Prion diseases occur when a misfolded form a protein appears and, rather than be cleared by cellular machinery, propagates by induces normally folded protein to adopt the misfolded conformation. The infectious unit of prion disease is a linear aggregate of misfolded protein termed an amyloid. Amyloid appearance is normally a rare event, but the presence of other aggregates promotes its formation. While these aggregates are thought to act by templating the formation of a stable amyloid nucleus, we find their primary role is downstream in promoting amyloid nucleus persistence. We develop a stochastic chemical master equation depicting the molecular dynamics in a yeast cell just after the introduction of an initial amyloid. We formulate the probability that prion disease successfully appears by considering the probability that the initial amyloid is lost in the initial cell and, through the analysis of these arrival time distributions, determine two common prion variants have different nucleus sizes. Our observations suggest that nascent nuclei are actively cleared by disaggregation below their minimum stable size to limit amyloid appearance in vivo and establish nucleus size as a key characteristic of amyloid conformational variants for the first time. (Received September 03, 2019)