

Archival Report

Cognitive Signatures of Depressive and Anhedonic Symptoms and Affective States Using Computational Modeling and Neurocognitive Testing

Nadja R. Ging-Jehli, Manuel Kuhn, Jacob M. Blank, Pranavan Chanthrakumar, David C. Steinberger, Zeyang Yu, Todd M. Herrington, Daniel G. Dillon, Diego A. Pizzagalli, and Michael J. Frank

ABSTRACT

BACKGROUND: Deeper phenotyping may improve our understanding of depression. Because depression is heterogeneous, extracting cognitive signatures associated with severity of depressive symptoms, anhedonia, and affective states is a promising approach.

METHODS: Sequential sampling models decomposed behavior from an adaptive approach-avoidance conflict task into computational parameters quantifying latent cognitive signatures. Fifty unselected participants completed clinical scales and the approach-avoidance conflict task by either approaching or avoiding trials offering monetary rewards and electric shocks.

RESULTS: Decision dynamics were best captured by a sequential sampling model with linear collapsing boundaries varying by net offer values, and with drift rates varying by trial-specific reward and aversion, reflecting net evidence accumulation toward approach or avoidance. Unlike conventional behavioral measures, these computational parameters revealed distinct associations with self-reported symptoms. Specifically, passive avoidance tendencies, indexed by starting point biases, were associated with greater severity of depressive symptoms ($R = 0.34$, $p = .019$) and anhedonia ($R = 0.49$, $p = .001$). Depressive symptoms were also associated with slower encoding and response execution, indexed by nondecision time ($R = 0.37$, $p = .011$). Higher reward sensitivity for offers with negative net values, indexed by drift rates, was linked to more sadness ($R = 0.29$, $p = .042$) and lower positive affect ($R = -0.33$, $p = .022$). Conversely, higher aversion sensitivity was associated with more tension ($R = 0.33$, $p = .025$). Finally, less cautious response patterns, indexed by boundary separation, were linked to more negative affect ($R = -0.40$, $p = .005$).

CONCLUSIONS: We demonstrated the utility of multidimensional computational phenotyping, which could be applied to clinical samples to improve characterization and treatment selection.

<https://doi.org/10.1016/j.bpsc.2024.02.005>

In the United States, the number of adults experiencing depression-related symptoms has quadrupled over the past 4 years (1,2). Probing distinctions between cognitive signatures of depressive and anhedonic symptoms is crucial because this differentiation not only enhances our comprehension of anhedonia but also contributes to a deeper understanding of depression. This is particularly significant because increased anhedonia severity has been linked to worse trajectories in depression (3–5), increased nonresponsiveness to treatments (6–8), and poorer quality of life (9).

Depression and anhedonia have both been associated with multiple affective states (10,11). Characterizing depression, some studies have reported diminished positive affect and excessive negative affect (12,13), while others have found pronounced sadness but intact positive affect (11,14–16).

Characterizing anhedonia, some studies have reported flat and blunted responses to pleasurable experiences (17,18), while others have found shorter and/or more variable positive affective responses to pleasurable experiences (19,20). Consequently, this wide range of affective states often manifests in diverse symptom profiles (i.e., phenotypes). Therefore, studies are needed to deconstruct these heterogeneous symptom profiles. For example, distinguishing anhedonia from other symptoms of depression may improve diagnostics and treatment selection, but to date, similarities and differences among cognitive signatures of dimensional depression, anhedonia, and affective states have rarely been explored.

Here, we characterize latent cognitive signatures of depressive symptoms, anhedonia, and affective states by decomposing behavior from (neuro)cognitive tests with

process-oriented models. This approach is known as multidimensional computational phenotyping (21–31). We focus on symptom severities rather than diagnostic categories, consistent with the conceptualization that mental health conditions generally exist on a continuum rather than as categories (24,31,32). First, we introduce the approach-avoidance conflict (AAC) task as a promising probe for characterizing depressive phenotypes (33–36), highlighting several novel features. Next, we introduce sequential sampling models as powerful process-oriented analytics.

Studying AAC Behavior

In AAC paradigms, participants decide to approach or avoid offers that include both rewarding and aversive features (37). Varying the relative magnitude of these rewarding and aversive features produces offers with different conflict levels. Recent studies found that individuals with major depressive disorder were characterized by distinct neural and behavioral patterns compared to individuals without major depressive disorder (28,38). Specifically, Pedersen *et al.* (28) found that people with major depressive disorder were less sensitive to reward and had lower tendencies to approach offers. However, past studies focused on categorical assessments and did not discriminate between cognitive signatures of dimensional depression, anhedonia, and affective states.

Using AAC paradigms to extract fine-grained signatures of different depression-related constructs requires modification of task specifics to increase their clinical sensitivity. This is because reward and aversion responses in AAC paradigms can be driven by multiple underlying constructs that need to be dissociated. For example, participants' experienced conflict level depends on their so-called marginal rate of substitution between reward and aversion—that is, the willingness to accept an additional unit of aversion for an additional unit of reward. Conversely, participants' sensitivity to changes in either reward or aversion depends on their marginal utilities.¹ Ultimately, reward and aversion responses can also be influenced by asymmetric costs of approach relative to avoidance choices (28,39). All these concepts can affect decision-making processes differently and may involve distinct neural pathways and signaling (40,41).

We implemented a modified AAC task (Figure 1A) to distinguish between the aforementioned concepts. First, offers were composed of reward and aversion by using money and shock as reinforcers. This allowed us to calibrate offers based on individuals' marginal rates of substitution and parametrically manipulate the amount of offered reward and punishment. Second, offers were created on a trial-by-trial basis to separately probe individuals' reward and aversion sensitivities. Third, we distinguished between positive and negative domains because reward and aversion sensitivity may depend on the sign of an offer's net value (42–45). Fourth, we distinguished between instrumental responses that are congruent with Pavlovian approach/avoidance tendencies, making them

more automatic than those that are incongruent with these tendencies (Figure 1B) (39,46,47).

Sequential Sampling Modeling

We focused on a process-oriented account by fitting sequential sampling models (SSMs) to behavioral data from the AAC task (24,48). Conventional performance measures and alternative cognitive models (e.g., signal detection theory models) focus either on response times (RTs) or response frequencies (24). Conversely, SSMs simultaneously account for the entire RT distribution and the relative frequency of each response option, thus providing richer analytical information (49,50). SSMs simulate behavior with processes that sequentially accumulate information up to a decision threshold (49,51–53). This allows the decomposition of behavior into distinct, quantifiable mental components with established psychological interpretations.

The diffusion decision model (54) is a prominent SSM (Figure 2A) with 4 main parameters (28,50). Specifically, drift rate (v) reflects the quality of evidence accumulation. In our context, higher drift rates indicate easier decisions, such that evidence accumulates more rapidly, resulting in faster RTs and more frequent approach choices. Boundary separation (a) reflects the required amount of evidence for reaching decisions. Larger boundary separations yield more consistent choices (i.e., less variability in choosing different actions for offers with similar levels of evidence), resulting in slower (and more skewed) RTs. Starting points (z) indicate initial response biases (e.g., due to asymmetric costs of stimulus-response mapping). In our context, larger starting points indicate greater biases toward approach choices, which leads to large changes in the tail and leading edge of the RT distributions. Finally, longer nondecision time (T_{er}) indicates longer perceptual encoding and response execution times that occur outside the decision process, shifting the entire RT distribution but without affecting its shape.

Only a few studies have examined AAC behavior with computational models, and most of them did not use SSMs (55–59). The few studies that applied SSMs only used the classic diffusion decision model and focused on categorical assessments of depression (28,34,60,61). However, different SSMs assume distinct dynamics in decision-making processes that can lead to different behavioral predictions (49,52,62,63). For example, collapsing boundaries (Figure 2B) are used to model the declining need for additional evidence as time passes (e.g., when participants become increasingly impatient or when externally or internally imposed response deadlines are imposed) (64–67). Therefore, we tested different models to find the one that accounted best for the behavioral pattern (24,68).

METHODS AND MATERIALS

Participants

Fifty adults were recruited through the Harvard Psychology Community Study Pool. Inclusion was restricted to adults between ages 18 and 45 years who were fluent in English and not color blind; note that this study pool does not consist solely of Harvard students. Participants were not preselected

¹The change in subjective value for a marginal increase in reward, keeping constant aversiveness. Marginal utilities reflect the relative change of consumed reward and aversion.

