Week 3 Discussion Forum Peter’s post

For this discussion, I chose Huntington’s Chorea as my topic.

Huntington’s Chorea is a neurodegenerative disease that has no cure. Treatment is confined to symptom management. HD attacks cognitive, behavioral, and motor systems in the afflicted, and is a progressively degenerative disorder. This neurodegenerative disease is genetic, and the hallmark symptoms are the decline of normal functioning, increased disability, and a progressive loss of independence, including the uncontrolled movement of the upper body including the arms, in the lower body the legs, as well as uncontrolled movement in the face and head, which is where the term (chorea) comes. Uncontrolled, and involuntary body movement is another hallmark of Huntington’s Disease. For the Huntington’s patient, extensive care is inevitable. Additionally, a gradual, but eventual decline in mood and behavior as well, as depression, anger, anxiety, and irritability generally follow a diagnosis of Huntington’s Disease.

The diagnosis for Huntington’s Disease is usually made through a genetic test. However, a doctor might evaluate medical history, family history, and closely examine the symptoms to possibly rule out other degenerative illnesses to make an informed evaluation. “Using a blood sample, the genetic test analyzes DNA for the HD mutation by counting the number of CAG repeats in the huntingtin gene.” (About Huntington’s Disease, 2011)

Ultimately, a decline in memory, reasoning capability, judgment and concentration, and being aware and cognizant of one’s surroundings because of the memory deficiency that can occur with Huntington’s. Another symptomatic aspect of Huntington’s is obsessive-compulsive behavior. The patient may repeat things or attempt the same tasks over and over again, as well as repeating questions or statements.

“Huntington’s disease (HD) is a rare, genetic, neurodegenerative, and ultimately fatal disease with no cure or progression-delaying treatment currently available. HD is characterized by a triad of cognitive, behavioural and motor symptoms.” (Ohlmeier, Saum, Galetzka, Beier, &Gothe, 2019, p. 1) As stated in the article, Huntington’s has no real, specific treatment to stave off the progression of the symptoms, nor is it yet known exactly what causes this genetic effect in a particular chromosome. What is know is that a single chromosome carries this defect. “Huntington's disease is a progressive brain disorder caused by a single defective gene on chromosome 4 — one of the 23 human chromosomes that carry a person’s entire genetic code.” (Huntington’s Disease, 2020, par. 1)

The general onset of Huntington’s Disease is somewhere between the ages of 40 and 50 years. Additionally, and unfortunately, from the time of diagnosis, the average length of time for survival is between 10 and 15 years as Huntington’s Diseased if a fatal disease with no cure or substantial treatment yet. The answer or positive findings in research have to this point evaded researchers, but with the advent of gene technology, it is hopeful that in the near future, more positive forms of treatment, and perhaps, the knowledge to turn off the defective actions of a particular chromosome, such as chromosome 4 that is known to be the culprit in Huntington’s Disease.

This Huntington’s disease (HD) is a neurodegenerative disorder due to autosomal inheritance. Its inception is normally found between 40 and 50 years of age. It is reported in the year 1983 that the gene for HD represents from chromosome number 4 and in the year 1993, its gene was unravelled. (Srirama, Vineela, Tejaswi, &Thanmavi, 2019, p. 2) Although research has illuminated the dark with knowledge regarding the genetic inheritability of Huntington’s Disease, and more is known about where the disease originates, research has not yet discovered a way to reverse this defect, nor to develop any significant treatment to slow its progress.

The degeneration of neurons is at the root of causing Huntington’s Chorea. Specifically, the degeneration of the GABAergic and acetylcholinergic neurons provide a lack of control being output by these neurons, which subsequently allows abnormal signal processing that monitors motor control.

The symptoms of Huntington’s disease are produced by the degeneration of GABAergic and acetylcholinergic neurons in the caudate and putamen. The loss of inhibition provided by these GABA-secreting neurons increases the activity of the GPe, which then inhibits the subthalamic nucleus. As a consequence, the activity level of the GPi decreases, and excessive movements occur. (Carlson, & Birkett, 2017, sec. 8.3)

References

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