Stellingen behorende bij het proefschrift

Fragile X-associated Tremor/Ataxia Syndrome: RNA or RAN

1. A key pathogenic event in FXTAS is the expression of a toxic polyglycine-containing protein, FMRpolyG, that disrupts the neuronal nuclear lamina architecture (*this thesis*).
2. FMRpolyG expression plays a role in the toxic gain-of-function mechanism underlying the medical co-morbidities in FXTAS (*this thesis*).
3. Mitochondrial dysfunction causes the early demise of inducible FXTAS mice without the presence of ubiquitin-positive inclusions (*this thesis*).
4. Early therapeutic intervention is essential for FXTAS patients (*this thesis*).
5. Small chemical compounds that shield the CGG repeat show great potential to ameliorate FXTAS symptoms (*this thesis*).
6. The molecular pathology of microsatellite disorders is more complex than is currently appreciated (*Zu et al., PNAS, 2011*).
7. Heritable human genetic modifications, including CRISPR/CAS technology, pose serious risks and the therapeutic benefits are tenuous (*Lanphier et al., Nature, 2015*).
8. Preclinical animal experiments investigating human autoimmune diseases have been invaluable for the discovery of key immune processes, basic disease mechanisms and candidate immune-targeting strategies with clinical utility (*Roep et al., Nature Medicine, 2012*).
9. Moderate coffee drinking at midlife significantly delays the onset of dementia in the sunset years (*Cao et al., Journal of Alzheimer's Disease, 2012*).
10. A DNA-based storage system is the ultimate data storage solution (*Bornholt et al., ASPLOS, 2016*).
11. Science is a marathon, not a sprint!

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Rotterdam, June 22th, 2016