Multidimensional Computational Phenotyping

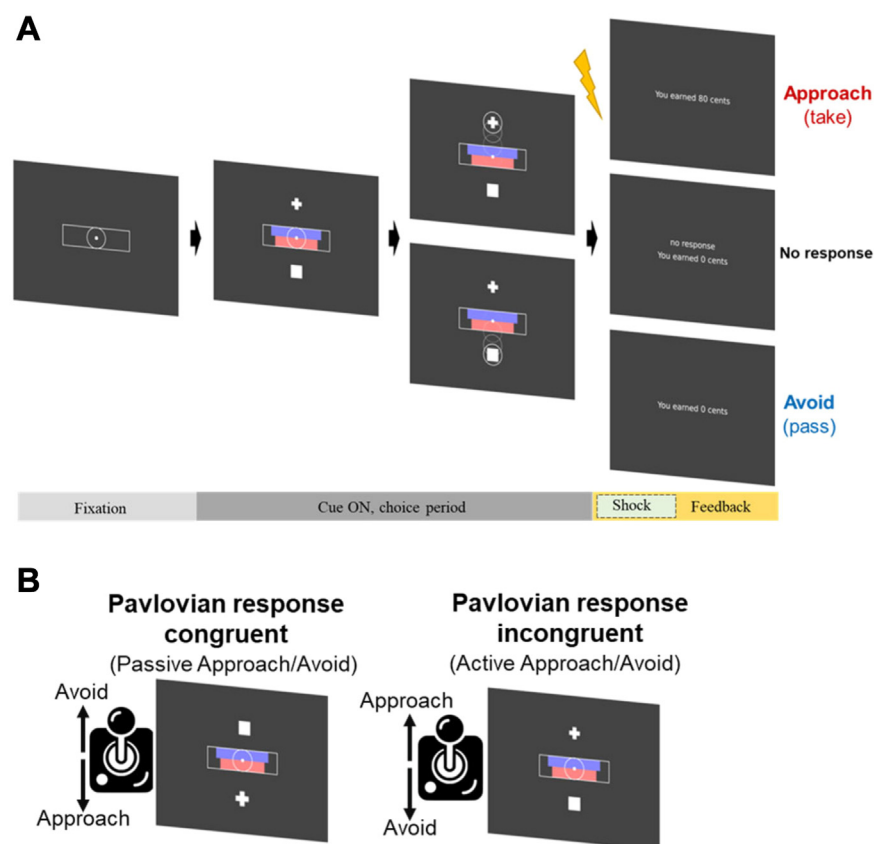


Figure 1. Approach-avoidance conflict task. **(A)** Participants decided to approach or avoid offers with monetary reward (magnitude represented by blue bars) and electrical shock (magnitude represented by red bars). **(B)** Trials were counterbalanced including either Pavlovian response-congruent trials or Pavlovian response-incongruent trials.

based on clinical measures or evaluated using clinical interviews. They received \$22.50 for performing the AAC task and completing the clinical questionnaires and a performance-based bonus (maximal \$27.10). For process-oriented computational analyses, all 50 datasets were used, whereas 2 participants were omitted from questionnaire-based analyses because they did not complete the self-report assessments.

AAC Paradigm

A total of 105 offers were presented one at a time. Each offer was composed of a monetary reward component and an aversive (electrical shock) component displayed by horizontal bars (Figure 1). After a fixation period, response symbols (i.e., a plus sign represented approach choices, while a square represented avoidance choices) were simultaneously presented with the offer. Offers were dynamically created on a trial-by-trial basis for each participant. See the Supplement for additional details.

Beck Depression Inventory-II

The Beck Depression Inventory-II (69) assesses the severity of depressive symptoms (70,71). Participants rate each symptom during the past 2 weeks on a scale from 0 (not feeling or experiencing the symptom) to 3 (feeling or experiencing the symptom to an extreme extent). Raw scores range from 0 to 63, with scores below 13 indicating minimal to no depression

severity. Scores from 14 to 19, 20 to 28, and >29 indicate mild, moderate, and severe depression severity, respectively.

Snaith-Hamilton Pleasure Scale

The Snaith-Hamilton Pleasure Scale (72) assesses hedonic capacity (41,73,74) and consists of 14 statements that assess an individual's capacity to experience pleasure. Participants indicate their agreement with each statement, considering the previous few days, on a scale from 1 (definitely agree) to 4 (definitely disagree). Total scores range from 14 to 56, with higher scores representing more anhedonia.

Positive and Negative Affect Schedule

The Positive and Negative Affect Schedule (PANAS) (75) assesses positive affect and negative affect. Respondents indicate how strongly they identify with 20 descriptions on a scale of 1 (very slightly or not at all) to 5 (extremely) based on their mood during the past 2 weeks (75). The PANAS yields 2 scores (PANAS-positive affect and PANAS-negative affect) ranging from 10 to 50, with higher scores indicating greater levels of positive or negative affect.

Visual Analog Mood Scale

The Visual Analog Mood Scale (76) assesses current mood states. Participants view horizontal lines ranging from 0 to 100, each corresponding to a bipolar mood spectrum: happy-sad,

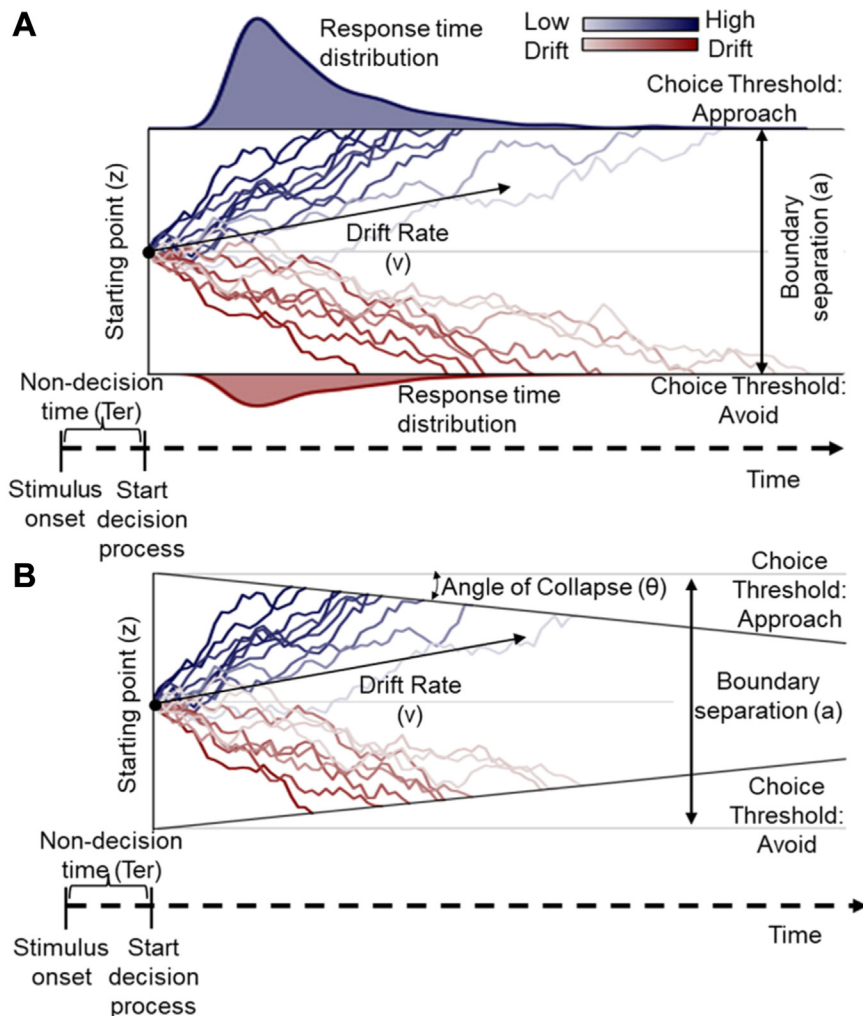


Figure 2. Computational account of behavior in the approach-avoidance conflict task decomposed decision processes into distinct and quantifiable latent cognitive components. **(A)** The diffusion decision model [Ratcliff (54)] simulates decisions by noisy evidence processes that evolve over time toward one of the 2 boundaries that represent the 2 response options. The set of model parameters (e.g., T_{er} , z , a , v) reproduces behavior, whereby each parameter quantifies a distinct cognitive signature of the decision dynamics. **(B)** An alternative sequential sampling model with linear collapsing decision boundaries that presumes different decision dynamics than the diffusion decision model.

tense-relaxed, and friendly-hostile. Participants choose a point on each line that best characterizes their current mood. We converted the scores on each scale such that higher scores indicated more negative affect.

