

Huntington's Disease: current therapies and future directions

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Abstract. Huntington's disease is a neurodegenerative disease that have significantly negative impact to cognitive function. In the worldwide range, approximately 5-10 individuals are affected per 100,000 people. At molecular level, the expanded CAG repeats lead to misfolding and aggregation of the huntington protein, which can interfere with cellular metabolism, including transcription, mitochondrial function, and other important physiological processes. Though scientists already have a well-established theory for the pathology of Huntington's Disease, no effected cure has been developed due to the heavy genetic base of the disease. Despite the genetic barrier to overcome, many therapies have been created to alleviate the symptoms. In this primer, four main therapies are discussed who reduce the mutant huntington protein amount at post-transcriptional level: Antisense Oligonucleotides, Ethyl-Eicosapentaenoic Acid, autophagy modification, and intrabody based immunotherapy. Within each module, it is described how these therapies can reduce the level of mHTT in molecular level and correct the symptom. Development history is also touched upon briefly and discussion about the current status of each approach is made. Clinical prospective and future direction is included at the end as well.

Keywords: Huntington's Disease, Neurodegenerative Disease, Therapy.

1. Introduction

Neurodegenerative disease is one of the biggest challenges in modern society, where patients progressively loss their neuronal cells both structurally and functionally. Among the prevalent ones, Huntington's disease is one of the most well-known and studied one. In almost all cases, Huntington's Disease is inherited from parents in an autosomal dominant fasion. The disease can start at any age, but clinical data has shown that most patients usually begin their symptoms in middle age (30-50 years old), and gradually increase the severity. Early symptoms include slight mood or mental disabilities and, as the disease progresses, uncontrollable movements can occur [1].

At molecular level of the disease, mutations occur in huntingtin protein (HTT), which is an essential protein for neuronal development. In all cells types HTT is detected, while its specific functions are unclear. Nonetheless, its interactions with proteins involved in transcription, cell signaling, and intracellular transporting were discovered. Particularly, the gene contains multiple repeats of cytosine-adenine-guanine (CAG) in series, forming a structure called polyglutamine tract. Generally, people have fewer than 27 repeated glutamines in the polyglutamine tract is diagnosed as normal. Once the glutamine repeat exceeds 36 times, this conformation will cause the HTT protein to misfold. As a result, the misfolded protein forms aggregation and ultimately leads to neuronal cell dysfunction. The prevalence of HD varies across different regions around the world, with an average of 5-10 cases per 100,000 people [2]. Though for many years, extensive effort and worked have been done, no effective cure is developed for HD and other similar neurodegenerative diseases. The major obstacle for developing a cure is the genetic basis for the disease and the late onset of symptoms. In medical genetics, many pre-natal genetic screen and risk evaluation does reduce the disease population considerably, but large number of patients still exist in the world, which further raise the importance of developing an effective approach against HD. Fortunately, several approaches are widely accepted to ameliorate the symptoms. In this article, four different types of therapies (antisense oligonucleotide, Ethyl-EPA, autophagy regulation, and intrabody) are discussed in terms of their mechanism, current status, and prospect.

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