Somatic Mutation and Adaptive Flexibility: Neural Plasticity and the *NF1* Gene in Hominin Evolution and Beyond

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The unique, wild-type hominin variant of the *NF1* gene displays a strong association with skills fundamental to oral knowledge systems. The sequence of the *NF1* protein product has also shown remarkable utility in distinguishing phylogenetic relationships between primate species. Further study now suggests that *NF1* mutations play significant roles in adaptation at both the germline (intergenerational/speciation) and somatic (individual lifespan; e.g., acclimation, learning) levels.

First recognized for its role in the human tumor disorder Neurofibromatosis Type 1, homologs of the *NF1* gene have since been identified and studied in a diverse array of organisms including social amoebae, fruit flies, cephalopods, pigs, rodents, canines, and non-human primates. The *NF1* gene is associated with development of the nerve sheath and the regulation of cellular growth, proliferation and signaling. In fruit flies, rodents, and humans, germline defects in the *NF1* gene have been linked to deficits in communication, learning, and spatial reasoning.

In response to environmental factors, the social amoeba transitions between unicellular and multicellular structure, and asexual and sexual reproduction. *NF1* knockout amoebae are able to greatly expand their dietary options, at some expense to chemotactic motility. The fully-motile phenotype can be rescued through transition to a multicellular state, or through sexual reproduction. We describe here the somatic mechanism by which certain *NF1* alleles provide comparable adaptive neural flexibilities. These enable a plasticity in neural pathways that is especially enhanced by the paralogous *NF1* mutations of social, intelligent, and/or broadly-dispersed (flexibly-adapted) species and which produce, arguably, the greatest adaptive flexibilities in hominins.

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