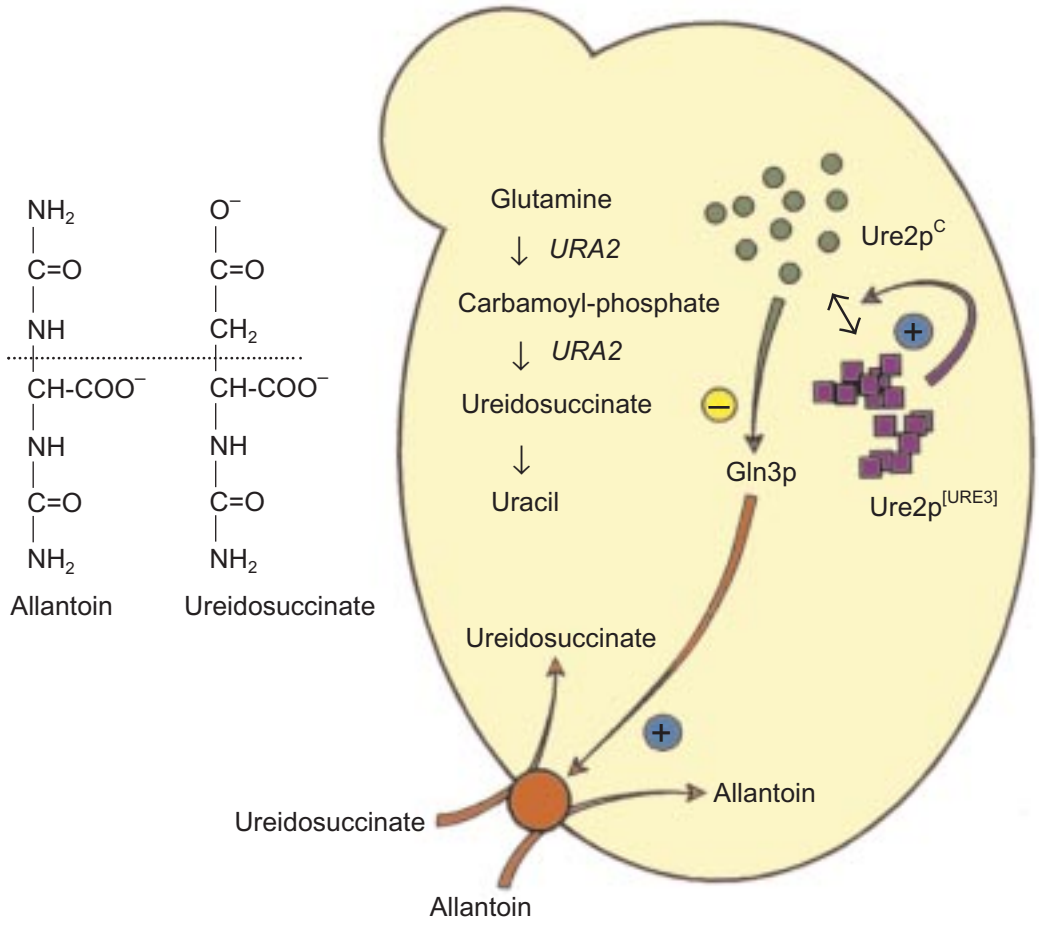

Fig. 1. The prion hypothesis for [URE3]. The [URE3] phenotype corresponds to a loss of function of $Ure2p^{C}$ but the genetic experiments argue for a model in which no mutations are involved. The protein would exist in a functional form ($Ure2p^{C}$) and also in a non-functional form ($Ure2p^{[URE3]}$). This altered form would have acquired the remarkable property of being able to convert the wild-type protein to the abnormal form, similar to the mechanism described for mammalian prions. Structural similarity between the poor nitrogen source allantion and the ureidosuccinate well explains the relationship between Nitrogen Catabolic Repression and ureidosuccinate entry.

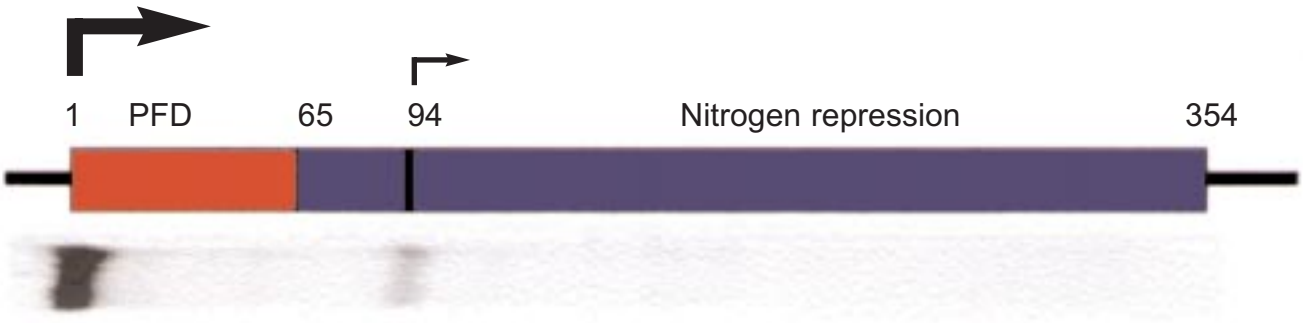

Fig. 2. Prion-forming domain (PFD) and nitrogen repression (catalytic) domains of Ure2p shown in red and blue as defined by R. Wickner [16]. At the bottom: Ure2p cell-free translation products (yeast extract)—full-length protein and 30-kD catalytic fragment. Arrows show the start points of translation initiation.