Mood and Anxiety Symptom Questionnaire

The Mood and Anxiety Symptom Questionnaire (77) assesses symptoms related to anxiety and depression. Participants rate the presence of 62 symptoms during the past week on a scale from 1 (very slightly or not at all) to 5 (extremely). We used the anxiety-related subscores—general distress: anxiety and anxious arousal—to account for anxiety-related symptoms.

Cognitive and Behavioral Avoidance Scale

The Cognitive and Behavioral Avoidance Scale (78) assesses avoidance behavior associated with anxiety and depression. Thirty-one items describe different avoidant behaviors that participants rate on a scale from 1 (not at all) to 5 (extremely). A higher score indicates more avoidance tendencies.

Questionnaire Assessment

All scales had excellent internal reliability (Cronbach's alpha ranging from 0.82 to 0.94) (see the Supplement and Figure S1).

Analytics

We fit different versions of SSMS to single-trial RTs and choices within a Bayesian hierarchical framework using the open-source toolbox HDDM (64,65,79) (see the Supplement). Then, we selected the best model in terms of both deviance information criterion and posterior predictive checks. Because offers (i.e., presented reward, aversiveness, conflict) varied on each trial, model parameters specified by these stimulus attributes also varied on a trial-by-trial basis. We provide model comparison in the Supplement and focus on clinical relationships with the best-fitting model. The Supplement also provides parameter recovery assessments by generating simulated data (using the participants' offers and the estimated model parameters as inputs) and then determining whether the fitted parameters were recovered. We examined posterior

Multidimensional Computational Phenotyping

distributions of estimated coefficients to assess their significance in simultaneously predicting RTs and choices on a trial-by-trial basis. For brevity, we report point estimates (posterior means) and 95% CIs for all covariates in the [Supplement](#).

Computational Phenotyping

To explore links between symptoms and computational model parameters, we first conducted correlational analyses between the parameters of the best-fitting model and raw questionnaire scores. We examined the linearity of correlations with scatterplots ([Supplement](#)). After identifying statistically significant associations, we assessed the clinical relevance of these associations with multivariate regression models. Specifically, the questionnaire scores served as dependent variables, while the model parameters served as predictors. Covariates were z scored before entering the regression models. We compared model performance with *F* test analyses. We provide additional sensitivity analyses in [Tables S8](#) and [S9](#). To establish the benefits of SSM parameters over conventional performance measures, we estimated multivariate regression models with mean RTs and choice frequencies as predictors (with severity scores as dependent variables).

RESULTS

Our sample ($n = 50$) included 35 women (mean age = 29 years, $SD = 7$ years) with a broad range of symptom severity related to depression and anhedonia (but low levels of anxiety) (see [Table S1](#) and [Figure S2](#)).

Relative Frequency and Speed of Decisions Depended on Domain Type

[Figure 3](#) simultaneously presents choice frequencies and RT quantiles. Offers with positive net values (reward minus aversiveness) comprise the positive domain, while those with negative net values comprise the negative domain. Across both domains, the frequency of approach decisions increased as the offers' net values increased. This pattern is consistent with an evidence accumulation model wherein the strength of net evidence accumulation (drift rate) for approach is proportional to the difference between reward and aversion.

Models With Linear Collapsing Boundaries Performed Best

The SSM with linear collapsing boundaries outperformed other SSM versions in terms of both the deviance information criterion and posterior predictive checks ([Tables S2](#), [S3](#); [Figures S3](#), [S4](#)). Overall, simulations showed good parameter recovery, particularly for the parameters that are included in the main analysis presented below ([Figure S5](#)). The recovery of drift rates for the positive domain was poorer than for the negative domain, leading to lower power to detect effects in the positive domain. [Figure S6](#) shows that this was due to the trial-by-trial creation of offers, which led to a higher sampling of reward-aversion combinations for the negative domain (due to less consistent choices of approach relative to avoidance) than the positive domain. Therefore, we do not overly interpret the difference between positive and negative domains.

The best-fitting model ([Figure 3B](#); [Figure S7](#)) included linear boundary collapses that varied by conflict (defined as the

absolute difference between reward and aversion). Drift rates toward approach varied by reward, aversion, and domain type. Higher conflict was associated with decreased boundary separation (a : mean posterior point estimate $\beta = -0.035$, $SD = 0.017$) and slower collapsing rates (θ : $\beta = -0.031$, $SD = 0.012$). Statistics for posterior distributions are presented in [Table S4](#), and correlations between parameters are provided in [Figure S8](#).

Multidimensional Computational Phenotyping

The severity of depression and anhedonia were moderately correlated ($R = 0.51$, $p \leq .001$). Correlational analyses between best-fitting model parameters and questionnaire scores identified cognitive signatures of depression severity, anhedonia, and affective states ([Figure 4A](#)). [Figure 4B, C](#) shows the linear associations for 2 parameters (also see [Figures S9](#) and [S10](#)).

Distinguishing Between Depressive and Anhedonic Symptoms

Greater depression severity was associated with weaker approach biases on a Pavlovian congruent (passive avoidance) trial (zPB_c : $R = -0.34$, $p = .019$) ([Figure 4A](#)), accounting for asymmetric effects in the RT distributions of avoidance versus approach choices. Moreover, greater depression severity was associated with longer nondecision times (T_{er} : $R = 0.37$, $p = .011$) ([Figure 4A](#)), accounting for right-shifted RT distributions of both choice types.²

A multivariate regression model with depression severity as the dependent variable and computational parameters (T_{er} , zPB_c) as independent variables showed that depression severity was related to both nondecision time (T_{er} : coefficient $\beta = 0.319$, $SD = 0.134$, $p = .022$) and passive avoidance tendencies (zPB_c : $\beta = -0.286$, $SD = 0.134$, $p = .038$), adjusted $R^2 = 0.179$. Subsequent *F* test analyses illustrated that this multivariate model (M1), which included both parameters as main effects, outperformed alternative, univariate models ([Table S5](#)).

Dissecting Reward and Aversion Sensitivity and Their Associations With Affective States

Increased reward sensitivity in the negative domain ([Figure 4A](#): $V_{reward,neg}$) was associated with lower positive affect ($p = .022$) and more sadness ($p = .042$). This means that for adults who endorsed lower positive affect and more sadness, marginal reward increases were less effective in switching choices from avoidance to approach when offers had negative net values.

While positive affect and sadness were inversely related to reward sensitivity in the negative domain, more tension was associated with increased aversion sensitivity in that domain ($V_{averse,neg}$: $p = .025$) ([Figure 4A](#)). More tension was also associated with faster decision boundary collapses as conflict increased ($\theta_{conflict}$: $p = .049$) ([Figure 4A](#)). However, the

²Follow-up analyses showed a strong positive association between depression severity and severity of universal avoidance behavior ($R = 0.81$, $p < .001$) as measured by the total score on the Cognitive and Behavioral Avoidance Scale (see [Methods and Materials](#)). Cognitive and Behavioral Avoidance Scale-related avoidance severity was also solely associated with longer nondecision time ($R = 0.31$, $p = .031$).

