

Stress-responsiveness and brain serotonin functioning in aggressive and non-aggressive feral rats and mice.

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This presentation summarizes the broad individual differences in aggressiveness that exist in a wide variety of animal species, including humans, and its relationship with several other behavioral, autonomic/neuroendocrine and neurochemical characteristics. Based on our observations in feral rats and mice that the individual level of offensive aggressive behavior (i.e., the tendency to defend the home territory) is strongly related to the way they react to various other social and non-social challenges, it is argued that the individual's level of aggressiveness is an important indicator/component of a more trait-like behavioral physiological response pattern (coping strategy or personality) to environmental demands. The coping style of aggressive animals is principally aimed at a (pro)active control or manipulation of a stressor whereas the non-aggressive individuals tend to passively accept or shy away from it. The aggressive/proactive and docile/passive behavioral coping styles are clearly associated with distinct patterns of autonomic/endocrine (re)activity that are highly functional and adaptive to physiologically/metabolically enable and support the expressed behavioral reaction pattern. Besides a huge overlap of the central nervous circuitries serving aggressive behavior and physiological stress-responsiveness, recent functional neuroanatomical evidence support the view that distinct but parallel and highly interacting neural circuitries underlie the expression of active/aggressive and passive/non-aggressive coping strategies. The functional activity of these neural networks, and thereby the expression of the respective aggression/coping style, is modulated by a wide variety of molecular substrates (i.e., neurotransmitters, hormones, cytokines and their respective metabolic enzymes, receptors and intraneuronal signaling molecules). However, most of these molecules appear to interact either directly or indirectly on diverse components of the serotonin (5-HT) system. Obviously this evolutionary ancient and well conserved neurotransmitter system remains the primary molecular orchestrator of aggression and coping style. The nature of this linkage, however, is not simple and it has proven difficult to unravel the precise role of this amine in the predisposition for and execution of aggressive behavior. The dogma that 5-HT inhibits aggression is obsolete now and the 5-HT deficiency hypothesis of aggression needs to be revised. Ongoing research on the functional status of 5-HT system before, during and after the execution of normal adaptive and abnormal pathological forms of aggression has led to the following view: Display of normal aggressive behavior aimed at social dominance and coherence is positively related to 5-HT neuronal (re)activity, whereas an inverse relationship develops between tonic 5-HT activity and pathological forms of aggression (e.g., impulsive violence). Profound functional changes in the premier autoregulatory sites that control firing and 5-HT release of the serotonergic neurons, i.e., presynaptic 5-HT_{1A} and 5-HT_{1B} autoreceptors and serotonergic reuptake transporters, are hypothesized to causally underlie this transition of normal adaptive aggressive behavior into abnormal excessive forms that cause harm and injury.

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