ADHD, trauma and attachment – A new etiological model

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**Introduction:**
ADHD is described as a multifactorial disease. Genetic factors as well as early environmental factors such as prenatal exposure to nicotine/alcohol but also quality of parental care giving and early deprivation play an important role in the development and course of the disorder. In recent etiological studies gene x environment (GxE) interactions are considered as probable mechanism by which those early-development environmental insults can lead to long term alterations in phenotype.

**Etiological model:**
Genetic polymorphisms involved in dopamine and serotonin neurotransmission, previously reported to be associated with ADHD, as well as disorganised attachment may mediate susceptibility to environmental adversity early in life. Parental insensitivity and unresolved mourning contribute to the development of attachment insecurity / disorganisation, insufficient emotional regulation and ADHD like symptoms. But which children are especially susceptible to early adversity factors and how do they effect HPA-Axis functioning as a neurobiological marker?

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**Attention-deficit hyperactivity disorder (ADHD) (Child)**

- Dopamine receptor gene polymorphism DRD4
- Gene - Environment Interaction
- Other candidate genes (DAT1, HTTLPR, DRD1-5 etc.)
- Unresolved trauma in child
- Unresolved trauma in mother

**Brain development (Child)**

- HPA - axis reactivity

**Conclusion:**
As the perinatal period seems to be particularly involved in both ADHD and attachment disorders, early guidance and possibly early interventions should be developed and assessed for mothers and caregivers with risk-factors, as well as for their children.

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