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Exploring Drug Targets for Huntington Disease: A Comprehensive Analysis

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Description

Huntington Disease (HD) is an autosomal dominant neurodegenerative disorder and a common cause of mortality. It affects both sexes and is prevalent in all ethnic group globally. The incidence reported is 5-7/100,000 in western population. As the age of onset can range from 2-85 year with majority of the cases being diagnosed between 30-40 years of life, hence it can be grouped as adult-onset disorder. It is a progressive loss in the motor, cognitive and behavioral abilities. MRI scans reveals that mostly neurons of basal ganglia are affected that are involved in muscles helping in movements of body. The main cause of HD is expansion of CAG repeat in the *HTT* genes beyond the threshold size. Presently, there is no cure for HD and the current treatment include management of symptoms using antipsychotics and antidepressants. Thus, understanding the pathways and molecular pathophysiology might be helpful in designing the possible leads for drug discovery. The review focusses on current possible drug targets for HD and shed some lights on the use of NGS-based techniques to determine the etiology of HD that can be further used in the therapeutic design for HD Supportive therapy involving many forms of medical and social care is suggested in order to lessen the impact of symptoms on the patient. An understanding of the various pathways and cellular events that are disrupted in HD might be helpful in designing possible drug targets. But this is quite challenging in case of HD owing to the fact that precise function of *HTT* is still not clear and it interacts with several proteins and is involved in plethora of pathway. Cellular therapies can be prospectively used for restoring atrophied tissues and thus provides important therapeutic possibility. Stem cell transplantation strategy for HD treatments can be utilized for replacing the dysfunctional or lost neurons. For this methods for obtaining the optimal Neural Stem Cells (NSCs) are to be developed from various sources, like brain, Pluripotent Stem Cells (PSCs), and somatic cells of the HD patients By genetically engineering stem cells to over-express neurotrophic factors or preconditioning those with compounds that can stimulate the production of neurotrophic factors their effect can be enhanced. For instance, a recent study demonstrated that intra-cerebral transplantation of BDNF-overexpressing human NSC (HB1.F3.BDNF) into the contra-lateral side of unilateral Quinolinic Acid (QA)-lesioned striatum promoted migration, differentiation and functional restoration in HD rat model. The question that has to be considered with care is that which of these pathways and molecular events have a pivotal role in the pathology many studies have been carried out in an attempt to find possible drug target for Huntington. HD is a fatal condition that worsens with time and affects both the proband and their family. It has an impact not only physically but also psychologically and socially on individuals who experience it. Many articles have surfaced that have advanced our understanding of the illness, including its pathophysiology and natural history as well as patient management, which has improved over the past 20 years. The pathogenesis and development of HD are influenced by a variety of factors. A lot of clinical trials have failed, and there are currently few medicines available. Preclinical research is now being done on HTT-reducing gene-editing methods such as CRISPR-Cas9, transcription activator-like effector nucleases, and zinc finger proteins. However, these methods need the injection of viral vectors needs more regulations and trail before therapeutic use. Cerebrospinal fluid and serum biomarkers, such mutant HTT and neurofilament light chain, can predict and monitor the course of the illness and are among the earliest detected abnormalities in HD.