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What makes a protein sequence a prion?

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Typical amyloid diseases such as Alzheimer'Vs and Parkinson's were thought to exclusively result from de novo aggregation but recently it was shown that amyloids formed in one cell can cross-seed aggregation in other cells or even individuals following a prion-like mechanism. Despite the large experimental effort devoted to understanding the phenomenon of prion transmissibility, it is still poorly understood how this property is encoded in the primary sequence. In many cases, prion structural conversion is driven by the presence of relatively large glutamine/asparagine (Q/N) enriched segments. Several studies suggest that it is the amino acid composition of these regions rather than their specific sequence that accounts for their priogenicity. However, our analysis indicates that it is instead the presence and potency of specific short amyloid-prone sequences that occur within intrinsically disordered Q/N-rich regions that determine their prion behavior modulated by the structural and compositional context. This provides a basis for the accurate identification and evaluation of prion candidate sequences in proteomes in the context of a unified framework for amyloid formation and prion propagation.

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Proteogenomics and the dark matter in proteomics

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The steady increase of sequencing data enabled the detection of non-coding transcripts as a normal component in the RNA ensemble of every eukaryotic cell. Beside a wide variety of shorter RNA sub-types there are also long and very long ncRNAs described. Based on new findings from ribosome profiling and bioinformatic analyses it has been proposed that some shorter sequence stretches show coding potential. Here we will present recent studies on proteogenomic analyses of microbes like Dehalococcoides and Helicobacter. In these studies the simple search of mass spectrometric data against the six frame translation of the genomic sequence is sufficient to unravel novel proteins or coding sequences. The experimental approach has also been supported by novel prediction tools like RNAcode. The validation has been done by SRM measurements. For typical model systems and especially those with very large genomes like human the bioinformatics challenge is much harder. We developed a stepwise filtering by using transcriptome data instead of genomic information and substraction of already known coding sequences. In this study we used transcriptomic data from polarized human immune cells and detected up to 300 novel coding sequences which some also are correlating with different immune cell sub-types. For a proper detection of novel coding sequences the stringency of the database search and the way of validation are critical issues and we will show the several layers in this process. In summary, the combination of proteomics and bioinformatics will enable the further development of the field of proteogenomics in the future.

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