane-bound electron carrier. Given the connections between Parkinson's disease and mitochondrial activity we wondered whether vitamin K2 exerts a similar electron carrier function in eukaryotic cells as well. Using fly genetics and mitochondrial biochemistry, we show that vitamin K2 is necessary and sufficient to transfer electrons in Drosophila mitochondria. Heix mutants show severe mitochondrial defects that are rescued by vitamin K2 and similar to ubiquinone, vitamin K2 transfers electrons in Drosophila mitochondria, resulting in more efficient ATP production. Thus, mitochondrial dysfunction in Parkinson's disease models is rescued by vitamin K2 that serves as a mitochondrial electron carrier, aiding to maintain normal ATP production. Similar to the defects in PD mutant Drosophila mitochondria, reduced efficiency at the level of the electron transport chain have been observed in PD patients as well, and it will be interesting to test if vitamin K2 can be used as a therapeutic strategy in the treatment of PD patients that suffer from mitochondrial defects.

Biography

In 2008, Melissa Vos started his Ph.D. in the Laboratory of Patrik Verstreken to find modifiers of the Parkinson's related gene *pink1*. During recent years and in collaboration with Dr. Vanessa Morais, he have gained much experience in mitochondrial biology and experimental procedures. Following my Ph.D. graduation and our recent published paper where we find that vitamin K_2 can improve phenotypes in Parkinson models in flies, he is eager to test if vitamin K_2 exerts a positive effect in Parkinson's patients.

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