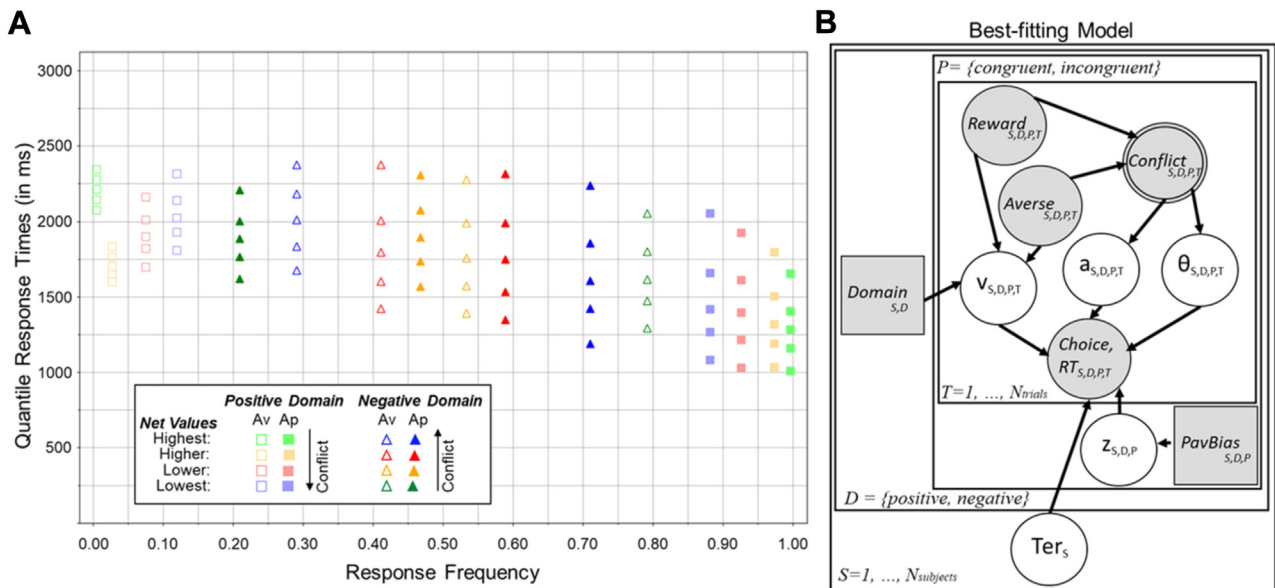


Figure 3. Reducing conflict increased the frequency of approaches and enhanced the consistency of decisions. **(A)** Quantile-probability plot for offers that were equally binned (for each domain separately) into 4 conditions (highest, higher, lower, lowest) based on their net values. Note that offers were binned for illustration purposes only. The x-axis represents choice frequency of approach (filled symbols) and avoidance (unfilled symbols). Vertical columns represent the quantile response times, referring to the 0.1, 0.3, 0.5 (median), 0.7, and 0.9 quantiles when moving from bottom to top. As conflict decreased, approaches occurred more frequently, and their speed became less dispersed (more consistent). The 2 domains also induced distinct effects on the relative speed of approach vs. avoidance decisions. **(B)** Graphical representation of the best-fitting model that included linear collapsing boundaries. Shaded nodes represent observed variables, and nonshaded nodes represent estimated parameters. Circles represent continuous variables; squares represent discrete variables. Conflict refers to the absolute difference between reward and aversion and is therefore computed and double bordered. Drift rates varied by offers and their domains, while boundary separation and their linear collapses varied by conflict. Starting points varied by active/passive approach tendencies. θ , angle of linear collapse; a , boundary separation; Ap , approach; Av , avoidance; D , domain; P , Pavlovian congruency; $PavBias$, Pavlovian bias; RT , response time; S , subjects; T , trials; T_{er} , nondesideration time; v , drift rate; z , starting points.

multivariate model (M1) with tension as the dependent variable and both parameters ($v_{averse,neg}$, $\theta_{conflict}$) as main effects did not perform better than a univariate model (M2) with only $v_{averse,neg}$ as a covariate (Table S5). Therefore, tension seemed to be predominantly associated with increased aversion sensitivity in the negative domain.

Cognitive Signatures of Negative Affect

More negative affect was associated with decreased boundary separation (a : $p = .005$) (Figure 4A), leading to less consistent response patterns for offers close to the border of the positive and negative domain. Additionally, more negative affect was associated with more active approach tendencies (zPB ; $p = .023$) (Figure 4A). Multivariate regression models revealed an association between the magnitude of negative affect and a main effect of boundary separation (a : $\beta = -0.366$, $SD = 0.128$, $p = .007$) and its interaction with active approach tendencies (a -by- zPB ; interaction: $\beta = -0.335$, $SD = 0.131$, $p = .014$), but the main effect of active approach was no longer significant (zPB ; $\beta = 0.137$, $SD = 0.135$, $p = .318$), adjusted $R^2 = 0.272$. Therefore, participants with less cautious response patterns exhibited more negative affect, even more so if they also demonstrated active approach tendencies. Subsequent F test analyses showed that the multivariate model (M1) that included both model parameters and their interaction outperformed alternative multivariate and univariate models (Table S5).

Computational Phenotyping Versus Summary Statistics

Next, we evaluated whether computational parameters were better predictors of clinically relevant constructs than conventional performance measures. Figure 5 shows the estimated coefficients from multivariate regression models with clinical constructs (1 per subplot) as dependent variables. Model A included conventional performance measures as covariates, while models B and C included computational parameters as covariates. As marked by the asterisks (indicating statistical significance) in Figure 5, only the computational parameters were related to clinical constructs (except for the case where mean RT was related to negative affect, shown in Figure 5C). Additional statistics are provided in Tables S6 and S7.

DISCUSSION

In an unselected community sample with varying symptoms, we probed cognitive signatures related to depressive symptom severity, anhedonia, and affective states in an AAC task with computational modeling. The SSM that accounted best for the decision dynamics included linear collapsing boundaries that varied by conflict, starting points that varied by response modes, and domain-specific drift rates that distinguished between reward and aversion sensitivity. Critically, this process-oriented account deconstructed behavior into separate and

