654.09 / E21 - The novel protein FAM171B is recruited into intracellular polyQ aggregates

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Authors
J. KIRLIN, *G. M. GOELLNER;
Minnesota State Univ., Mankato, MN

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J. Kirlin: None. **G.M. Goellner:** None.

Abstract
Expansion mutation within polyglutamine (polyQ) tract proteins is known to underlie a number of severe neurodegenerative disorders such as Huntington's Disease and Spinocerebellar Ataxia. One of the pathologic hallmarks of polyQ expansion disease is the aggregation of mutant proteins into intracellular inclusion bodies. Our lab is actively investigating FAM171B- a relatively uncharacterized protein that also contains a stretch of consecutive glutamines within its primary amino acid sequence and is likely expressed in the nervous system. Since it too contains a short polyQ stretch, we surmised that FAM171B may also be recruited into polyQ aggregates formed by known pathologic proteins. To test this hypothesis, we transfected cells with an expanded version of SCA7-GFP to form intracellular aggregates, and utilized FAM171B specific antibodies, immunofluorescence, and confocal microscopy to assay FAM171B's intracellular location in relation to the SCA7 inclusion bodies. Our findings indicate that a portion of FAM171B does indeed alter its normally dispersed cytoplasmic subcellular distribution into SCA7 aggregates that locate near the peri-nuclear region of human glioblastoma tissue culture cells. Thus, the novel protein FAM171B may play a role in the molecular mechanisms underlying polyQ disease pathology.

Abstract Citation