Mutant huntingtin disrupts the nuclear pore complex in Huntington's Disease. The current study conveys obscure cellular mechanisms and provides a basis for developing treatment.

Stop looking to see whether your skin is clear, because you may be looking at the wrong pores! Huntington's Disease (HD) is a neurodegenerative disorder that is characterized by rapid, involuntary successions of muscular contraction and relaxation. The disease is caused by the abnormal replication of CAG, a 3-base sequence within the genetic code (DNA) of the huntingtin gene (HTT). HTT genes include a range of 9-35 CAG sequences, but any number that exceeds that range results in Huntington's Disease. The time of HD onset depends on the number of CAG sequences, as indicated by juvenile HD (JHD) patients carrying at least 75 CAG repeats (Saudou and Humbert, 2016). Consequently, the amino acid sequence, which is the protein's structural code, develops extensive chains of the amino acid glutamine that are characteristic of the mutant huntingtin protein (mHTT). mHTT is cut into fragments which then form toxic bundles called aggregates, which cause the destruction of countless cellular components. Damage by mHTT aggregates primarily takes place in the basal ganglia (BG), a set of brain structures that encode the decision to move. One of the structures of the BG, the striatum, consists of neural cells called spiny projection neurons (SPNs). SPNs exhibit patterns in which they ýre in one striatal region but cancel their ýring in the other. Such patterns are implicated in the selection of a motor sequence to induce speciýc movements (Purves et al., 2018). SPNs sustain the most damage by mHTT, and their degeneration corresponds to the emergence of unwanted involuntary movements in HD.

Transport between the nucleus and cytoplasm (nucleocytoplasmic transport or N/C transport) allows for the entry of ions and small pro-

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