Multidimensional Computational Phenotyping

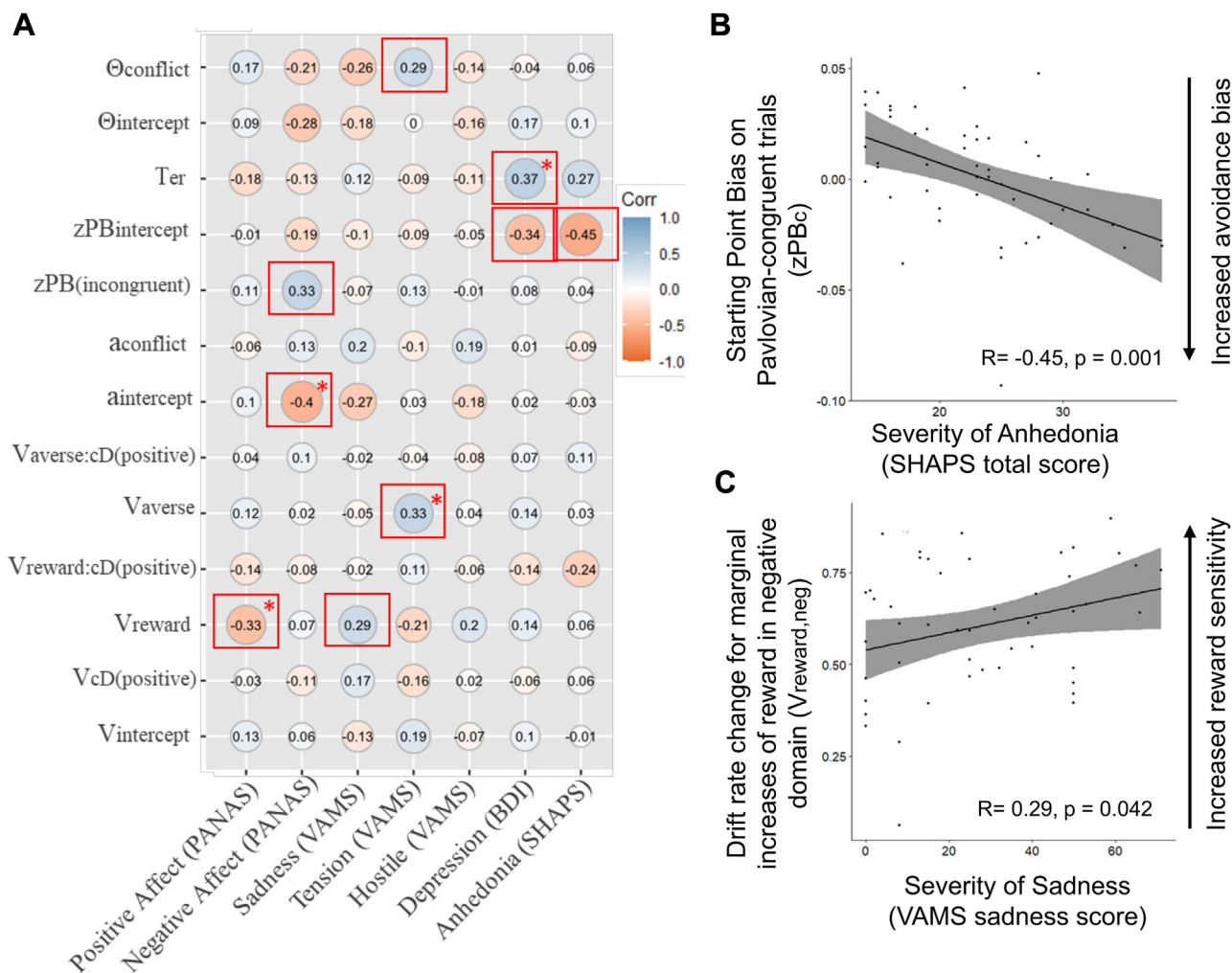


Figure 4. Different cognitive characteristics of depressive and anhedonic symptoms and affective states. **(A)** Depressive symptoms, anhedonia, and affective states were correlated with distinct cognitive signatures as indexed by varying model parameters (a refers to boundary separation, cD refers to the domains [positive, negative], PB refers to the Pavlovian response incongruent/congruent trials, Ter refers to nondesideration time, θ refers to angle of linear collapse, v refers to drift rate, and z refers to starting points). Significant (p values $< .05$ and unadjusted for multiple comparisons) correlations are surrounded by red boxes. For correlation pairs that showed a moderate to strong correlation strength ($R \geq 0.30$), we also estimated p values adjusted using false discovery rate correction. The correlations that showed an adjusted p value $< .05$ are also marked by an asterisk. **(B)** Linear association between anhedonia and starting point bias for Pavlovian-congruent trials. Black dots indicate data. Means (solid lines) and corresponding 95% CIs are shown as shaded intervals. **(C)** Linear association between sadness and drift rate for marginal reward changes in the negative domain. BDI, Beck Depression Inventory; Corr, correlation; PANAS, Positive and Negative Affect Schedule; SHAPS, Snaith-Hamilton Pleasure Scale; VAMS, Visual Analog Mood Scale.

quantifiable components (cognitive signatures) that were associated with distinct clinical constructs. We also demonstrated the utility of computational phenotyping over conventional performance measures by showing that, with one exception, SSM parameters were more predictive of symptom scores.

Our adaptive AAC task, together with computational modeling, allowed us to separate behavioral effects due to conflict and impatience (indexed by boundary separation and boundary collapses), response biases (indexed by starting points), and reward and aversion sensitivity (indexed by drift rates). In previous studies, estimated reward and aversion sensitivity could have been influenced by individual differences in preferences, marginal

rates of substitutions, and/or differences in relative potency, timing, and duration of reward and aversion (41,80).

Anhedonia and depressive symptoms were both associated with more passive avoidance tendencies. However, greater depressive symptoms were uniquely associated with longer nondesideration times. At first, this may seem to contradict findings from previous studies that found depression characteristics to manifest in drift rates and starting point biases (81,82). However, these studies focused on categorical assessments of depression and used tasks (e.g., perceptual discrimination tasks) that are meant to tap into other cognitive constructs. This is important to consider because task specifics determine the precise interpretations of model parameters (24).

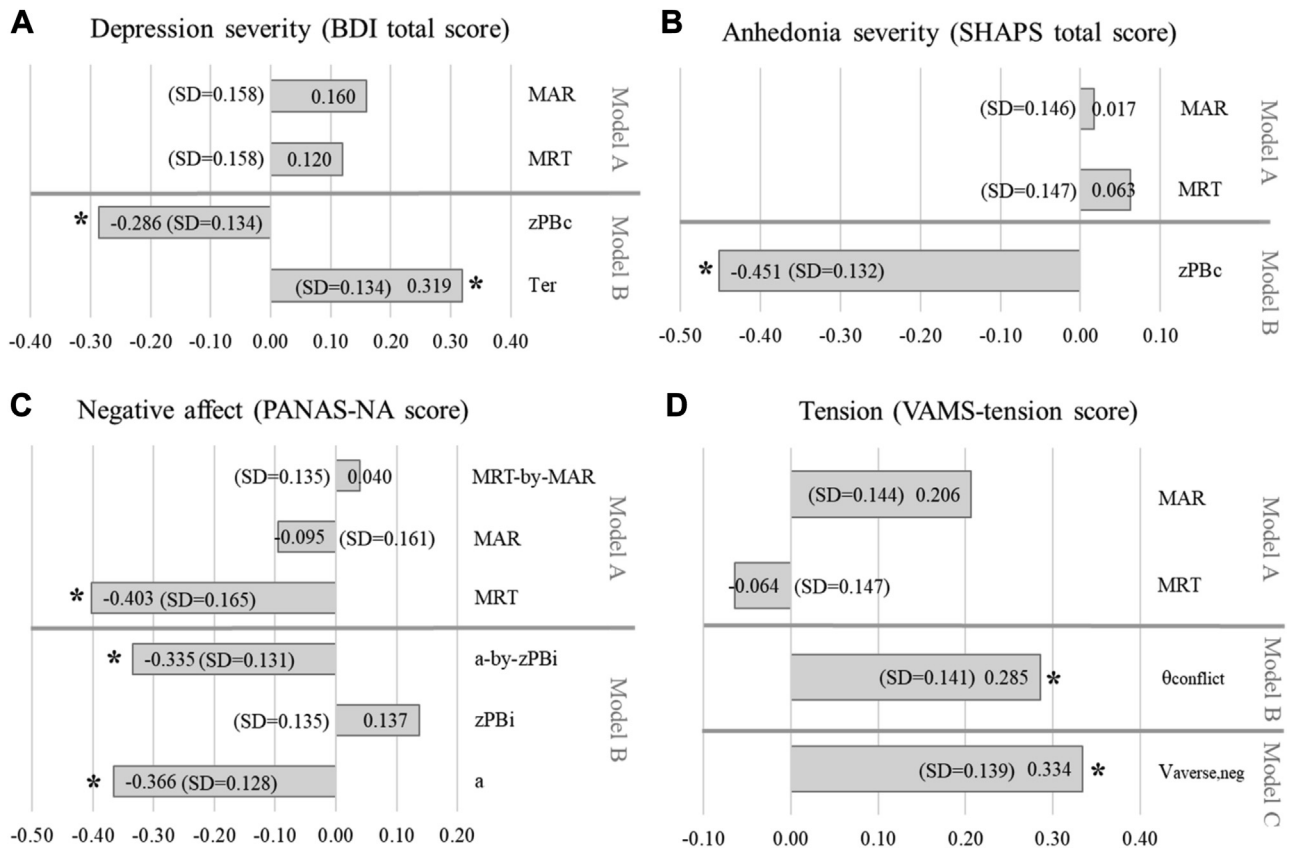


Figure 5. Computational parameters were correlated with symptom scores, whereas summary statistics (mean frequency rates and mean response times) were not. Shown are regression coefficients (means and SDs) of different models specified subsequently. Asterisks indicate significant regressors ($p < .05$). Regression outputs are provided in [Tables S5 and S6](#). **(A)** Models with depression severity as the dependent variable. Model A included main effects of mean approach frequency (MAR) and mean response time (MRT) as covariates. Model B included main effects of nondecision time (T_{er}) and starting point bias on Pavlovian-incongruent trials ($zBias_s$) as covariates. **(B)** Models with anhedonia severity as the dependent variable. Model A included main effects of MAR and MRT as covariates. Model B included the main effect of starting point bias on Pavlovian-congruent trials ($zBias_c$) as covariate. **(C)** Models with negative affect (NA) as the dependent variable. Model A included main effects of MAR and MRT as covariates. Model B included main effects of boundary separation (for average conflict trials) and starting point bias on Pavlovian-incongruent trials ($zBias_s$) as well as their interaction as covariates. **(D)** Models with tension severity as the dependent variable. Model A included main effects of MAR and MRT as covariates. Model B included the rate of the boundary collapse on trials with higher conflict than average (θ_{conflict}) as covariate. Model C included aversion sensitivity (indicated by drift rate) in the negative domain ($V_{\text{averse,neg}}$) as covariate. BDI, Beck Depression Inventory; PANAS, Positive and Negative Affect Schedule; SHAPS, Snaith-Hamilton Pleasure Scale; VAMS, Visual Analog Mood Scale.

Therefore, the specifics of our task (i.e., forming a representation of reward relative to aversion by extracting the relative size of horizontal bars without providing an explicit reference point) might have made it more sensitive to detecting clinical differences in early-stage components of decision processes.

Clinical measures of depression and anhedonia comprise heterogeneous symptom profiles. Therefore, it is not surprising that measures of more specific affective states show stronger associations with reward and aversion sensitivities. Our findings highlight that distinct latent cognitive signatures (quantified and estimated by the computational model parameters) can help to define different phenotypes (e.g., decreased positive affect in a subgroup of people with depression) (83,84).

