Prions: involution and adaptation or just proteins?

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Abstract. In this paper we aim to briefly and concisely discuss what prions are and what their possible origins are. Prions are unconventional infectious agents associated with neurodegenerative diseases. They lack genetic material and are primarily composed of misfolded proteins. The most plausible origin involves the spontaneous misfolding of normal cellular proteins, leading to the formation of prions. This misfolding may occur due to genetic mutations, environmental factors, or other triggers. Prions can also be transmitted between individuals through the consumption of infected tissues. Despite their atypical nature, prions have evolved a unique mechanism for propagation by inducing conformational changes in healthy proteins. While the precise details of prion evolution remain a subject of ongoing research, their distinctive features challenge conventional notions of infectious agents and contribute to our understanding of complex biological processes.
Key Words: atypical infectious agents, proteinaceous compounds, misfolded proteins.

Introduction. Sometimes it is difficult for us to precisely classify certain animal taxa (Petrescu-Mag & Popa 2018; Păpuc et al 2022), and sometimes it is difficult to separate living things into kingdoms (Petrescu-Mag & Proorocu 2022a). An equally difficult task is to draw a line between the living and the non-living (Petrescu-Mag & Proorocu 2022b). In this paper we aim to briefly and concisely discuss what prions are and what their possible origins are.

Prions. Prions are atypical infectious agents that cause neurodegenerative diseases in their host organisms (Artikis et al 2022). What sets prions apart from most other pathogens, such as bacteria, viruses, or fungi, is that they lack genetic material like DNA or RNA (Artikis et al 2022). Instead, prions are abnormal proteinaceous compounds that can induce changes in the structure of normal proteins in the host organism (Kraus et al 2021) (Figure 1).

The diseases caused by prions are collectively known as transmissible spongiform encephalopathies (TSEs) (Artikis et al 2022; Crestini et al 2022). These diseases primarily affect the brain and central nervous system. One well-known example of a prion disease in humans is Creutzfeldt-Jakob disease (CJD) (Kraus et al 2021), which can lead to severe neurological symptoms, including memory loss, behavioral changes, and impaired muscle coordination.

Prions can be transmitted through the consumption of infected tissues, and these diseases can affect various species, including humans, cattle, and other mammals (Kraus et al 2021). The study of prions is a complex field of scientific research, and understanding them contributes to the development of diagnostic methods and strategies for preventing the spread of prion diseases (Crestini et al 2022).

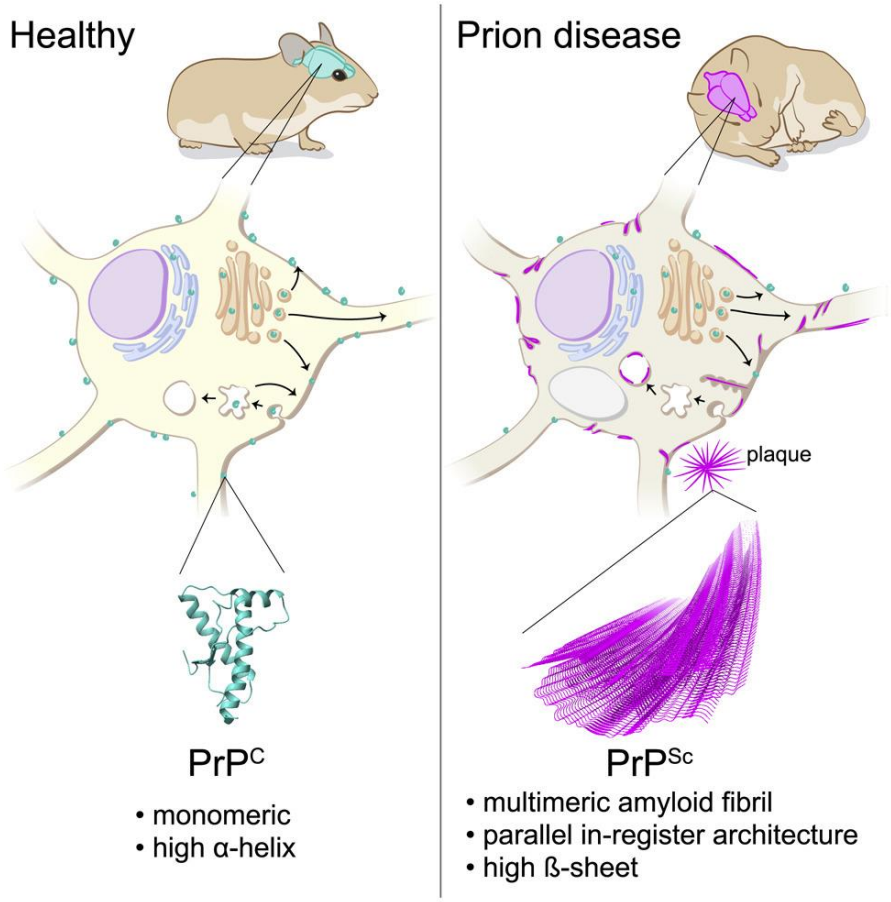

Figure 1. Prions as pathogenic infectious agents (Kraus et al 2021).

