On the track of the white tiger: Pigmentation could be linked to prion diseases, and location explains why[1]

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Summary

Certain rodent pigmentation mutants spontaneously develop brain spongiform changes.
It is hypothesized that animals, and possibly humans, characterized by certain pigmentation gene variants could be more susceptible to prion diseases, which are characterized by this type of neuropathology.
This hypothesis could be explained by the common location of the prion protein and several important pigmentation genes in the same chromosome. This common location can promote the joint transfer of both pigmentary and prion protein genes to the progeny. Pigmentation genes could also play a role in regulating protein folding and aggregation.
Understanding the relationship between pigmentation genes and prion genes could lead to identify pigmentation variants at higher risk of prion diseases and understand the etiopathogenesis of these still invariably lethal disorders.

[1] Full text available online: MEDICAL HYPOTHESES (2009)
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