Title: Focusing inward: A timely yet daunting challenge for clinical psychological science

Ernst H.W. Koster[1], Igor Marchetti[2], & Ivan Grahek[3]

1) Ghent University, Belgium
2) University of Trieste, Italy
3) Brown University, USA

Corresponding author:
Ernst Koster
Department of Experimental-Clinical and Health Psychology, Ghent University, Belgium.
E-mail: Ernst.Koster@Ugent.be

Ivan Grahek:
Department of Cognitive, Linguistic, & Psychological Sciences and Carney Institute for Brain Science, Brown University, Providence, RI 02912, USA

Igor Marchetti:
Department of Life Sciences, Psychology Unit, University of Trieste, Trieste, 34128, Italy

Amir and Bernstein propose a dynamical model of internally-directed cognition aimed at explaining the complex interactions between current goals, negative affect, and attentional selection in working memory. They connect the literature on internal attention, working memory, affect, rumination, and mindwandering to propose a formal mathematical model of internally-directed cognition. In this paper, they do not just provide a window on how people become stuck in loops of negative thinking, but they also provide a nice example of how clinical psychological science can move towards more formal theoretical models.

In taking such an exciting step, we believe that this work also encounters some of the challenges faced by the formal models of maladaptive cognition. Below we discuss some of these issues, not in order to criticize the current work, but to open a discussion, which we feel is paramount as the field of clinical psychology moves in the direction of developing formal theoretical models. In brief, the three main issues are: 1) the proposed model does not build on the existing cognitive models; 2) the model increases rather than decreases the complexity of the phenomenon; 3) there are no standard/alternative frameworks to compare the A2T model to, and it is not clear which kind of data or experiments could corroborate or falsify the model.

New models should build on the existing formal models of cognitive processes

The reproducibility crisis in psychology (Simmons et al., 2011; Open Science collaboration, 2015) has led to significant changes in the way we conduct research, which include preregistration and better statistical methodology (Benjamin et al., 2018; Nosek et al., 2018). In the slipstream of this movement, a reinvigorated discussion has been opened on the role and current status of theory in psychology (e.g., Fried, 2021; Grahek et al., 2021; Haslbeck et al., 2021). Clearly, clinical psychological science has no shortage of rather vague, descriptive theories that are difficult to test and disprove. Many areas of psychology are moving in the direction of developing stronger theories, which could guide experimentation and increase the overall rigor of psychological science. In this context, clinical psychology is faced with the task of creating formal mathematical models of important phenomena, including the ones which the A2T model tackles. This effort, often referred to as computational psychiatry (Montague et al., 2012; Huys et al., 2016), is showing a lot of promise. The crucial part of this effort is to develop computational models that are relevant for understanding psychopathology, but also have direct links with the existing formal models from cognitive science. In this way, clinical psychology can build on the existing models, and extend them in order to better understand psychopathology. Such efforts are already present in many domains including decision-making (Huys et al., 2015), learning (Brown et al., 2021), working memory (Collins et al., 2017), and cognitive control (Dillon et al., 2015; Grahek et al, 2019).

This is where the A2T model will require the most work in its further development. While the authors mention some of the models that map onto the components of their model (Hazy et al., 2007), all of these components are modeled at a very high level right now. While this is a necessary and a great first step, the model would benefit from incorporating the architecture of existing models of attentional control, working memory, and affect. Unless this is done, we are missing the opportunity to do cumulative science and develop an integrated understanding of clinical processes occurring within a normative framework. In past years, this has been raised in the area of cognitive control in depression, where theory in clinical psychological science developed without attention to the basic cognitive-experimental science on cognitive control (Grahek, Everaert, Krebs & Koster, 2018).

Degrees of freedom in model construction

There are several risks in modeling specific clinical phenomena on their own. The most important one is that unless more complex models are built on validated basic cognitive science models, there are too many degrees of freedom in model construction, which makes them too broad to be falsified. A dynamical model with several free parameters can produce many interesting behaviors. While this is a great strength of applying mathematical models to understand living systems, it can also be their greatest weakness. Unless a model is constrained by previous modeling work (e.g., reinforcement learning or working memory models), it suffers from the problem of an arbitrary number of parameters which can be added to the model in order to ensure that it produces desired behavior. The increasing number of parameters can then lead to many different paths which generate the same behavior, which in turn increases the complexity of the explanation of the phenomenon, rather than the desired simplification of the explanation. For example, in the A2T model ruminative responses (i.e. repeated selection of negative material) can occur due to: 1) the overall higher number of negative memories in long-term memory which increases the probability of the selection of negative material; 2) higher reactivity to negative affect; 3) lower cognitive control (leading to less task-relevant information processing/the context parameter); and 4) having an explicit goal to ruminate. While such problems are present across cognitive science (for example in both motor and cognitive control research, cf. Ritz et al., 2022), development of new models should aim to overcome them. Ideally, models should be able to reduce the number of possible paths that generate a certain phenomenon, rather than increase them. Unless this is done, an endless number of different models can be created and they will all be able to account for the existing data. The best way to avoid this issue is to further develop cognitive models aimed at explaining certain phenomena in basic cognitive science, to see whether they can also successfully deal with clinical phenomena.

In that case, different competing models from cognitive science can be compared against each other in their ability to explain clinical phenomena and individual differences. This will be of great advantage for both clinical and cognitive science.