How they reproduce? Unlike typical infectious agents such as bacteria or viruses, prions do not reproduce in the traditional sense because they lack genetic material like DNA or RNA. Instead, prions propagate by inducing conformational changes in normal, healthy proteins within the host organism (Artikis et al 2022).

The normal proteins, when exposed to the misfolded prion proteins, can adopt the abnormal prion conformation. This conversion process is thought to involve the transformation of a normal protein into a misfolded, infectious form (Artikis et al 2022). The accumulation of these misfolded proteins disrupts the normal cellular function, especially in the brain, leading to the neurodegenerative effects associated with prion diseases (Kraus et al 2021; Crestini et al 2022).

The ability of prions to induce the misfolding of normal proteins is what allows them to spread and perpetuate within an organism. This process does not involve the traditional replication mechanisms observed in living organisms with genetic material. Instead, it is a self-propagating conformational change that leads to the accumulation of misfolded proteins, causing damage to the nervous system and other tissues (Crestini et al 2022).

Involution of viruses or just proteins? The idea that prions represent a form of involution, or a regression to a simpler state, in terms of infectious agents is not a widely accepted or mainstream scientific viewpoint. Prions are considered unique and distinct entities with their own set of characteristics and mechanisms of action.

Prions are abnormal, misfolded proteins that have the ability to induce similar misfolding in normal, healthy proteins (Caughey et al 2022). This process leads to the accumulation of these misfolded proteins, causing neurodegenerative diseases (Crestini et al 2022). Prions lack genetic material and do not fit into the traditional classification of living organisms, as they do not have cells, metabolism, or other features associated with living entities (Petrescu-Mag & Proorocu 2022b).

The study of prions has raised interesting questions about the nature of infectious agents and the definition of life (Jheeta et al 2021; see also Harris & Hill 2021 in the case of viruses). However, it is more accurate to view prions as a unique class of infectious agents with distinct properties (Cortez et al 2022; Sprunger & Jackrel 2021), rather than as a form of involution or regression from more complex living entities. Scientific understanding of prions continues to evolve, and researchers are actively investigating their structure, behavior, and the mechanisms by which they cause disease (Sprunger & Jackrel 2021).

However, what is the origin of prions? The origin of prions is not entirely clear, and it remains a topic of scientific investigation and debate (Cortez et al 2022; Galkin et al 2023). The most accepted hypothesis suggests that prions originate from normal cellular proteins that undergo a conformational change, adopting an abnormal, misfolded structure. There are several theories about how this misfolding might occur.

Spontaneous misfolding. Normal proteins may spontaneously misfold into the prion conformation due to environmental factors, genetic mutations, or other unknown triggers (Bohl et al 2023).

Infection from external sources. Prions can be transmitted between individuals through the consumption of infected tissues (Kamali-Jamil et al 2021). In this scenario, an external source introduces the misfolded prion protein into a new host, where it can induce the misfolding of normal proteins.

Genetic predisposition. Some individuals may have a genetic predisposition that makes them more susceptible to prion misfolding. Mutations in the gene encoding the normal cellular protein may increase the likelihood of it adopting the misfolded prion conformation (Sanz-Hernández et al 2021).

Environmental factors. Certain environmental conditions may influence the likelihood of prion misfolding (Bartz 2021). For example, exposure to specific substances or changes in the cellular environment could contribute to the conversion of normal proteins into misfolded prions.

While these hypotheses provide some insight, the precise mechanisms underlying the initial formation of prions are still not fully understood. Research in this area continues to explore the molecular processes involved in prion formation and transmission.

Conclusions. Prions are unconventional infectious agents associated with neurodegenerative diseases. They lack genetic material and are primarily composed of misfolded proteins. The most plausible origin involves the spontaneous misfolding of normal cellular proteins, leading to the formation of prions. This misfolding may occur due to genetic mutations, environmental factors, or other triggers. Prions can also be transmitted between individuals through the consumption of infected tissues. Despite their atypical nature, prions have evolved a unique mechanism for propagation by inducing conformational changes in healthy proteins. While the precise details of prion evolution remain a subject of ongoing research, their distinctive features challenge conventional notions of infectious agents and contribute to our understanding of complex biological processes.

Conflict of interest. The authors declare that there is no conflict of interest.

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