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科苑快讯

科学家发现亨廷顿氏症导致的分子破坏

荷兰乌得勒支大学(Utrecht University)的科学家发现，由突变的亨廷顿蛋白(huntingtin protein)聚集而成的有毒蛋白会破坏核膜，导致神经元中的DNA损伤和基因失调。这一突破可能将类似的机制与其他神经退行性疾病联系起来，揭示了神经元损伤的共同途径，并为治疗干预提供了潜在的新靶点。

亨廷顿氏病(Huntington's disease)是一种毁灭性的神经组织生长异常病变，由HTT基因突变引起，会使细胞产生异常大量的亨廷顿蛋白，它们聚集在细胞内并破坏细胞。但是，其导致神经细胞死亡的机制尚不清楚。

核膜(nuclear envelope)是保护和调节细胞核内染色体的屏障，允许其根据需要打开、关闭基因。研究人员发现，亨廷顿蛋白破坏了核膜底层的蛋白质网，使核膜更容易破裂。研究人员通过专业技术看到了微小的原纤维从聚集体(图中绿色部分)中伸出，穿过核膜下面的网状结构(图中洋红色部分)。这可能损害细胞在核膜破裂后重新密封的能力，严重损害了核膜的屏障功能，随着时间推移，还可能导致细胞DNA的损伤和神经元基因的失调。

包括某些类型的肌萎缩性侧索硬化症(amyotrophic lateral sclerosis)、额颞叶痴呆(frontotemporal dementia)在内的其他几种神经变性疾病，都与细胞核内蛋白聚集体的形成有关。研究人员推测，核聚集体诱导的核膜破裂，是神经退行性病变的一个共同因素，它引发了一系列失控过程，最终导致神经元死亡和神经炎症。

(高凌云编译自2024年8月16日SciTechDaily网站)