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**Capturing the Long-Term Sequelae of Child Maltreatment:
Neurocognitive Alterations, Complex Posttraumatic Stress Disorder
and the Impact of Psychotherapy**

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Approximately 70% of the general population experience a traumatic event during lifetime. However, only a small proportion develop Posttraumatic Stress Disorder (PTSD) (5.6%). Considerable efforts have been made to investigate individual differences as well as characteristics of the trauma itself that both may contribute to the development of PTSD. The experience of child maltreatment (CM) in contrast to the experience of a traumatic event during adulthood has repeatedly found to be correlated to significant higher rates of PTSD. On a neurocognitive level, cognitive dysfunctions together with functional and structural brain alterations seem to characterize individuals with PTSD compared to traumatized healthy subjects. Although a cumulative effect of trauma can be assumed, the role of type and timing of CM has become of particular interest when investigating neurocognitive correlates contributing to the development of PTSD. Emerging evidence points to sensitive periods and specificity of CM-subtypes to differentially impact neurocognitive correlates in individuals with and without PTSD. However, research is still at a very early stage. The development of PTSD in the aftermath of prolonged and severe CM is often associated with clinical features that extend beyond classic PTSD symptoms such as affective dysregulation, negative self-concept and disturbances in relationships. This complex form of PTSD (cPTSD) has therefore been included in the 11th revision of the World Health Organization's International Classification of Diseases (ICD-11). Numerous studies in PTSD patients related to various trauma types have already provided evidence for neurocognitive alterations. However, the empirical database on neurocognitive correlates of cPTSD is quite limited at this time. Understanding alterations in cognitive and neural processes could optimize treatments in order to improve long-term outcomes of individuals with cPTSD. Several psychotherapeutic approaches have been developed for PTSD treatment and have been shown to be successful in treating PTSD symptoms as well as neurocognitive alterations. However, those treatments have mostly been developed for survivors of adult-trauma PTSD. Meta-analyses demonstrated substantially lower effect sizes of psychotherapeutic treatments in CM-related cPTSD indicating poorer treatment response. Even though preliminary data point to the effectiveness of psychotherapy on normalizing neurocognitive correlates in cPTSD patients, these data are in clear need of replication. To fill this gap, the aim of the doctoral thesis was to examine the long-term sequelae of CM with an emphasis on neurocognitive correlates of CM-related PTSD and cPTSD and the impact of psychotherapy on these measures. For this purpose, three experimental studies were conducted. Two studies investigated the role of cPTSD and CM history on neurocognitive correlates. Study I investigated the role of psychopathology and CM history on functional correlates of cognitive control and emotional interference. Study II focused on the effects of CM on structural brain correlates with an emphasis on type and timing of traumatization. The third study aimed to examine whether 12 months of psychotherapy (DBT-PTSD or CPT) lead to an improvement of neurocognitive alterations in patients with cPTSD. In study I, patients with cPTSD showed poorer behavioral outcome and an increased need for activation within prefrontal cognitive control networks, while confronted with trauma-related stimuli as compared to healthy controls with and without CM history. After 12 months of psychotherapy (study III), the pathologically increased emotional interference in cPTSD patients was found to be "normalized" on both neuronal and behavioral measures (reflected in faster reaction times, less errors and decreased activation within limbic and prefrontal brain regions). It can be concluded that psychotherapy helped patients by working with trauma-related memories, cognitions and emotions to integrate new adaptive information to distinguish between threat and safety to habituate towards trauma-related material. Regarding structural brain correlates of CM, study II demonstrated a negative correlation between global CM severity and bilateral amygdala volume. Interestingly, this effect was

driven by the severity of neglect. Moreover, results point to an effect of timing of CM exposure at 10-11 years of age and 13 years of age, for both bilateral amygdala and hippocampal volume. Regarding type x timing analyses, results revealed sensitive periods during 10-12 years of age and 13-14 years of age for the severity of neglect, affecting right amygdala volume. Moreover, results point to a sensitive period during 14 - 16 years of age for the severity of neglect affecting left amygdala volume. Likewise, a sensitive time window for the severity of neglect were identified during 9-13 years of age, affecting bilateral hippocampal volume. The results of the present thesis provide further support that exposure to CM lead to long-term stress-induced cumulative changes in the neurobiological system. Moreover, the results provide further evidence for a type and timing model of CM, as a complementary approach in the understanding of the impact of CM across the entire lifespan on neurocognitive correlates. Longitudinal studies, however, are needed to get insight on causal relationships.