

Correspondence between reinforcement learning phenotypes and transdiagnostic clinical symptomatology across development

Noam Goldway (noamgoldway@nyu.edu)

New York University Psychology Department
6 Washington Pl, New York, NY 10003, USA

Nora Harhen (nora.harhen@nyu.edu)

New York University Psychology Department
6 Washington Pl, New York, NY 10003, USA

Qingqing Yang (yang.6118@osu.edu)

The Ohio State University, Department of Psychology
Columbus, OH 43210, USA

Sophia Nielsen (sln8985@nyu.edu)

New York University Psychology Department
6 Washington Pl, New York, NY 10003, USA

Levi Solomyak (lsolomyak@gmail.com)

The Hebrew University of Jerusalem Department of Cognitive and Brain Sciences
Jerusalem 9190401, Israel

Sam Zorowitz (szorowi1@gmail.com)

Princeton University Princeton Neuroscience Institute & Psychology Department
40 Woodlands Way Princeton, NJ 08540, USA

Gal Shoval (shovgal@tauex.tau.ac.il)

Tel Aviv University Sackler Faculty of Medicine
Ramat Aviv, 69978 Tel Aviv, Israel

Eran Eldar (eran.eldar@mail.huji.ac.il)

The Hebrew University of Jerusalem Department of Cognitive and Brain Sciences,
Jerusalem 9190401, Israel

Yael Niv (yael@princeton.edu)

Princeton University Princeton Neuroscience Institute & Psychology Department
40 Woodlands Way Princeton, NJ 08540, USA

Catherine Hartley (cate@nyu.edu)

New York University Psychology Department
Center for Neural Science
6 Washington Pl, New York, NY 10003, USA

Abstract

Adolescence is marked by neurocognitive changes and heightened vulnerability to psychiatric disorders. Yet, it remains unclear how age-related changes in learning processes known to contribute to adult psychopathology may contribute to the emergence of psychiatric symptoms. In a sample of 889 individuals aged 10–25, we related reinforcement learning computational phenotypes to psychopathology measured along multiple dimensions of dysfunction. Participants completed three learning tasks targeting sensitivity to positive versus negative outcomes, Pavlovian bias (innate versus instrumental behavior), and model-based control (goal-directed planning). Factor analysis of self-report questionnaires identified four symptom dimensions: rumination, physiological anxiety, impulsivity/dysregulation, and anhedonia. Symptom expression varied with age, and each symptom domain demonstrated a distinct computational signature.

Keywords: Reinforcement Learning; Computational Psychiatry; Development

Introduction

During adolescence, as individuals gain independence and encounter new social and intellectual challenges (Sanders, 2013; Verhoeven et al., 2019), brain systems involved in reinforcement learning also undergo major developmental shifts (Hauser et al., 2019). This developmental window coincides with increased risk for anxiety, mood disorders, psychosis, and other conditions (Kessler et al., 2005; Patton et al., 2016). Understanding how neurocognitive development confers vulnerability to psychopathology is critical (Paus et al., 2008). Here, we examined whether specific reinforcement-learning computations implicated in adult psychiatric symptomatology may modulate underlying psychiatric risk across development (Goldway et al., 2023).

Methods

A total of $N = 889$ participants from the general population, aged 10 to 25 years were divided into a discovery sample ($N = 456$) and a replication sample ($N = 433$). Here, we present results from the discovery sample only, as recruitment for the study is still ongoing.

Participants completed a battery of three reinforcement-learning tasks and self-report questionnaires assessing transdiagnostic psychiatric symptoms, including OCD (OCI), anxiety (SCARED), depression (BDI), ADHD (ASRS), impulsiveness (BIS), emotion dysregulation (TIDES), intolerance of uncertainty (IUS), rumination (RRS), and anhedonia (SHAPS), over a three-week period.

