

Research Summary for Sierra Hansen

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The function of DJ-1 in the protection of mitochondria against oxidative stress in *C. elegans*

Parkinson's Disease (PD), once believed to have no genetic cause, has subsequently been shown to have a strong genetic component in some cases. Mitochondrial dysfunction is widely believed to be centrally involved in the etiology of PD. However, the mechanisms by which the mitochondria become pathologically damaged are incompletely understood. DJ-1 is a small protein that plays a role in preventing oxidative stress and mitochondrial dysfunction, and therefore may be important for understanding the molecular basis of PD. This study will examine the mechanisms by which DJ-1 partially localizes mitochondria under oxidative stress and helps to maintain their normal function. As the DJ-1 superfamily is distributed among all three domains of life, there are many potential model systems for its investigation. We will use the roundworm *C. elegans* as a model. One of the most thoroughly characterized multicellular organisms, the fate of all 959 of its somatic cells is known and mapped at each stage of development. Furthermore, *C. elegans* contains two DJ-1 homologues, DJR-1.1 and DJR-1.2, whose similarities to their single DJ-1 counterpart are 55% and 44%, respectively. We will characterize the role of DJ-1 in mitochondrial function using a combination of structural biological, biochemical, and cell biological/organismal approaches.