Meriin et al. Vol. 157, No. 6, June 10, 2002. Pages 997–1004.

The following paragraph contains a citation (Osherovich and Weissman, 2001) mistakenly omitted in the original publication of the article.

Our data that the polypeptide with expanded polyQ forms inclusions only in the presence of the [RNQ^+] prion are consistent with the latest report showing that aggregation of the MJD protein with polyQ expansion is facilitated in so-called [PIN^+] cells (Osherovich and Weissman, 2001). [PIN^+] is a non-Mendelian genetic trait promoting emergence of [PSI^+] (Derkatch et al., 1997; Osherovich and Weissman, 2001). It was shown that [PIN^+] may be caused by a number of potential prion-forming proteins, for example, Rnq1 and New1 (Derkatch et al., 2001). Interestingly, deletion of the *NEW1* gene did not significantly affect aggregation of 103Q polypeptide, suggesting the major role of [RNQ^+] prion in this process. Whether aggregates of prion form of Rnq1, which has a QN-rich domain, could directly serve as a nucleation site for polyQ, or Rnq1 acts on the polyQ aggregation indirectly is yet to be established.

Osherovich, L.Z., and J.S. Weissman. 2001. Multiple Gln/Asn-rich prion domains confer susceptibility to induction of the yeast [PSI^+] prion. *Cell.* 106:183–194.