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Sp21-035

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Spermatozoan Metabolism as a Non-traditional model for the study of Huntington's Disease

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Huntington's Disease (HD) is a fatal autosomal dominant neurogenerative disease manifested through motor dysfunction and cognitive deficits. Decreased fertility is also observed in male patients, likely due to altered spermatozoan function, thus resulting in reduced fertilization potential. Although some pharmaceuticals are currently utilized to mitigate these symptoms, an effective treatment which halts pathogenesis of the disease is yet to be approved by the FDA. This review addresses the HD pathway in neuronal and spermatozoan metabolism, including gene and protein expression in both neurons and spermatozoa indicated in the pathogenesis of HD. More specifically, zinc-containing and zinc-ion interacting proteins are thought to regulate or be regulated by zinc ion homeostasis. Identification of genes and relevant diagnostic biomarkers including elements of pyruvate and mitochondrial complex-I-dependent respiration may be advantageous for early diagnosis, management, and treatment of the disease. This review aims to provide analysis of HD pathogenesis and explore the comparative role of zinc in both neuronal function and spermatozoan capacitation, a physiological process that grants spermatozoa the ability to fertilize. Ongoing studies aim to characterize the products of genes implicated in HD pathogenesis that are expressed in both neurons and spermatozoa to facilitate study of future treatment avenues in HD and HD related male infertility. The emerging link between zinc homeostasis and HD pathway could lead to new treatments and diagnostic methods linking genetic spermatozoan defects with somatic comorbidities.