Early social deprivation-induced violent aggression: behavior, emotional background and neural substrates

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Disturbed social relations during childhood (e.g., social neglect) often lead to aggression-related psychopathologies in adulthood. Social isolation also increased aggressiveness in laboratory animals. We have recently shown in rats\(^1\), that social isolation from weaning not only increases the level of aggressiveness, but results in abnormal attack patterns and deficits in social communication. Moreover, socially deprived rats showed a marked increase in defensive behaviors, which can also be considered abnormal, as these rats also showed considerably more offensive aggression than their controls. We have suggested that the social deprivation-induced abnormal aggression models the aggression-related problems resulting from early social neglect in humans, and studies on its brain mechanisms may increase our understanding of the mechanisms underlying psychopathologies resulting from early social problems.

More recent data from our laboratory show that this type of abnormal aggressiveness is associated with a marked hyperarousal as shown by an exacerbated autonomic activation during aggressive encounters. Aggressive interaction-induced neuronal activation patterns were different from both those shown by control (socially non-deprived) rats as well as rats submitted to another model of abnormal aggression (hypoarousal-driven aggression). Interestingly, neurons of the hypothalamic attack area were over-activated in this model, which was not seen with any model studied earlier except for rats in which aggressiveness was induced by the stimulation of the hypothalamic attack area. The findings obtained with this model show that the brain mechanisms underlying normal rivalry aggression, hypoarousal-driven (glucocorticoid deficiency-induced), and hyperarousal-driven (social deprivation-induced) aggression are markedly different, suggesting that the treatment of aggression-related psychological disorders may be aggression type-specific.