

Selected references – Barbara Sahakian

Roiser, J. P. & Sahakian, B. J. (2013). Hot and cold cognition in depression. *CNS Spectr.*, 18, 139-149.

Notes: We discuss the importance of cognitive abnormalities in unipolar depression, drawing the distinction between "hot" (emotion-laden) and "cold" (emotion-independent) cognition. "Cold" cognitive impairments are present reliably in unipolar depression, underscored by their presence in the diagnostic criteria for major depressive episodes. There is good evidence that some "cold" cognitive abnormalities do not disappear completely upon remission, and that they predict poor response to antidepressant drug treatment. However, in many studies the degree of impairment is moderately related to symptoms. We suggest that "cold" cognitive deficits in unipolar depression may in part be explicable in terms of alterations in "hot" processing, particularly on tasks that utilize feedback, on which depressed patients have been reported to exhibit a "catastrophic response to perceived failure." Other abnormalities in "hot" cognition are commonly observed on tasks utilizing emotionally valenced stimuli, with numerous studies reporting mood-congruent processing biases in depression across a range of cognitive domains. Additionally, an emerging literature indicates reliable reward and punishment processing abnormalities in depression, which are especially relevant for hard-to-treat symptoms such as anhedonia. Both emotional and reward biases are strongly influenced by manipulations of the neurochemical systems targeted by antidepressant drugs. Such a pattern of "hot" and "cold" cognitive abnormalities is consistent with our cognitive neuropsychological model of depression, which proposes central roles for cognitive abnormalities in the generation, maintenance, and treatment of depressive symptoms. Future work should examine in greater detail the role that "hot" and "cold" cognitive processes play in mediating symptomatic improvement following pharmacological, psychological, and novel brain circuit-level interventions
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Clark, L., Chamberlain, S. R., & Sahakian, B. J. (2009). Neurocognitive mechanisms in depression: implications for treatment. *Annual Review of Neuroscience*, 32, 57-74.

Notes: Mood disorders collectively account for a substantial proportion of disease burden across the globe and have a devastating impact on quality of life and occupational function. Here we evaluate recent progress in understanding the neurocognitive mechanisms involved in the manifestation of mood disorders. We focus on four domains of cognitive function that are altered in patients with depression: executive control, memory, affective processing, and feedback sensitivity. These alterations implicate a distributed neural circuit composed of multiple sectors of the prefrontal cortex in interaction with subcortical regions (striatum, thalamus) and temporal lobe structures (amygdala, hippocampus). Affective processing and feedback sensitivity are highly sensitive to serotonergic manipulation and are targeted by antidepressant treatments. By drawing together cognitive, neuroanatomical, and pharmacological tiers of research, we identify treatment targets and directions for future investigation to identify people at risk, minimize relapse, and maximize long-term beneficial outcomes for those suffering from depression

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Roiser, J. P., Cannon, D. M., Gandhi, S. K., Taylor, T. J., Erickson, K., Wood, S. et al. (2009). Hot and cold cognition in unmedicated depressed subjects with bipolar disorder. *Bipolar.Disord.*, *11*, 178-189.

Notes: OBJECTIVES: Neuropsychological studies in subjects with bipolar disorder (BD) have reported deficits on a variety of cognitive measures. However, because the majority of subjects were medicated at the time of testing in previous studies, it is currently unclear whether the pattern of deficits reported is related to BD itself or to psychotropic medication. We addressed this issue by examining cognitive performance in a group of unmedicated, currently depressed subjects with BD. METHODS: Forty-nine unmedicated subjects who met DSM-IV criteria for BD, depressed phase, and 55 control subjects participated in this study. Most patients were diagnosed with bipolar II disorder. Performance on emotion-dependent, or 'hot', and emotion-independent, or 'cold', cognitive tasks was assessed using tests from the Cambridge Neuropsychological Test Automated Battery. RESULTS: The groups were well matched with respect to general intelligence and demographic variables. Deficits in the unmedicated depressed BD group were apparent on tests tapping 'hot' cognitive processing, for example the Cambridge Gamble task and the Probabilistic Reversal Learning task. However, other than a deficit on the Spatial Span test in the depressed BD subjects, the groups performed equivalently on most measures of 'cold' cognitive processing, for example visual memory, attention, and working memory. CONCLUSIONS: These data suggest that deficits on tests involving reward processing, short-term spatial memory storage, and sensitivity to negative feedback in depressed BD subjects represent an effect of the illness itself and not mood-stabilizing medication

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