Reinforcement-learning tasks: The valence asymmetry task (Niv et al., 2012) measured sensitivity to signed prediction errors (the extent of learning from positive vs. negative prediction errors). On each trial, participants chose between a certain cue (+5 points) and a risky cue that yielded +10 or 0 points with varying probabilities (20%, 50%, or 80% for different cues). Feedback was provided on both chosen and unchosen options, allowing learning through both experience and counterfactual feedback (Figure 1A). The Pavlovian Bias Task (Zorowitz et al., 2023) measured how automatic tendencies to approach rewards or withdraw from punishments interfere with instrumental learning. Participants saw "robot" stimuli and had to learn, via feedback, whether to respond ("Go") or withhold a response ("No-Go"). Trial types varied by reward and punishment outcome domain and action requirement, producing Pavlovian-congruent and incongruent conditions. Feedback was probabilistic (Figure 1B). Finally, in the Model-Based/Model-Free Task (Decker et al., 2016), participants chose between spaceships leading probabilistically to different planets, followed by a second-stage choice between aliens yielding rewards based on drifting probabilities. The task structure allowed distinguishing whether participants relied on an action's reward history alone (model-free) or also on the task's transition structure (model-based) when making choices (Figure 1C). Behavior was analyzed using variants of previously published

computational models (Rosenbaum et al., 2022; Decker et al., 2016; Zorowitz et al., 2023)

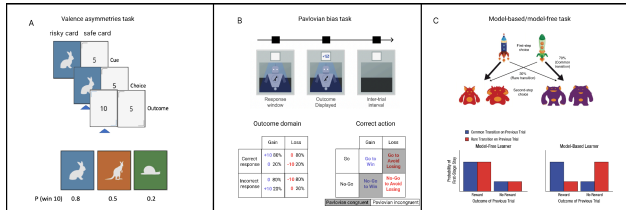


Figure 1: Three reinforcement learning tasks

Results

Developmental trajectory of psychiatric symptomatology. We used factor analysis to identify underlying dimensions of psychopathology by clustering self-report items based on shared variance (Figure 2A). Ruminative intrusion and physiological anxiety both increased with age, with significant linear and quadratic effects (age: $p < .001$; age²: $p < .001$). In contrast, impulsivity and dysregulation decreased significantly as individuals grew older (age: $p < .001$). Anhedonia showed a non-linear trajectory, peaking during adolescence (age²: $p < .05$) (Figure 2B).

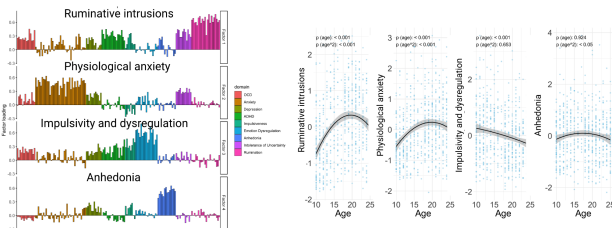


Figure 2: Developmental trajectory of psychiatric symptomatology in our sample aged 10-25

Developmental trajectory of learning computations. Parameter estimates derived from task behavior revealed age-related shifts in learning processes, replicating past findings (Nussenbaum et al., 2020; Rosenbaum et al., 2022). As individuals aged, they became less optimistic, as reflected by lower initial Q-values ($p < .001$) and increased sensitivity to negative outcomes, particularly for the unchosen option (chosen: $p = .87$; unchosen: $p < .001$) in the valence asymmetry task. Pavlovian biases remained stable across age ($p = .325$), while model-based learning showed a significant increase with age ($p < .05$).

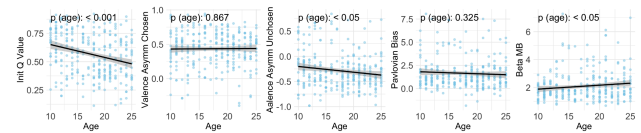


Figure 3: Developmental trajectory of psychiatric symptomatology.

