

Attentional biases in anxiety and depression: current status and clinical considerations

Cognitive models of anxiety and depression postulate that these conditions are marked by negative attentional biases, i.e. increased or exaggerated attention to disorder-relevant negative information. These biases are not regarded as mere correlates of these disorders, but are thought to play a major role in their development and maintenance.

Temperamental factors such as neuroticism are thought to bias cognitive processes (e.g., attention, interpretation and memory) in such a way that negative information is prioritized, which can heighten the risk for anxiety disorders^{e.g.,1}. Likewise, depression is thought to be associated with difficulty to disengage attention from negative information and thoughts, which can play a key role in persistent negative thinking and sustained negative affect^{e.g.,2}. Based on these ideas, procedures have been developed to assess attentional biases and their role in psychopathology and, more recently, to correct these biases.

The most commonly adopted assessment procedures are cognitive-experimental tasks in which behavioral data (e.g., reaction times) are used to infer whether participants preferentially allocate attention to negative information as compared to neutral or positive one. In the dot probe task, for instance, individuals are presented with two spatially separated stimuli for a brief period of time (500 ms). One of these stimuli is negative (a negative word or picture), while the other is neutral. After offset of these stimuli, a small probe immediately appears on the location previously occupied by the negative or neutral stimulus. The speed of detection of the probe allows to infer where individuals allocated attention (e.g., faster response to probes replacing negative stimuli indicates a bias towards those stimuli).

Meta-analytic evidence supports the association between attentional biases and levels of anxiety and depression^{e.g.,3}. However, there are some inconsistencies in this empirical work^{e.g.,4}, in part due to the problematic psychometric properties of several frequently used behavioral tasks. This has led to the development of studies in which either eye-tracking data are collected (e.g., gaze fixation and duration) or psychophysiological markers of attention (e.g., event-related potentials) are examined. These measures allow to capture attention more reliably and can more easily evaluate attentional processes as they develop over time.

Despite this extensive research, there is still disagreement on the precise nature of attentional biases, as well as debate about whether the most frequently used measures adequately capture the dynamic nature of these biases (e.g., fluctuations between orienting towards and away from disorder-relevant information⁵). Progress has also been hampered by a predominant focus on visual attention to external stimuli, whereas many of the relevant stimuli for anxiety and depression may be internal (feelings and thoughts).

There is also a substantive literature on the mechanisms through which attentional biases could contribute to the development of anxiety and depression. For instance, in prospective studies, higher levels of attentional bias to negative information predicted increased stress reactivity, sustained negative mood, and higher levels of persistent negative thinking², which could in turn give rise to symptoms of anxiety and depression. As such, attentional bias could be a central driver of the Research Domain Criteria (RDoC) constructs of sustained threat and loss, which are of key relevance in anxiety and depression⁶.

Debates about the causal impact of attentional biases on psychopathology have also been fueled by studies using attentional bias modification (ABM) procedures, that is, procedures designed to correct attentional biases. The most frequently used procedure is a modified dot probe task where the task-relevant probe almost always follows the neutral information and rarely the negative one. In order to respond quickly to the probe, one thus has to learn to inhibit the tendency to orient to negative information. If this training generalizes to real life situations, it could in principle help reduce anxiety or depression. Despite initial encouraging findings, meta-analyses have shown that these procedures have only a limited and inconsistent impact on attentional biases and symptomatology⁷.

In response to these disappointing findings, novel procedures are being developed that try to correct attentional biases in methodologically as well as conceptually different ways. In these approaches, participants are made aware of their attentional bias, for instance, by using gaze-contingent feedback. More specifically, individuals are presented with displays in which both positive and negative information is presented, such as scrambled sentences (e.g., “life/my/a/party/is/mess”) that can be unscrambled in a positive (“my life is a party”) or negative way (“my life is a mess”). Eye-tracking methodology allows to detect when individuals allocate attention disproportionately to negative words in the scrambled sentences, which is then signaled back to them. Hence, they are trained to regulate their attention in more adaptive ways.

In laboratory studies, these procedures are effective in modifying attentional bias, which subsequently reduces rumination and increases positive reappraisal. There is also initial evidence for the efficacy of online and app-based versions of these procedures, which is important for dissemination purposes⁸. Yet, rigorous evaluation of clinical efficacy is required before clinical application is warranted.

Computer-based ABM tasks are only one way of targeting attentional biases for clinical purposes. There are in fact a host of clinical interventions that may be effective by targeting disorder-relevant attentional processes. For instance, mindfulness-based cognitive therapy for depression and metacognitive therapy for anxiety and depression contain exercises to correct attentional biases for negative information. Moreover, some theories on the impact of antidepressant medication and neurostimulation suggest that reductions in negative processing biases could be among the key mechanisms of change in these treatments⁹.

In summary, there is an increasing interest in clinical interventions targeting attentional biases in anxiety and depression, provided their role in the maintenance and exacerbation of these conditions. Yet, further progress can be made in terms of conceptual precision and ecological validity. The term “attentional bias” is still used to refer to markedly different phenomena, such as shifting, maintaining or redirecting attention towards and/or away from disorder-related stimuli. These conceptual problems restrict our ability to precisely measure and train attentional biases and hampers the study of the underlying (neural) mechanisms.

Moreover, there can be substantial discrepancies in laboratory versus real-world assessment of social attention. Thus, if researchers wish to capture clinically relevant aspects of attentional biases and determine their influence on psychopathology, the step to the real world, using portable eye-trackers and virtual reality, seems crucial.

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This paper was supported by a Ghent University grant (BOF16/MET_V/002). The two authors contributed equally to the work.

1. Eysenck MW. Anxiety: the cognitive perspective. Mahwah: Lawrence Erlbaum Associates, 1992.
2. De Raedt R, Koster EHW. *Cogn Affect Behav Neurosci* 2010;10:50-70.
3. Bar-Haim Y, Lamy D, Pergamin L et al. *Psychol Bull* 2007;133:1-24.
4. Van Bockstaele B, Verschuere B, Tibboel H et al. *Psychol Bull* 2014;140:682-721.
5. Zvielli A, Bernstein A, Koster EHW. *Clin Psychol Sci* 2015;3:772-88.
6. Gibb BE, McGeary JE, Beevers CG. *Am J Med Genet B Neuropsychiatr Genet* 2016;171:65-80.
7. Fodor LA, Georgescu R, Cuijpers P et al. *Lancet Psychiatry* 2020 ;7:506-14.
8. Sanchez-Lopez A, van Put J, De Raedt R et al. *Behav Res Ther* 2019;118:110-20.
9. Harmer CJ, Goodwin GM, Cowen PJ. *Br J Psychiatry* 2009;195:102-8.