Model comparison

Formal models of clinical phenomena are most useful when they can be compared against each other. This allows for rapid progress in empirical research because often new types of data are required in order to distinguish between different models. However, comparison between models is only possible when they are building on a common set of modeling assumptions. If new models are built only to account for clinical phenomena, without direct connections to the existing models from cognitive science, comparison among them will be very difficult. This is why it is crucial for clinical science to build on the existing cognitive models, which will allow for model comparison. While demonstrating that a model can capture the data well is a crucial step, comparison among competing models is necessary for a good mechanistic understanding of which key processes are paramount in describing a phenomenon.

Being very stringent about the model construction and comparison is particularly important when clinical psychological science starts to examine phenomena at the level of internal mental processes, where we have a limited window on the behavioral components associated with such processes. Although we fully agree with Amir and Bernstein that we need to increase our focus on internal mental processes, experimental psychopathology has favored examining external attention processes since these are somewhat easier to capture. In order to develop formal theory on internal mental processes, it will be crucial to specify the behavioral and/or neural outputs that can be expected in order to allow for empirical validation.

In conclusion, we believe that the authors are taking the right step in developing a formal model of internally directed cognition. This is a very hard task, and specifying the critical components of the model, and including dynamics between the different processes is a great step forward. However, modeling such complex psychological phenomena comes with many challenges, and further development of the model should seek to address some of them. We believe that following the path of computational cognitive science in addressing these challenges could be of great benefit to clinical science. Using and further developing formal mathematical models of cognitive processes in order to advance the understanding of psychopathology holds great promise and will certainly push current boundaries in the study of psychopathology.

References

Benjamin, D. J., Berger, J. O., Johannesson, M., Nosek, B. A., Wagenmakers, E. J., Berk, R., ... & Johnson, V. E. (2018). Redefine statistical significance. *Nature human behaviour*, *2*(1), 6-10.

Brown, V. M., Zhu, L., Solway, A., Wang, J. M., McCurry, K. L., King-Casas, B., & Chiu, P. H. (2021). Reinforcement learning disruptions in individuals with depression and sensitivity to symptom change following cognitive behavioral therapy. *JAMA psychiatry*, *78*(10), 1113-1122.

Collins, A. G., Albrecht, M. A., Waltz, J. A., Gold, J. M., & Frank, M. J. (2017). Interactions among working memory, reinforcement learning, and effort in value-based choice: A new paradigm and selective deficits in schizophrenia. *Biological psychiatry*, *82*(6), 431-439.

Dillon, D. G., Wiecki, T., Pechtel, P., Webb, C., Goer, F., Murray, L., ... & Pizzagalli, D. A. (2015). A computational analysis of flanker interference in depression. *Psychological medicine*, *45*(11), 2333-2344.

Huys, Q. J., Daw, N. D., & Dayan, P. (2015). Depression: a decision-theoretic analysis. *Annu Rev Neurosci*, *38*(1), 1-23.

Huys, Q. J., Maia, T. V., & Frank, M. J. (2016). Computational psychiatry as a bridge from neuroscience to clinical applications. *Nature neuroscience*, *19*(3), 404-413.

Fried, E.I. (2020) Lack of Theory Building and Testing Impedes Progress in The Factor and Network Literature, Psychological Inquiry, 31, 271-288, DOI:10.1080/1047840X.2020.1853461

Gershman, S. J., & Lai, L. (2021). The Reward-Complexity Trade-off in Schizophrenia. *Computational Psychiatry*, *5*(1), 38–53.

Grahek, I., Everaert, J., Krebs, R.M., & Koster, E. H.W. (2018). Understanding depression-related cognitive control dysfunctions: How cognitive neuroscience can inform clinical models. Clinical Psychological Science, 6, 264-280.

Grahek, I., Shenhav, A., Musslick, S., Krebs, R.M., & Koster, E.H.W. (2019). Motivation and Cognitive Control in Depression. *Neuroscience and Biobehavioral Reviews, 102, 371-381*.

Grahek, I., Schaller, M., & Tackett, J. L. (2021). Anatomy of a psychological theory: Integrating construct-validation and computational-modeling methods to advance theorizing. *Perspectives on Psychological Science*, *16*(4), 803-815.

Haslbeck, J. M. B., Ryan, O., Robinaugh, D. J., Waldorp, L. J., & Borsboom, D. (2021). Modeling psychopathology: From data models to formal theories. Psychological Methods. Advance online publication. https://doi.org/10.1037/met0000303

Hazy, T. E., Frank, M. J., & O'reilly, R. C. (2007). Towards an executive without a homunculus: computational models of the prefrontal cortex/basal ganglia system. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *362*(1485), 1601-1613.

Montague, P. R., Dolan, R. J., Friston, K. J., & Dayan, P. (2012). Computational psychiatry. *Trends in cognitive sciences*, *16*(1), 72-80.

Nosek, B. A., Ebersole, C. R., DeHaven, A. C., & Mellor, D. T. (2018). The preregistration revolution. *Proceedings of the National Academy of Sciences*, *115*(11), 2600-2606.

Open Science Collaboration. (2015). Estimating the reproducibility of psychological science. *Science*, *349*(6251).

O'Reilly, R. C., & Frank, M. J. (2006). Making working memory work: a computational model of learning in the prefrontal cortex and basal ganglia. *Neural computation*, *18*(2), 283-328.

Ritz, H., Leng, X., & Shenhav, A. (2022). Cognitive control as a multivariate optimization problem. *Journal of Cognitive Neuroscience*, *34*(4), 569-591.

Simmons, J. P., Nelson, L. D., & Simonsohn, U. (2011). False-positive psychology: Undisclosed flexibility in data collection and analysis allows presenting anything as significant. *Psychological science*, *22*(11), 1359-1366.