Maastricht University

Excessive aggression, ADHD- and ASD-like phenotypes in TPH2- and brain ganglioside-deficient mice

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Propositions

1. Partial deficiency of neuronal serotonin following heterozygosity in the *Tph2* gene (*Tph2*$^{+/-}$) causes abnormal aggression and sociability in stressed male and female mice (*this thesis*).

2. These behavioral changes are accompanied by alterations in dopamine and norepinephrine metabolism, aberrant expression of myelination proteins, markers of stress, and plasticity (*this thesis*).

3. Downregulation of myelination marker PLP1 in *St3gal5*$^{-/-}$ mice deficient for brain gangliosides is associated with dominant behavior, and behaviors reminiscent of autism spectrum disorders (*this thesis*).

4. Inflammatory stress in *St3gal5*$^{-/-}$ mice leads to aberrant expression of pro-inflammatory cytokines in the brain and in the periphery, and aggravated aggressive/dominant behavior (*this thesis*).

5. Abnormalities in myelination and neuroinflammation may constitute common molecular mechanisms leading to behavioral abnormalities in the *Tph2*$^{+/-}$ and *St3gal5*$^{-/-}$ mice (*this thesis*).

6. Modeling of gene-by-environment interaction rather than of genetic or environmental factors alone is useful for animal studies of neurodevelopmental disorders (*impact*).

7. Different types of environmental adversities may lead to similar pathological changes contributing to neurodevelopmental disorders.

8. Multiple risk genes act on early stages of neurodevelopment and the formation of brain circuits, thus conferring broad liability to psychiatric disorders.

9. Validation of experimental animal models of behavioral features related to psychiatric disorders requires the use of multiple tests and a variety of experimental conditions.

10. “It’s not stress that kills us, it is our reaction to it.” — Hans Selye