Higher positive affect was associated with decreased reward sensitivity (in the negative domain). This is consistent with previous research suggesting that positive affect can lead to optimistic biases in negative contexts and therefore less

sensitive responses to changes in reward (85). Moreover, we found that more sadness was associated with increased reward sensitivity in the negative domain. Because depression can lead to reduced positive affect (10,83) and/or increased sadness (17), our study shows how these affective states are linked to different decision-making biases, in addition to those influenced by the severity of depression itself.

Higher negative affect was associated with both elevated approach under active response mode and less consistent choice patterns for offers with average conflict levels. It is intriguing that positive and negative affect as measured by the PANAS (75) mapped onto different model parameters. However, this finding is consistent with the notion that positive and negative affect are divergent concepts rather than 2 sides of the same coin (86–89).

Tension has been proposed as one of the defining components of mood (90). We found more tension to be associated

Multidimensional Computational Phenotyping

with increased urgency signals (more impatience) as conflict increased (i.e., faster collapsing decision boundaries). This is consistent with previous research that associated relaxation (the opposite end of the Visual Analog Mood Scale spectrum from tense to relaxed) with low urgency signals (90).

Limitations and Outlook

We focused on 2 widely used measures to assess severity of depression [Beck Depression Inventory-II (69)] and anhedonia [Snaith-Hamilton Pleasure Scale (72)] and a few affect and mood measures known to be modulated by depression. More research is needed to link cognitive processes to other clinical measures that are sensitive to various aspects of depression and anhedonia. Specifically, future studies should target larger sample sizes and sample across the entire severity spectrum, as well as consider categorical assessments, comorbid diagnoses, and sex differences to further increase the generalizability of our results (74,77). While our sample showed only marginal variability in anxiety severity, future studies that apply multidimensional computational phenotyping are needed to dissociate anxiety-related and depression-related cognitive signatures as well as possible distinct neurobiological mechanisms. Finally, task design and model configurations should be co-developed to guarantee optimal parameter recovery. We emphasize that some model parameters showed better recovery in supplementary recovery analyses. While we focused our interpretation on the model parameters that showed robust recovery, other relationships may exist between parameters and scores that we did not have sufficient power to detect (e.g., measurement errors) due to a restricted range of symptoms.

The main purpose of this study was to explore associations between model parameters and symptom severity of depression, anhedonia, and affective states using an adaptive AAC task and computational modeling. These associations need to be tested more rigorously in future studies that also include more representative samples as detailed above.

Understanding how different affective states map onto distinct cognitive biases is important because it may help to define phenotypes of depression as well as new mechanisms that can be targeted in clinical interventions (26,74,91–93). Identifying how different affective states manifest in behavior is also critical for differential diagnostics and for assessing other co-occurring disorders (e.g., attention-deficit/hyperactivity disorder) that are often also characterized by mood disturbances but due to distinct hypothesized mechanisms (26,31,94,95).

ACKNOWLEDGMENTS AND DISCLOSURES

This work was partially supported by the National Institute of Mental Health (Grant Nos. P50MH119467 [to DAP and MJF]; P50MH106435-06A1 [to MJF]; T32MH126388-01 [to MJF and NRG-J]; R01MH084840 and R01MH115905-01 [to MJF]; R01MH111676 [to DGD]; R01MH108602, R37MH068376, and R01MH101521 [to DAP]; and K23NS099380 [to TMH]). The funders had no role in study design, data collection, or analysis. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. This work was conducted using computational resources and services at the Center for Computation and Visualization, Brown University, which is supported by the National Institutes of Health (Grant No. S10OD025181).

We thank the Nock Lab in the Department of Psychology at Harvard University for providing the facilities to conduct this study.

NRG-J has received research funding from the Swiss National Science Foundation and the National Institute of Mental Health. She also co-founded BGBehavior LLC. TMH has received consulting fees from Medtronic, Inc.; he has received stock options from MarvelBiome. MJF has received consulting fees from F. Hoffman La Roche Pharmaceuticals for activities unrelated to this project. Over the past 3 years, DAP has received consulting fees from Boehringer Ingelheim, Compass Pathways, Engrail Therapeutics, Karla Therapeutics, Neumora Therapeutics (formerly BlackThorn Therapeutics), Neurocrine Biosciences, Neuroscience Software, Otsuka, Sama Therapeutics, Sage Therapeutics, Sunovion, and Takeda; honoraria from the American Psychological Association, Psychonomic Society, and Springer (for editorial work) and from Alkermes; research funding from the Brain and Behavior Research Foundation, Dana Foundation, Wellcome Leap, Millennium Pharmaceuticals, and the National Institute of Mental Health; and stock options from Compass Pathways, Engrail Therapeutics, Neumora Therapeutics, and Neuroscience Software. No funding from these entities was used to support the current work, and all views expressed are solely those of the authors. All other authors report no biomedical financial interests or potential conflicts of interest.

ARTICLE INFORMATION

From the Carney Institute for Brain Science, Department of Cognitive, Linguistic, & Psychological Sciences, Brown University, Providence, Rhode Island (NRG-J, PC, MJF); Center for Depression, Anxiety and Stress Research, McLean Hospital, Belmont, Massachusetts (MK, JMB, DCS, DGD, DAP); Department of Psychiatry, Harvard Medical School, Boston, Massachusetts (MK, DGD, DAP); Warren Alpert Medical School of Brown University, Providence, Rhode Island (PC); and Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts (ZY, TMH).

NRG-J and MK are joint first authors.

DAP and MJF are joint senior authors.

Address correspondence to Nadja R. Ging-Jehli, Ph.D., at nadja@gingjehli.com.

Received Aug 9, 2023; revised Feb 3, 2024; accepted Feb 9, 2024.

Supplementary material cited in this article is available online at <https://doi.org/10.1016/j.bpsc.2024.02.005>.

REFERENCES

- Panchal N, Kamal R, Cox C, Garfield R (2021): The implications of COVID-19 for mental health and substance use. *Omeka* Available at: <https://is101fall2021.web.illinois.edu/items/show/467>. Accessed April 20, 2023.
- Reinert M, Fritze D, Nguyen T (2022): *The State of Mental Health in America 2023*. Alexandria, VA: Mental Health America.
- McMakin DL, Olino TM, Porta G, Dietz LJ, Emslie G, Clarke G, *et al.* (2012): Anhedonia predicts poorer recovery among youth with selective serotonin reuptake inhibitor treatment-resistant depression. *J Am Acad Child Adolesc Psychiatry* 51:404–411.
- Moos RH, Cronkite RC (1999): Symptom-based predictors of a 10-year chronic course of treated depression. *J Nerv Ment Dis* 187:360–368.
- Wardenaar KJ, Giltay EJ, van Veen T, Zitman FG, Penninx BWJH (2012): Symptom dimensions as predictors of the two-year course of depressive and anxiety disorders. *J Affect Disord* 136:1198–1203.
- Spijker J, Bijl RV, de Graaf R, Nolen WA (2001): Determinants of poor 1-year outcome of DSM-III-R major depression in the general population: Results of the Netherlands Mental Health Survey and Incidence Study (NEMESIS). *Acta Psychiatr Scand* 103:122–130.
- Uher R, Perlis RH, Henigsberg N, Zobel A, Rietschel M, Mors O, *et al.* (2012): Depression symptom dimensions as predictors of antidepressant treatment outcome: Replicable evidence for interest-activity symptoms. *Psychol Med* 42:967–980.
- Wang S, Leri F, Rizvi SJ (2021): Anhedonia as a central factor in depression: Neural mechanisms revealed from preclinical to clinical evidence. *Prog Neuropsychopharmacol Biol Psychiatry* 110:110289.

