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Magnetic nanoparticle/ magnetic fluid approach to control neuromuscular degeneration in Friedreich's ataxia

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In last two decades nanotechnology has brought a revolution in the advancement of material science, biology, biotechnology, genetics, medicine and many other areas of science and engineering. The ability of nanotechnologist to prepare material particles of nano size, coating them with a suitable surfactant and magnetizing these particles has made possible to study micro level biological entities such as cells, genes and proteins. In recent years magnetic nanoparticles have been used in cancer treatment, drug delivery and in many other medical fields. This leads us to think why this technology should not be introduced in F.A. research for which no treatment is available and patients suffer with lethal neuro- degenerative condition of F.A. making them totally dependent on others for even the simple day to day activities. The disease is genetic and unknowingly passes on in off-springs through the carrier parents. It is therefore necessary to control degeneration in F.A. patients and make their life comfortable. Friedreich's ataxia (FRDA) is an autosomal recessive inherited disease caused by (GAA) expansion of the *FXN* (frataxin) gene. FRDA is predominantly a neurodegenerative disease. It manifests in initial symptoms of poor coordination such as gait disturbance; it can also lead to scoliosis, heart disease (cardiomyopathy) and diabetes. *FXN* gene produces frataxin protein which is localized to the mitochondria. The function of *FXN* is not entirely clear, however, the primary role of *FXN* protein is the activation of iron-sulfur (Fe-S) cluster biogenesis in the mitochondria. In F.R.D.A. production of *FXN* protein is reduced. In affected individuals level of *FXN* drops approximately 5 to 30% than healthy individuals. The role of *FXN* protein is that of an iron chaperone, a companion of iron particle. Low *FXN* levels lead to insufficient biosynthesis of iron-sulfur clusters that are required for mitochondrial electron transport chain to ultimately generate adenosine triphosphate (ATP), the energy packet necessary to carry out metabolic functions in cells. *FXN* also regulates iron transfer in the mitochondria in order to provide a proper amount of reactive oxygen species (ROS) to maintain normal processes without *FXN*, the energy in the mitochondria falls, and excess iron causes extra ROS to be created, leading to further cell damage. Nanotechnology can be useful to solve this problem. We can introduce body compatible magnetic nanoparticles to pickup iron particles and direct them to sulphur to form iron-sulphur clusters. Thus iron-sulphur clusters can be formed even in the absence of *FXN* protein in F.A. patients. And thus the process of respiration in mitochondria will continue to produce energy in the form of ATP. As iron particles are utilized in formation of iron-sulphur clusters no free iron is left to get deposited on cells and cause cell death in FRDA patients.

Biography

Swasti Wagh has completed her MSc in Applied Mathematics in the year 1999. She is fighting with F.A. since last 25 years and now she is on a wheelchair. She has some research publications on Magnetic Fluid. Recently, her paper on Mathematical Modeling of F.A. is accepted for publication. Since last 10 years, she is organizing camps for ataxia awareness and for the welfare of ataxia patients. She gave a presentation at SNCI conference held at AIIMS, Delhi in Feb 2013 and a presentation in DSHD congress on FA held at AIIMS, Delhi in April 2015.

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