Huntington??™s disease

Huntington??™s DiseaseHuntington??™s disease is a genetic disease that causes degeneration of the nerve cells within the brain. Unfortunately, at this time there is not cure for the disease. This disease was discovered in 1872 by Dr. George Huntington. Hence, this is why the disease was named after him. Huntington??™s disease is one of the more tragic movement disorders because it does not show symptoms until later in life (late 50??™s or later), and if a parent has this disease the child is 50% more likely to acquire the disease. Structure(s) Involved in Huntington??™s DiseaseHuntington??™s disease is produced by a genomic deficiency in the chromosomes.

According to PubMed (2011): The defect causes a part of DNA, called a CAG repeat, to occur many more times than it is supposed to. Normally, this section of DNA is repeated 10 to 28 times. But in persons with Huntingtons disease, it is repeated 36 to 120 times. The cells that are affected are in the basal ganglia, which are structures deep inside the brain that control synchronizing movement. In the basal ganglia, Huntington??™s disease targets the neurons of the striatum, generally those in the caudate nuclei and the pallidum (Mollersen, Rowe, Larsen, Rognes, & Klungland, 2011). The brains cortex is also agitated, which controls cognitive reasoning, insight, and recollection.

Neurotransmitter System(s) Involved in Huntington??™s DiseaseThe main neurotransmitters involved in Huntington??™s disease are: dopamine, glutamate, and GABA. Dopamine controls thinking, hormone secretion, and ??? has been implicated in the control of emotions and feelings of pleasure and euphoria??? (Wilson, 2012). In Parkinson??™s disease

dopamine is lacking, and in Huntington??™s disease dopamine is malfunctioning.

This malfunctioning causes the body to jerk and move irrepressibly. Glutamate is an amino acid neurotransmitter. ??? Unfortunately, because amino acids are found inside all cells in the body, scientists overlooked their role as neurotransmitters for many years??? (Wilson, 2012). The CAG, as mentioned earlier, is naturally occurrence in cell division in reproduction. However, scientists hypothesize that people with Huntington??™s disease have an overproduction of glutamate that causes a form of toxicity (Mollersen, Rowe, Larsen, Rognes, & Klungland, 2011).

GABA ??? is considered to be the most important inhibitory neurotransmitter in the brain??? (Wilson, 2012). In people with Huntington??™s disease, GABA is the first neurotransmitter to be decreased. This set the stage for the other two neurotransmitters to go awry. Eventually that individual will develop other symptoms such as: moodiness, psychosis (often diagnosed as dementia in older adults), unsteady gait, abnormal facial movements, speech impairment, and/or confusion. Current Treatments for Huntington?? ™s DiseaseThe current treatment for Huntington??™s disease is to treat the symptoms and try to slow the progression of the illness. Regrettably, this illness is terminal, so most doctors will try to make their patients as comfortable as possible when the treatment options fail. ??? Many agents and surgical procedures have been evaluated in HD for their antichoreic efficacy, including dopamine-depleting agents, dopamine antagonists, benzodiazepines, glutamate antagonists, acetylcholinesterase inhibitors, dopamine agonists, antiseizure medications, cannabinoids, lithium, deep

brain stimulation and fetal cell transplantation??? (Frank & Jankovic, 2010). There is one drug that is only approved in the USA by the FDA called Xenazine (Tetrabenazine in the generic form).

This drug works by judiciously reducing dopamine. The Future Direction for Treatment of Huntington??™s DiseaseThe future of Huntington??™s disease lies in the hands of the researchers that are constantly trying to ascertain the cure to this devastating disease. Stem cell research is the future cure for Huntington??™s disease. Stem cells are nothing short of a miracle. They have the capability of changing into different cell types early in human development. They also have the ability to repair themselves and many tissues throughout the body. This is why scientists highly desire the green light to study how they can help people fight diseases that otherwise will eventually end up killing them.

Specifically, human embryonic stem cells (hESCs) are originated from embryos that have been produced from eggs that have been fertilized from a process called in vitro fertilization. This is why there are so many ethical issues regarding stem cell research. According to Gogel, Gubernator, & Minger (2011) the future of Huntington??™s disease and other neurological diseases is still in progress: * The necessity for cell replacement therapy is stimulated by the rising incidence of degenerative diseases based on demographic changes in an ageing society. * Different neurodegenerative diseases might require different cell sources that have yet to be determined. In addition, the optimal time point of implantation relative to disease onset will have to be a focus of future research. * Improvement of differentiation protocols will lead to more pure populations of transplantable cells derived

from either hESCs or even induced pluripotent stem cells. * Logistical, ethical, societal and economical hurdles have to be overcome in order to make cell replacement therapy available for the broad public.

ConclusionHuntington??™s disease is a terribly, tragic disease that is passed down generation from generation. There is currently not cure for this disease; doctors can only treat the symptoms. This disease affects the dopamine, glutamate, and GABA neurotransmitters in the basal ganglia located deep in the brain. Currently doctors prescribe various medications to address the multiple symptoms that are associated with Huntington??™s disease. Scientists and researchers are studying various methods regarding the use of embryonic stem cells to replace to malfunctioning cells in a person with this disease. In the future, Huntington??™s disease can be permanently eradicated if the ethical stipulations surround stem cell research is reevaluated.

ReferencesFrank, S., & Jankovic, J. (2010). Advances in the pharmacological management of Huntingtons disease. Drugs, 70(5), 561-571. doi: 10. 2165/11534430-000000000-00000Gogel, S., Gubernator, M.

, & Minger, S. (2011). Progress and prospects: stem cells and neurological diseases. Gene Therapy, 18(1), 1-6.

doi: 10. 1038/gt. 2010. 130Mollersen, L., Rowe, A.

D., Larsen, E., Rognes, T., & Klungland, A. (2011). Continuous and Periodic Expansion of CAG Repeats in Huntingtons Disease R6/1 Mice. Plos Pathogens, 7(2), 1-11. doi: 10.

1371/journal. pgen. 1001242PubMed. (2011).

Huntington??™s disease: Huntington chorea. Retrieved from http://www. ncbi. nlm. nih. gov/pubmedhealth/PMH0001775/Wilson, J. F. (2012).

Introduction to biological psychology. San Diego, CA: Bridgepoint Education, Inc.