Correspondence between reinforcement-learning phenotypes and transdiagnostic clinical symptomatology. Regression models incorporating task-derived parameters and age revealed developmental patterns linking learning computation to psychiatric symptoms. Ruminative intrusions were elevated in individuals with negative asymmetry in learning from unchosen outcomes ($p < .05$), with this effect being stronger in younger participants ($p < .05$). This symptom domain was also more pronounced in individuals with lower initial Q-values ($p < .01$) and reduced Pavlovian bias ($p < .001$). Physiological anxiety was associated with a stronger Pavlovian bias ($p < .01$), particularly during adolescence, and with negative asymmetry in learning from unchosen outcomes ($p < .001$). Impulsivity and dysregulation were elevated in adolescents with negative unchosen asymmetry ($p < .01$) and higher levels of Pavlovian bias ($p < .05$). Anhedonia was linked to higher Pavlovian bias ($p < .01$), especially when combined with negative asymmetry for unchosen outcomes ($p < .01$) and younger age ($p < .001$).

Conclusion

Consistent with past findings, both learning strategies and symptom dimensions changed with age; however, their associations were not stable across development and tended to peak during adolescence. Moreover, interactions between learning processes, such as Pavlovian biases and valence asymmetry, uniquely predicted symptom expression. These computational phenotypes offer insight into the mechanisms through which learning alterations may shape vulnerability to psychopathology during adolescence.

Funding acknowledgements

This work was supported by an Israel-USA CRCNS grant from the National Institute of Mental Health (Grant No. R01MH125564) and the Binational Science Foundation (BSF grant no. 2019801)

References

- Goldway, N., Eldar, E., Shoval, G., & Hartley, C. A. (2023). Computational Mechanisms of Addiction and Anxiety: A Developmental Perspective. *Biological Psychiatry*, *93*(8), 739–750.
- Hauser, T. U., Will, G.-J., Dubois, M., & Dolan, R. J. (2019). Annual Research Review: Developmental computational psychiatry. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *60*(4), 412–426.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593–602.
- Niv, Y., Edlund, J. A., Dayan, P., & O'Doherty, J. P. (2012). Neural prediction errors reveal a risk-sensitive reinforcement-learning process in the human brain. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, *32*(2), 551–562.
- Nussenbaum, K., Scheuplein, M., Phaneuf, C. V., Evans, M. D., & Hartley, C. A. (2020). Moving developmental research online: Comparing in-lab and web-based studies of model-based reinforcement learning. *Collabra. Psychology*, *6*(1). <https://doi.org/10.1525/collabra.17213>
- Patton, G. C., Sawyer, S. M., Santelli, J. S., Ross, D. A., Afifi, R., Allen, N. B., Arora, M., Azzopardi, P., Baldwin, W., Bonell, C., Kakuma, R., Kennedy, E., Mahon, J., McGovern, T., Mokdad, A. H., Patel, V., Petroni, S., Reavley, N., Taiwo, K., ... Viner, R. M. (2016). Our future: a Lancet commission on adolescent health and wellbeing. *Lancet*, *387*(10036), 2423–2478.
- Paus, T., Keshavan, M., & Giedd, J. N. (2008). Why do many psychiatric disorders emerge during adolescence? *Nature Reviews. Neuroscience*, *9*(12), 947–957.
- Rosenbaum, G. M., Grassie, H. L., & Hartley, C. A. (2022). Valence biases in reinforcement learning shift across adolescence and modulate subsequent memory. *eLife*, *11*, e64620.
- Sanders, R. A. (2013). Adolescent psychosocial, social, and cognitive development. *Pediatrics in Review*, *34*(8), 354–358; quiz 358–359.
- Schiele, M. A., Reinhard, J., Reif, A., Domschke, K., Romanos, M., Deckert, J., & Pauli, P. (2016). Developmental aspects of fear: Comparing the acquisition and generalization of conditioned fear in children and adults. *Developmental Psychobiology*, *58*(4), 471–481.
- Verhoeven, M., Poorthuis, A. M. G., & Volman, M. (2019). The role of school in adolescents' identity development. A literature review.

Educational Psychology Review, 31(1), 35–63.

Zorowitz, S., Karni, G., Paredes, N., Daw, N., & Niv,

Y. (2023). Improving the reliability of the

Pavlovian go/no-go task for computational

psychiatry research. In *PsyArXiv*.

<https://doi.org/10.31234/osf.io/eb697>