Stellingen behorende bij het proefschrift

Selective Glucocorticoid Receptor Modulation:
TARGETING THE BRAIN UNDER STRESS

1. Manipulation of splicing or expression of glucocorticoid receptor-coregulators is a feasible approach to modulate glucocorticoid receptor-signaling in the brain (this thesis).
2. Shifting the expression ratio of the two SRC-1 isoforms in favour of SRC-1a results in abrogation of *crh* expression regulation by glucocorticoids *in vivo* in the central amygdala and impairment of fear memory consolidation (this thesis).
3. The application of selective GR modulators is a significant improvement in treatment of hypercortisolemia-related psychopathology compared to the treatment with the GR antagonist mifepristone (this thesis).
4. Targeting of the interactions between steroid hormone receptors and coregulators may result in tissue- and gene-specific regulation of their effects (this thesis).
5. GR in the central amygdala plays a critical role in mediation of pavlovian fear responses (Kolber et al, *PNAS,* 2008, Vol (105):33).
6. Set-point of HPA-axis activity depends on epigenetic regulation of c*rh* gene expression (Elliott et al, *Nature Neuroscience*, 2010, Vol (13):11).
7. Studying the role of SGK-1 and FKBP5 in GR signaling may reveal more possible targets for pharmacological manipulation of the action of glucocorticoids (Anacker et al, *PNAS*, 2013, Vol (110):21; Klengel et al, 2013, *Nature Neurosciene*, Vol(16):1).
8. "Uncovering processes underlying the transition from adaptation to pathology requires a precise definition of "adaptation" and "pathology," as many responses and behaviors that may appear "maladaptive" make perfect sense in the appropriate context" (J.P. Herman, *Front. Behav. Neuroscience*, 2013, Vol(7)).
9. Happy people do good science.
10. In Academia, the notion that time is money should be better implemented.

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