



Introduction

Creutzfeldt-Jakob Disease (CJD) is a rare, degenerative brain disorder that belongs to a group of human and animal diseases known as transmissible spongiform encephalopathies (TSEs). Among its various forms, Sporadic Creutzfeldt-Jakob Disease stands out as the most common, yet mysterious manifestation of this devastating

trigger [3]. This inherent unpredictability has rendered the disease particularly challenging to study and understand.

Pathogenesis: Unraveling the Role of Prions

Central to the pathogenesis of sporadic CJD is the accumulation of abnormal prion proteins within the brain. Prions, misfolded proteins that can induce other proteins to adopt their aberrant conformation, propagate a cascade of pathological events, leading to neuronal damage and cell death. In sporadic CJD, these rogue prions proliferate unchecked, causing widespread neurodegeneration and the characteristic clinical manifestations of the disease.

Clinical Presentation: The Rapid Descent into Neurological Decline

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