9. Whitton AE, Kumar P, Treadway MT, Rutherford AV, Ironside ML, Foti D, *et al.* (2023): Distinct profiles of anhedonia and reward processing and their prospective associations with quality of life among individuals with mood disorders. *Mol Psychiatry*. [published online Jul 4].
10. Flores-Kanter PE, Garrido LE, Moretti LS, Medrano LA (2021): A modern network approach to revisiting the Positive and Negative Affective Schedule (PANAS) construct validity. *J Clin Psychol* 77:2370–2404.
11. van Roekel E, Bennis EC, Bastiaansen JA, Verhagen M, Ormel J, Engels RCME, Oldehinkel AJ (2016): Depressive symptoms and the experience of pleasure in daily life: An exploration of associations in early and late adolescence. *J Abnorm Child Psychol* 44:999–1009.
12. Blyzma LM, Taylor-Clift A, Rottenberg J (2011): Emotional reactivity to daily events in major and minor depression. *J Abnorm Psychol* 120:155–167.
13. Clark LA, Watson D, Mineka S (1994): Temperament, personality, and the mood and anxiety disorders. *J Abnorm Psychol* 103:103–116.
14. Peeters F, Nicolson NA, Berkhof J, Delespaul P, deVries M (2003): Effects of daily events on mood states in major depressive disorder. *J Abnorm Psychol* 112:203–211.
15. Power MJ, Tarsia M (2007): Basic and complex emotions in depression and anxiety. *Clin Psychol Psychother* 14:19–31.
16. Werner-Seidler A, Banks R, Dunn BD, Moulds ML (2013): An investigation of the relationship between positive affect regulation and depression. *Behav Res Ther* 51:46–56.
17. Husain M, Roiser JP (2018): Neuroscience of apathy and anhedonia: A transdiagnostic approach. *Nat Rev Neurosci* 19:470–484.
18. Saxena A, Luking KR, Barch DM, Pagliaccio D (2017): Individual differences in hedonic capacity, depressed mood, and affective states predict emotional reactivity. *Motiv Emot* 41:419–429.
19. Heininga VE, Van Roekel E, Ahles JJ, Oldehinkel AJ, Mezulis AH (2017): Positive affective functioning in anhedonic individuals' daily life: Anything but flat and blunted. *J Affect Disord* 218:437–445.
20. Heininga VE, Dejonckheere E, Houben M, Obbels J, Sienaert P, Leroy B, *et al.* (2019): The dynamical signature of anhedonia in major depressive disorder: Positive emotion dynamics, reactivity, and recovery. *BMC Psychiatry* 19:59.
21. Abramovitch A, Short T, Schweiger A (2021): The C Factor: Cognitive dysfunction as a transdiagnostic dimension in psychopathology. *Clin Psychol Rev* 86:102007.
22. East-Richard C R, Mercier A, Nadeau D, Cellard C (2020): Transdiagnostic neurocognitive deficits in psychiatry: A review of meta-analyses. *Canadian Psychology/Psychologie canadienne* 61:190–214.
23. Geana A, Barch DM, Gold JM, Carter CS, MacDonald AW, Ragland JD, *et al.* (2022): Using computational modeling to capture schizophrenia-specific reinforcement learning differences and their implications on patient classification. *Biol Psychiatry Cogn Neurosci Neuroimaging* 7:1035–1046.
24. Ging-Jehli NR, Ratcliff R, Arnold LE (2021): Improving neurocognitive testing using computational psychiatry—A systematic review for ADHD. *Psychol Bull* 147:169–231.
25. Ging-Jehli NR, Arnold LE, Roley-Roberts ME, deBeus R (2022): Characterizing underlying cognitive components of ADHD presentations and co-morbid diagnoses: A diffusion decision model analysis. *J Atten Disord* 26:706–722.
26. Ging-Jehli NR, Kraemer HC, Eugene Arnold L, Roley-Roberts ME, deBeus R (2023): Cognitive markers for efficacy of neurofeedback for attention-deficit hyperactivity disorder – Personalized medicine using computational psychiatry in a randomized clinical trial. *J Clin Exp Neuropsychol* 45:118–131.
27. Ossola P, Pike AC (2023): Editorial: What is computational psychopathology, and why do we need it? *Neurosci Biobehav Rev* 152:105170.
28. Pedersen ML, Ironside M, Amemori KI, McGrath CL, Kang MS, Graybiel AM, *et al.* (2021): Computational phenotyping of brain-behavior dynamics underlying approach-avoidance conflict in major depressive disorder. *PLoS Comput Biol* 17:e1008955.
29. Ratcliff R, Scharre DW, McKoon G (2022): Discriminating memory disordered patients from controls using diffusion model parameters from recognition memory. *J Exp Psychol Gen* 151:1377–1393.
30. Wiecki TV, Antoniadis CA, Stevenson A, Kennard C, Borowsky B, Owen G, *et al.* (2016): A computational cognitive biomarker for early-stage Huntington's disease. *PLoS One* 11:e0148409.
31. Ging-Jehli NR, Arnold LE, Van Zandt T (2023): Cognitive-attentional mechanisms of cooperation—with implications for attention-deficit hyperactivity disorder and cognitive neuroscience. *Cogn Affect Behav Neurosci* 23:1545–1567.
32. Fried EI, Flake JK, Robinaugh DJ (2022): Revisiting the theoretical and methodological foundations of depression measurement. *Nat Rev Psychol* 1:358–368.
33. Kirlic N, Young J, Aupperle RL (2017): Animal to human translational paradigms relevant for approach avoidance conflict decision making. *Behav Res Ther* 96:14–29.
34. Letkiewicz AM, Kottler HC, Shankman SA, Cochran AL (2023): Quantifying aberrant approach-avoidance conflict in psychopathology: A review of computational approaches. *Neurosci Biobehav Rev* 147:105103.
35. Lewin K (1935): *A Dynamic Theory of Personality*. New York, NY: McGraw-Hill.
36. Loijen A, Vrijns JN, Egger JIM, Becker ES, Rinck M (2020): Biased approach-avoidance tendencies in psychopathology: A systematic review of their assessment and modification. *Clin Psychol Rev* 77:101825.
37. Miller NE (1959): Liberalization of basic S-R concepts: Extensions to conflict behavior, motivation and social learning. In: Koch S, editor. *Psychology: A Study of a Science. General and Systematic Formulations, Learning, and Special Processes*. New York: McGraw-Hill, 196–292.
38. Ironside M, Amemori K-I, McGrath CL, Pedersen ML, Kang MS, Amemori S, *et al.* (2020): Approach-avoidance conflict in major depressive disorder: Congruent neural findings in humans and nonhuman primates. *Biol Psychiatry* 87(5):399–408.
39. Livermore JJA, Klaassen FH, Bramson B, Hulsman AM, Meijer SW, Held L, *et al.* (2021): Approach-avoidance decisions under threat: The role of autonomic psychophysiological states. *Front Neurosci* 15:621517.
40. Der-Avakian A, Markou A (2012): The neurobiology of anhedonia and other reward-related deficits. *Trends Neurosci* 35:68–77.
41. Rizvi SJ, Pizzagalli DA, Sproule BA, Kennedy SH (2016): Assessing anhedonia in depression: Potentials and pitfalls. *Neurosci Biobehav Rev* 65:21–35.
42. Ariely D, Huber J, Wertenbroch K (2005): When do losses loom larger than gains? *J Mark Res* 42:134–138.
43. Davis AL, Jehli N, Miller JH, Weber RA (2015): *Generosity across contexts*. Rochester, NY: Social Science Research Network; 2015 Mar [cited 2022 Jun 1]. Report No.: 2592357. Available at: <https://papers.ssrn.com/abstract=2592357>. Accessed June 1, 2022.
44. Kahneman D, Tversky A (1979): Prospect theory: An analysis of decision under risk. *Econometrica* 47:263–291.
45. Novemsky N, Kahneman D (2005): The boundaries of loss aversion. *J Mark Res* 42:119–128.
46. Deakin JFW, Graeff FG (1991): 5-HT and mechanisms of defence. *J Psychopharmacol* 5:305–315.
47. Hagura N, Haggard P, Diedrichsen J (2017): Perceptual decisions are biased by the cost to act. *eLife* 6:e18422.
48. Capra CM, Croson RT, Rigdon ML, Rosenblat TS (2020): *Handbook of Experimental Game Theory*. Cheltenham, UK: Edward Elgar Publishing.
49. Ratcliff R, Smith PL (2004): A comparison of sequential sampling models for two-choice reaction time. *Psychol Rev* 111:333–367.
50. Van Zandt T, Ratcliff R (1995): Statistical mimicking of reaction time data: Single-process models, parameter variability, and mixtures. *Psychon Bull Rev* 2:20–54.
51. Cartwright D, Festinger L (1943): A quantitative theory of decision. *Psychol Rev* 50:595–621.