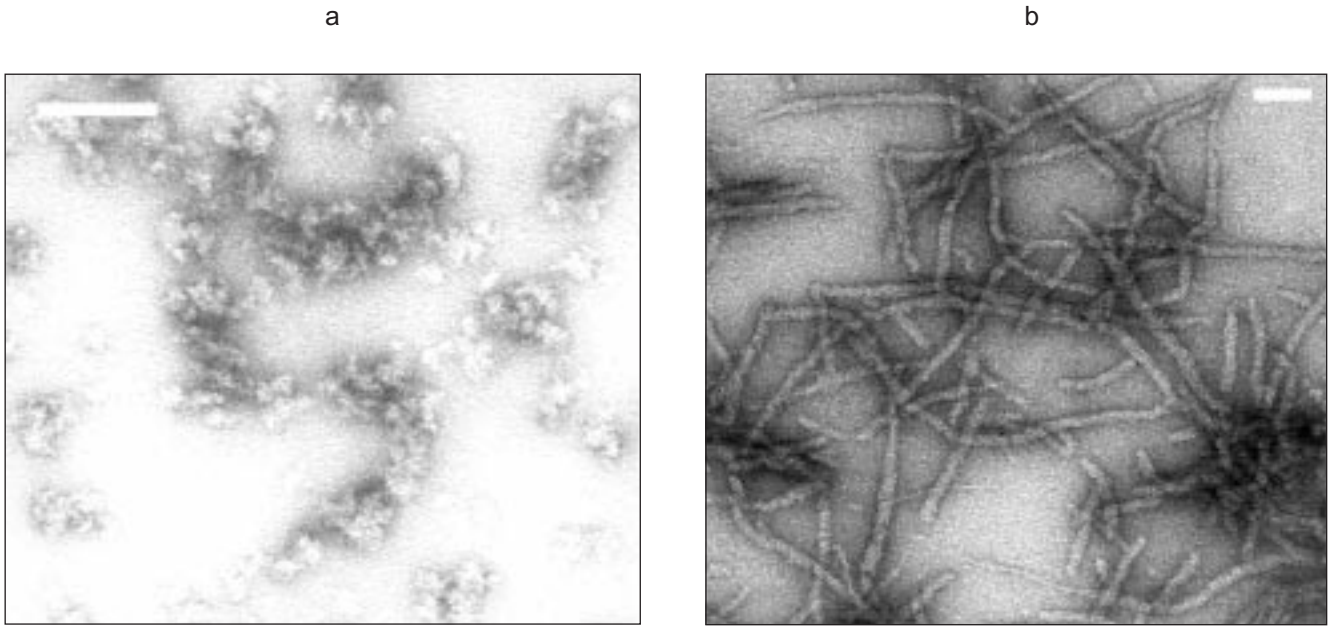

Fig. 3. Electron micrographs of negatively stained Ure2p oligomers. a) Ure2p assembly into high-molecular-weight heterogeneous oligomers induced by adjusting the pH of the solution from 7.5 to 6.4, the theoretical isoelectric point of the protein. b) Ure2p fibrils obtained upon autoassembly at pH 7.5. The white scale bar is 100 nm in (a) and (b).

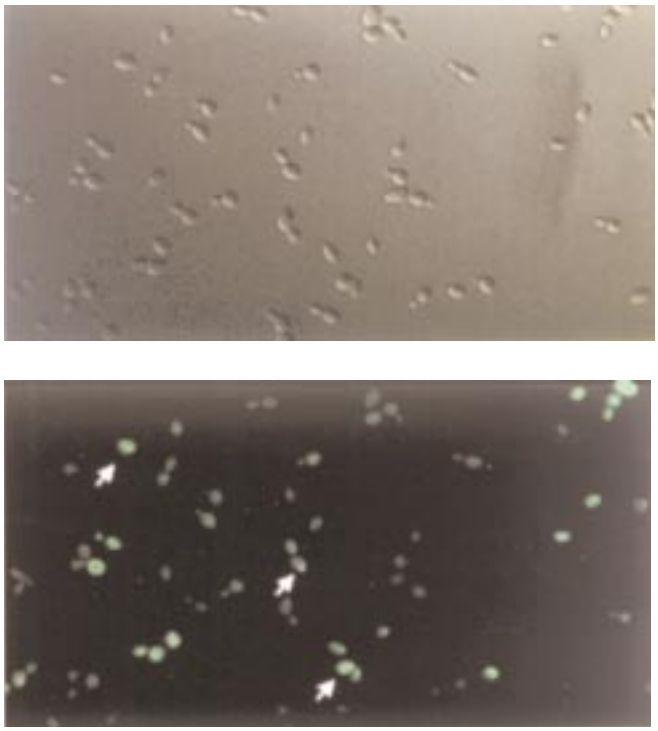

Fig. 4. Cellular distribution of Ure2–GFP fusion proteins. Yeast wild-type cells (strain CC30) were transformed with multicopy vectors allowing strong overproduction of chimeric proteins. Top, cells examined by light microscopy. Bottom, the same cells examined by fluorescent microscopy. Arrows point to cells bearing Ure2p–GFP aggregates.