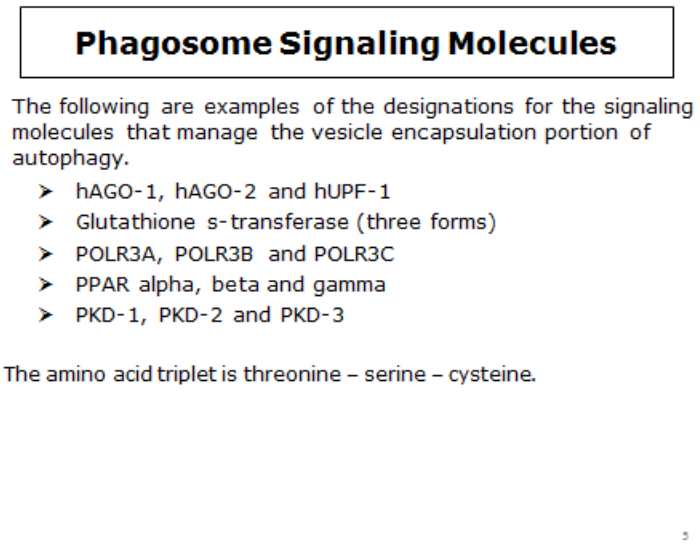
ALS: FUS Plaque

Modeling of cell surface signaling that is transferred into the cytoplasm (i.e. endocytosis)[1] has identified, with near certainty, that FUS plaque is selenium - zinc based with threonine - serine - cysteine as the amino acids and known as FOXM1.

Epigenetic modeling has identified FOXM1 as critical to activate phagosome processes in endocytosis. Accordingly, mutation of FOXM1 by excessive selenium could disrupt endocytosis with downstream consequences of preventing SOD1 (autophagy) from enabling the "kinase" to be "dissolved" with ALS being the outcome.

The following is provided for discussion purposes.



Phagosome Signaling Molecules

The following are examples of the designations for the signaling molecules that manage the vesicle encapsulation portion of autophagy.

- hAGO-1, hAGO-2 and hUPF-1
- Glutathione s-transferase (three forms)
- POLR3A, POLR3B and POLR3C
- PPAR alpha, beta and gamma
- PKD-1, PKD-2 and PKD-3

The amino acid triplet is threonine – serine – cysteine.

[1] http://www.mcfip.net/upload/Endocytosis%20Modeling%204-30-17.pdf