Multidimensional Computational Phenotyping

52. Forstmann BU, Ratcliff R, Wagenmakers EJ (2016): Sequential sampling models in cognitive neuroscience: Advantages, applications, and extensions. *Annu Rev Psychol* 67:641–666.
53. Smith PL, Ratcliff R (2015): An introduction to the diffusion model of decision making. In: Forstmann BU, Wagenmakers EJ, editors. *An Introduction to Model-Based Cognitive Neuroscience*. New York, NY: Springer, 49–70.
54. Ratcliff R (1978): A theory of memory retrieval. *Psychol Rev* 85:59–108.
55. Moughrabi N, Botsford C, Gruichich TS, Azar A, Heilicher M, Hiser J, *et al.* (2022): Large-scale neural network computations and multivariate representations during approach-avoidance conflict decision-making. *Neuroimage* 264:119709.
56. Paulus MP (2020): Driven by pain, not gain: Computational approaches to aversion-related decision making in psychiatry. *Biol Psychiatry* 87:359–367.
57. Smith R, Kirlic N, Stewart JL, Touthang J, Kuplicki R, Khalsa SS, *et al.* (2021): Greater decision uncertainty characterizes a transdiagnostic patient sample during approach-avoidance conflict: A computational modelling approach. *J Psychiatry Neurosci* 46:E74–E87.
58. Smith R, Kirlic N, Stewart JL, Touthang J, Kuplicki R, McDermott TJ, *et al.* (2021): Long-term stability of computational parameters during approach-avoidance conflict in a transdiagnostic psychiatric patient sample. *Sci Rep* 11:11783.
59. Talmi D, Dayan P, Kiebel SJ, Frith CD, Dolan RJ (2009): How humans integrate the prospects of pain and reward during choice. *J Neurosci* 29:14617–14626.
60. Chu S, Hutcherson C, Ito R, Lee ACH (2023): Elucidating medial temporal and frontal lobe contributions to approach-avoidance conflict decision-making using functional MRI and the hierarchical drift diffusion model. *Cereb Cortex* 33:7797–7815.
61. Rolle CE, Pedersen ML, Johnson N, Amemori KI, Ironside M, Graybiel AM, *et al.* (2022): The role of the dorsal-lateral prefrontal cortex in reward sensitivity during approach-avoidance conflict. *Cereb Cortex* 32:1269–1285.
62. Fengler A, Govindarajan LN, Chen T, Frank MJ (2021): Likelihood approximation networks (LANs) for fast inference of simulation models in cognitive neuroscience. *eLife* 10:e65074.
63. Fengler A, Bera K, Pedersen ML, Frank MJ (2022): Beyond drift diffusion models: Fitting a broad class of decision and reinforcement learning models with HDDM. *J Cogn Neurosci* 34:1780–1805.
64. Bowman NE, Kording KP, Gottfried JA (2012): Temporal integration of olfactory perceptual evidence in human orbitofrontal cortex. *Neuron* 75:916–927.
65. Cisek P, Puskas GA, El-Murr S (2009): Decisions in changing conditions: The urgency-gating model. *J Neurosci* 29:11560–11571.
66. Ditterich J (2006): Evidence for time-variant decision making. *Eur J Neurosci* 24:3628–3641.
67. Thura D, Beauregard-Racine J, Fradet CW, Cisek P (2012): Decision making by urgency gating: Theory and experimental support. *J Neurophysiol* 108:2912–2930.
68. Bogacz R, Brown E, Moehlis J, Holmes P, Cohen JD (2006): The physics of optimal decision making: A formal analysis of models of performance in two-alternative forced-choice tasks. *Psychol Rev* 113:700–765.
69. Beck AT, Steer RA, Brown G (1996): Beck Depression Inventory–II. American Psychological Association. Available at: <http://doi.apa.org/getdoi.cfm?doi=10.1037/t00742-000>. Accessed January 24, 2023.
70. Furukawa TA (2010): Assessment of mood: Guides for clinicians. *J Psychosom Res* 68:581–589.
71. Smarr KL, Keefer AL (2020): Measures of depression and depressive symptoms. *Arthritis Care Res* 72(suppl 10):608–629.
72. Snaith RP, Hamilton M, Morley S, Humayan A, Hargreaves D, Trigwell P (1995): A scale for the assessment of hedonic tone the Snaith–Hamilton pleasure scale. *Br J Psychiatry* 167:99–103.
73. Nakonezny PA, Carmody TJ, Morris DW, Kurian BT, Trivedi MH (2010): Psychometric evaluation of the Snaith–Hamilton pleasure scale in adult outpatients with major depressive disorder. *Int Clin Psychopharmacol* 25:328–333.
74. Trøstheim M, Eikemo M, Meir R, Hansen I, Paul E, Kroll SL, *et al.* (2020): Assessment of anhedonia in adults with and without mental illness: A systematic review and meta-analysis. *JAMA Netw Open* 3:e2013233.
75. Watson D, Clark LA, Tellegen A (1988): Development and validation of brief measures of positive and negative affect: The Panas scales. *J Pers Soc Psychol* 54:1063–1070.
76. Stern RA, Arruda JE, Hooper CR, Wolfner GD, Morey CE (1997): Visual analogue mood scales to measure internal mood state in neurologically impaired patients: Description and initial validity evidence. *Aphasiology* 11:59–71.
77. Clark LA, Watson D (1991): Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *J Abnorm Psychol* 100:316–336.
78. Ottenbreit ND, Dobson KS (2004): Avoidance and depression: The construction of the Cognitive–Behavioral Avoidance Scale. *Behav Res Ther* 42:293–313.
79. Wiecki TV, Sofer I, Frank MJ (2013): HDDM: Hierarchical Bayesian estimation of the Drift-Diffusion Model in Python. *Front Neuroinform* 7:14.
80. Sherdell L, Waugh CE, Gotlib IH (2012): Anticipatory pleasure predicts motivation for reward in major depression. *J Abnorm Psychol* 121:51–60.
81. Lawlor VM, Webb CA, Wiecki TV, Frank MJ, Trivedi M, Pizzagalli DA, Dillon DG (2020): Dissecting the impact of depression on decision-making. *Psychol Med* 50:1613–1622.
82. Pizzagalli DA, Iosifescu D, Hallett LA, Ratner KG, Fava M (2008): Reduced hedonic capacity in major depressive disorder: Evidence from a probabilistic reward task. *J Psychiatr Res* 43:76–87.
83. Garcia-Guerrero S, O’Hora D, Zgonnikov A, Scherbaum S (2023): The action dynamics of approach-avoidance conflict during decision-making. *Q J Exp Psychol (Hove)* 76:160–179.
84. Kessler RC, Adler L, Barkley R, Biederman J, Conners CK, Demler O, *et al.* (2006): The prevalence and correlates of adult ADHD in the United States: Results from the national comorbidity survey replication. *Am J Psychiatry* 163:716–723.
85. Wichers M, Jacobs N, Derom C, Thiery E, van Os J (2007): Depression: Too Much Negative Affect or Too Little Positive Affect? *Twin Res Hum Genet* 10(suppl1):19–20.
86. Mehrabian A (1997): Comparison of the PAD and PANAS as models for describing emotions and for differentiating anxiety from depression. *J Psychopathol Behav Assess* 19:331–357.
87. Watson D, Weber K, Assenheimer JS, Clark LA, Strauss ME, McCormick RA (1995): Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *J Abnorm Psychol* 104:3–14.
88. Watson D, Clark LA (1991): Mood and anxiety symptom questionnaire. American Psychological Association. Available at: <http://doi.apa.org/getdoi.cfm?doi=10.1037/t13679-000>. Accessed January 24, 2023.
89. Zevon MA, Tellegen A (1982): The structure of mood change: An idiographic/nomothetic analysis. *J Pers Soc Psychol* 43:111–122.
90. Thayer RE (1996): *The Origin of Everyday Moods: Managing Energy, Tension, and Stress*. Oxford: Oxford University Press.
91. Garland EL, Atchley RM, Hanley AW, Zubieta JK, Froeliger B (2019): Mindfulness-Oriented Recovery Enhancement remediates hedonic dysregulation in opioid users: Neural and affective evidence of target engagement. *Sci Adv* 5:eaax1569.
92. Winer ES, Jordan DG, Collins AC (2019): Conceptualizing anhedonias and implications for depression treatments. *Psychol Res Behav Manag* 12:325–335.
93. Ging-Jehli NR, Painter QA, Kraemer HA, Roley-Roberts ME, Panchyshyn C, deBeus R, Arnold LE (2024): A diffusion decision model analysis of the cognitive effects of neurofeedback for ADHD. *Neuropsychology* 38:146–156.
94. American Psychiatric Association (2013): *Diagnostic and Statistical Manual of Mental Disorders, 5th ed.* Washington, DC: American Psychiatric Publishing.
95. McIntosh D, Kutcher S, Binder C, Levitt A, Fallu A, Rosenbluth M (2009): Adult ADHD and comorbid depression: A consensus-derived diagnostic algorithm for ADHD. *Neuropsychiatr Dis Treat* 5:137–150.