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Cognitive effort deficits in depression:

Autonomic correlates and clues to potential rescue

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Commentary on Westbrook et al. (2022). Economic choice and heart rate fractal scaling indicate that cognitive effort is reduced in depression and boosted by sad mood. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging.*

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Depression is a major public health concern with enormous societal costs in terms of reduced social, occupational, and academic functioning; elevated risk of onset, persistence, and severity of physical health conditions; and increased early mortality (1). Disruptions in both affective and cognitive processing have been characterized as hallmarks of major depressive disorder, with cognitive impairments originally posited to reflect reduced cognitive capacity as a result of affective disruption. However, in recent years, a more nuanced and integrative characterization of cognitive and affective impairment in depression has emerged. Building on accounts arguing that cognitive control is effortful, with implementation depending not only on capacity but on the benefits and costs of control, individuals with depression have been proposed to show over-sensitivity to potential costs and under-sensitivity to potential benefits of cognitive effort (2). This implies that disrupted cognition in depression might not necessarily reflect a core deficit in cognitive capacity, but instead the ability to deploy cognition in a motivated and goal-adaptive fashion. However, alterations in the weighting of costs and benefits of cognitive effort in depression, potential biological etiology, and potential interventions for rescue, are not well-understood.

In the current issue of *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging,* Westbrook et al. (3) address this question in a provocative new study that combines economic choice behavior from the cognitive effort discounting (COGED) paradigm, autonomic measures of effort during task performance, and effects of mood manipulation on discounting performance in individuals with current depression, remitted depression, and never-depressed controls. The COGED requires participants to choose between an easy and a difficult version of an N-back working memory task

(e.g., 1-back vs. 2-, 3-, or 4-back) for a smaller versus larger monetary payoff, respectively, on each trial. Participant choices guided titration of the relative difference between payoffs until an indifference point (where participants were equally likely to choose the easy or difficult option), where the difference in payoffs was quantified as the subjective cost of the difference in cognitive effort between options. Consistent with theoretical frameworks suggesting altered cost-benefit tradeoffs for cognitive effort in depression, Westbrook and colleagues reported that depressed individuals discounted more steeply (i.e., required a higher relative payoff for the difficult task to reach indifference point) than individuals with remitted depression and controls, suggesting decreased willingness to exert cognitive effort relative to gained reward. Individual differences in discounting slope were associated with lower scores on a daily life functioning measure. A novel autonomic correlate of exerted effort, heart rate fractal scaling, was blunted during subsequent N-back task performance, uniquely in depressed steep discounters: this relationship was not observed in depressed shallow discounters or in remitted/control participants across discounting slopes. Finally, a between-subjects mood induction (neutral vs. sad mood) prior to the COGED revealed that sad mood induction decreased effort discounting (i.e., increased willingness to exert effort relative to reward) specifically in depressed individuals to levels observed in the control group. These findings provide empirical evidence for the assertion that depression might be associated with altered cost-benefit weighting of cognitive effort, rather than decreased ability to deploy cognitive effort *per se*. This characterization might help reconcile conflicting prior findings in the literature, where depression has been associated with reduced cognitive effort in some studies but poorer calibration of

cognitive effort to demand in others (i.e., overuse of effort on easy tasks and withdrawal of effort on more difficult tasks; (4)).

Westbrook et al. introduce fractal scaling of heart rate variability data as a novel measure of subjective cognitive effort, reporting a positive relationship with increasing working memory load during N-back performance in all participant groups *except* depressed steep discounters. Fractal scaling is argued to quantify variation in the complexity of physiological dynamics across timescales; this analytical approach has been previously used with heart rate variability data to index physical effort (5) as well as with neural activity, measured using EEG, to index cognitive effort (6). Importantly, this prior work suggests that fractal scaling might increase monotonically with increasing effort, even past the point of capacity limit. This might be a key advantage of this novel metric, given that other cardiovascular measures of effort such as pre-ejection period (PEP) have been observed to increase from low to high task demand and decrease again when task demands exceed capacity, potentially reflecting *objective* engagement rather than *subjective* cognitive effort (which arguably could continue increasing with increasing task demand, even as demand exceeds capacity limits). This approach offers exciting possibilities, given that metrics of subjective effort have been challenging to reliably characterize. Further, the observation that blunted autonomic response was uniquely observed in depressed, steep discounters might offer hints to underlying mechanisms of disrupted effort deployment in depression. This disruption in autonomic response was only observed in individuals with current depression and not remitted, in contrast to some aspects of cognitive control that appear disrupted in both (7): this suggests that autonomic blunting is not necessarily part of an underlying phenotype

associated with heightened risk of depression more broadly. Also, blunted autonomic reactivity was not observed in steep discounters across participant groups: steep discounters in the remitted depression and control groups showed increases in heart rate fractal scaling with increasing task load similarly to shallow discounters, suggesting that autonomic blunting is not a necessary and causal driver of decreased cognitive effort independent of depression. Westbrook et al. suggest that blunted autonomic response might act as a constraint on cognitive effort deployment specifically in depression, but characterizing directional relationships between these processes, clarifying why such constraints might be uniquely observed in currently depressed individuals, and obtaining further support for the validity of fractal scaling as a marker of subjective cognitive effort more broadly, remain important issues for future research.

One of the most intriguing findings that Westbrook et al. report is the observation that sad (vs. neutral) mood induction is subsequently associated with shallower effort discounting in currently depressed individuals (but not other participant groups). This finding should be considered with caution, given that the mood induction was between-subjects, and runs contrary to a priori predictions that increased sad mood should be associated with further discounting of cognitive effort in depression, where elevated negative affect and increased attention to negative emotional stimuli are both typically observed. In discussing this surprising observation, Westbrook et al. note that sadness, especially when elicited by external and controllable stimuli (such as sad movies or music), has been suggested as a form of identity validation for depressed individuals and associated with desired affective experience (8), offering potential benefits to cognition. We further speculate that such validating affective experiences might be

particularly important given that depression has been associated with flattened affect, apathy, and restricted emotional range *as well as* elevated negative affect, and that apathy has been identified as a key transdiagnostic correlate of functional impairment in psychopathology more broadly (9). Mixed-methods research examining online disclosure and discussion of mental illness suggests the desire for affective experience as a potential antidote to apathy ("I'm so bored but I don't want to do anything. I'm so tired but all I do is sleep. I want to feel something…", (10)). Taken together, these findings suggest that the relationship between sad mood and depression may be more complex than originally characterized; identity-validating affective experiences (particularly when external and controllable) may help ameliorate apathy symptoms in depressed individuals and promote cognitive engagement. At present, this possibility remains highly speculative; this approach may not offer benefits to all depressed individuals, given that depression is linked with heterogeneous symptoms and potential risk factors across individuals. However, this evidence suggests an important new direction for future research on interventions to address cognitive and motivational impairment in depression that are both mechanistically grounded and aligning with accounts of lived experience.

In conclusion, Westbrook et al. provide exciting new evidence suggesting that alterations in cost-benefit tradeoff for cognitive effort can be observed in depression and might be accompanied by blunted autonomic reactivity as measured by heart rate fractal scaling, a novel measure of subjective cognitive effort. They further report that sad mood induction might enhance motivated effort in depression, contrary to their a priori predictions but potentially consistent with broader accounts suggesting that apathy

can drive functional impairment in psychopathology and may potentially be counteracted by validating affective experiences. While future work will need to empirically test for these possibilities more directly, disrupted effort deployment and its rescue by affect appear promising as potential candidate mechanisms for cognitive impairment and intervention in depression.

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