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Sommario/riassunto	<p>Proteinopathy is a collective term used to classified neurodegenerative diseases associated with the progressive accumulation of toxic protein molecules in specific brain regions. Alzheimer's disease (AD) is a well-known proteinopathy characterize by the accumulation of A peptides and tau proteins. The accumulation of these toxic molecules in the brain starts many years before any clinical presentation, being the onset in the range of 65 to 72 years of age. Therefore, age is considered a risk factor due, in part, to the loss of molecular competence to clear the brain from these toxic protein molecules. This fact, supported by years of research, demonstrates that brain cells activate a neuroprotective mechanism upon detection of a pathobiological signal that (if the detrimental conditions persist) precedes the activation of the neurodegeneration pathway. The progressive brain region specific neuronal death in neurodegenerative diseases also indicates that the transition from neuroprotection to neurodegeneration is individually triggered in cells of the affected brain region. Thus, molecular understanding of the pathophysiology associated with proteinopathies needs to take in consideration this intricate transition process, especially when genomics and proteomics approaches are used. Research directed to understand the pathogenesis and pathophysiology of neurodegenerative diseases uncovered the putative role of different molecular mechanisms associated with neurodegeneration. Among the molecular mechanisms</p>

identified are proteolysis, epigenetics, microRNA, transcriptional regulation, innate and adaptive immune system, phagocytosis and autophagocytosis, exo/endocytosis, unfolded protein response, cytoskeleton defects, unregulated signaling molecules (i.e. kinases and phosphatases), trafficking molecules, cell cycle, neurogenesis/neurodevelopment, among others. Interestingly, all these molecular mechanisms have been identified through the analysis of tissue from animal models or human post-mortem pathologically confirmed cases, but their specific role in neurodegeneration is still unclear. Thus, it is plausible to consider that all these pathways play a role at a particular phase of the neurodegeneration process or, simply, are driven by the agonal state of the tissue examined. Hence, an important conundrum that researchers face today is the use of heterogeneous brain tissue samples in the quest to identify biomarkers associated with the pathogenesis or pathophysiology of neurodegenerative diseases. At this junction of the neurodegeneration field, this research topic aims to critically assess the current literature on molecular mechanisms associated with neurodegeneration and the approaches used to dissect their putative pathophysiological role. The studies could include the interplay between neuroprotective and neurodegenerative signals in neurodegeneration, dissecting the molecular role of identified biomarkers, bioinformatics tools that facilitate data mining, dissecting pathways or molecular mechanisms, stages of protein aggregation (oligomers vs tangles; who did it?), aging brain and brain fitness (A natural selection process), adaptive protein response to environmental insults and cellular signals, expression profile associated with neurological disorders and health. Therefore, this Research Topic is expected to cover a wide range of subjects related to unravel the interplay between neuroprotective and neurodegenerative signals in neurodegeneration.

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