The Pathogenesis of Friedreich's Ataxia Using Proteomic Analysis of DRG

Abstract

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Friedreich's Ataxia is the only known genetic disorder that requires the inheritance of two copies of the abnormal FXN gene to cause the disease. Both parents must carry the defective gene in order for the individual to get this disease. FA is a guanine-adenine-adenine trinucleotide repeat in the frataxine gene. The purpose of this experiment was to prove that there is a increase in the response of the dorsal root ganglion to the antibody from patients with Friedreich's Ataxia compared to patients without Friedreich's Ataxia. To prove this, completed immunohistochemistry with tissue lysates. Immunohistochemistry itself has many steps to it including tissue fixation, antigen retrieval, and the labeling of the slides. The last step of immunohistochemistry, or the labeling of the slides, will lead us to the next step in our experiment which the double label immunoflourescence procedure. In this procedure, we put anti-mouse IgG and KIT pY936 on the positively charged glass slides. Then we were able to look at these slides under a confocal microscope. From this, we were able to see that more nuclei

were responsive in the patients that had Friedreich's Ataxia compare to the patients without this disease.