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The dysregulation of the "Behavioural Activation System": An independent dimension

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Abstract

Dysregulation of the Behavioural Activation System (BAS) is considered to a core vulnerability factor for bipolar affective disorders. To date, there is no self-report measurement that is aimed to assess this dysregulation independently from interindividual differences in the sensitivity of the BAS. Based upon the BIS/BAS-Scales (Carver & White, 1994) we developed a scale assessing the dysregulation of the BAS (DYS scale). One-hundred-seventy people completed the new inventory and – for validation – the PANAS and the HCL-32. The factor analysis suggested a three-factor solution. The newly generated items loaded on a common factor, thus confirming a dimension independent of BAS and BIS. Positive correlations between the BAS scale and positive affect and between the BIS scale and negative affect were found. The DYS scale was unrelated to current affect as postulated. Using the HCL-32 people with probable bipolar disorders showed significantly higher BIS but not BAS scores, and there was a trend for elevated scores on the dysregulation scale.

Keywords: Behavioural Activation System; Behavioural Inhibition System; Bipolar disorders; Vulnerability; Positive affect; HCL-32

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1. Introduction

Bipolar disorders seem to be much more prevalent than originally assumed with about 3–6.5% (e.g., Angst et al., 2003). Depue and Iacono (1989) presented a model proposing that the Behavioural Facilitation System – better known as Behavioural Approach System or Behavioural Activation System (BAS; e.g., Fowles, 1980) is associated with the vulnerability for bipolar disorders. Normally, two superordinate neurobehavioural systems – the BAS and the Behavioural Inhibition System (BIS) – are distinguished but according to Depue and colleagues only BAS is of relevance for bipolar disorders (Depue & Iacono, 1989; Depue & Zald, 1993). Referring to the 'old' reinforcement sensitivity theory (see Corr, 2004), BAS is thought to control appetitive motivation and reacts to signals of reinforcement or expectations of reward. Therefore, it is theoretically linked to experiencing positive affect such as hope, elation, and happiness. A high BAS sensitivity should express itself in a stronger goal-orientation, reaction to signals of reward and more positive affect (e.g., Depue & Iacono, 1989; Johnson et al., 2000).

Originally Depue and Iacono (1989) postulated that extreme changes in BAS activity are responsible for the symptoms of an affective episode. For example: high levels of BAS activation lead to higher incentive-reward motivation, greater sensitivity to rewarding stimuli, and also stronger seeking for pleasure and excitement. These are highly likely to result in positive affect. When positive affect increases it can become euphoria which is one core symptom of mania. Correspondingly, low levels of BAS activation (e.g., lack of incentive-reward motivation, anhedonia) would be a model for depression and characterized by low positive affect (e.g., Joiner, Brown, & Metalsky, 2003; Watson, Clark, & Carey, 1988a). Therefore (hypo-) manic and depressive episodes can be seen as opposite manifestations of a single dimension, i.e., BAS activity.

However, interindividual differences in BAS reactivity cannot totally explain interindividual differences in vulnerability to bipolar disorders. Such differences in BAS reactivity cannot explain how and why people fluctuate between normal and pathological states. If these individual differences were determining the risk for bipolar disorders, people with high BAS reactivity should be likely to be or become manic while those with low BAS reactivity would be at risk for depression only. Furthermore such a model cannot explain how individuals that seldom experience positive mood (i.e., have a low BAS reactivity) can develop mania. Therefore, an additional factor seems to be important and to represent a core vulnerability for bipolar disorders: the instability or dysregulation of the BAS (Depue & Zald, 1993; Johnson et al., 2000). It cannot be directly observed but has to be derived from behaviour and affect. In doing so, however, it has to be differentiated from the general reactivity of the BAS.

The dysregulation of the BAS was called "regulatory strength" by Depue and Zald (1993). It is thought to control BAS reactivity and to be a trait-like factor. If the regulatory strength is low, it is assumed that bipolar symptoms occur when the BAS activity reaches or trespasses a certain hypothesized threshold. It is assumed that in people with sufficient regulatory strength the BAS activity returns to the individual baseline within a certain time frame after it has been activated or de-activated. If individuals have, however, a low regulatory strength, an increased level of activation of the BAS is likely not to return to the baseline as usual (e.g., after goal attainment or receiving the reward) but remain either on a high level or even rise more, i.e., turn into mania. The opposite can happen after a de-activation of the BAS, therefore increasing the likelihood of depression. Therefore, low regulatory strength increases the risk of a dysregulation of the BAS.