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Exploring the Link Between a Prior Belief for Active Avoidance and Apathy, Anhedonia, and Depression: A Network Analysis

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ABSTRACT

BACKGROUND: Learned helplessness has been suggested as a mechanism through which anhedonia in depression is developed. It has been theorized that this stems from a generalized pessimistic prior belief about the probability of success when attempting to actively avoid negative outcomes, thereby resulting in apathy and reduced motivation. However, how such a prior belief may relate to depression and apathy is unknown. Here, we leveraged a novel method based on cognitive tasks and Bayesian modeling to extract a reliable generalized prior belief expressing the probability with which negative outcomes are expected to be actively avoidable.

METHODS: We quantified this prior belief in 521 nonclinical participants. Then we used Bayesian network analysis to explore how the prior's mean was related to total scores of the Patient Health Questionnaire-9 (PHQ-9) depression scale, specific items of the PHQ-9, and 6 subscales measuring apathy, motivation, anhedonia, and emotional reactivity.

RESULTS: We found that the mean of the prior belief was positively related to the tendency to get motivated to initiate and maintain goal-directed actions as measured with the Apathy Motivation Index (AMI) and not hedonic capacity as measured by the Snaith-Hamilton Pleasure Scale. Moreover, the same reverse-coded behavioral apathy subscale in the AMI was related to the total score of the PHQ-9 independently of hedonic capacity. Finally, the prior belief itself was not directly linked to depressive symptoms or PHQ-9 total scores.

CONCLUSIONS: These results indicate that our behavioral measure of helplessness is indirectly related to depressive symptoms through behavioral activation and independently of hedonic capacity.

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Depression is one of the main causes of disability worldwide (1), but its clinical management is far from ideal (2–4), partly because the diagnostic system does not consider the neurocognitive systems believed to underlie the disorder (5–7). One possible path forward involves deep cognitive and behavioral phenotyping of patients to characterize the fundamental axes of variation of brain function (8–10).

Learned helplessness is a phenomenon used as a model for depression (11–15). It refers to the lack of active attempts to avoid negative outcomes in animals or humans who previously experienced uncontrollable aversive stimulation in another context (13,16). Learned helplessness thereby involves learning about the lack of control and generalizing it to new contexts where avoidance is possible. Beyond inhibition active avoidance, experiencing uncontrollable negative outcomes also impacts reward-seeking behaviors (17–19). Thus, repeated exposure to uncontrollable stressors can dampen the brain's reward system and lead to anhedonia (20–22), a cardinal symptom of depression referring to the inability to feel pleasure or interest in activities (23). As such, learned helplessness may play a key role in the development of reduced motivation and anhedonia.

Despite the importance of anhedonia in depression, there is no consensus on its mechanisms (24–26). This is reflected in the existence of a plethora of behavioral measures and scales [for review, see (26)]. Some measures capture the reduction in pleasure (liking) [e.g., (27)], while others capture deficits in motivation (wanting) [e.g., (28)], indexing apathy (29). Therefore, being able to quantify behavioral indices of helplessness would help to parcel out the variability in the expression of depression symptoms.

In a Bayesian framework, learned helplessness has been modeled as a generalization of a pessimistic prior belief for action/outcome contingencies (30,31), the prior belief being that of the probability of avoidance given an action. We recently developed a method to quantify this prior belief involving 2 new cognitive tasks and computational modeling (32). In both tasks, participants decide whether to attempt active avoidance actions, but the tasks differ in framing and valence, allowing us to test whether the prior belief is task independent and general (33–35). This method was validated using choice data from a community sample of online participants. Using extensive model comparisons, we showed that a Bayesian model with a task-invariant prior belief for active

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avoidance provided the best fit of participants' behavior. Moreover, we showed that the quantified prior belief was reliable over repeated measures. However, in that preregistered study, we only examined pairwise correlations between the prior belief and 2 general self-report measures. There, we found a weak correlation between the mean of the prior belief and positive affect, which was used as a proxy for depression because of its association with the condition (36–38).

Here, we extend the findings of our previous study in a larger sample by using network analysis to explore the relationship between the prior belief for active avoidance and self-reported measures of apathy, anhedonia, and depression symptoms. Network analysis provides a nuanced understanding of how any set of measured items interact and identifies the most interconnected items when considering all other items in the network (39–41). Furthermore, it creates a way to investigate the link between each pair of measures while controlling for all other included measures (partial correlation). This makes the network approach useful to inform hypotheses about how specific constructs or cognitive mechanisms interact with each other.

We are not aware of any previous research that has used network analysis in combination with computational measures and psychopathology. Instead, in previous studies, computational parameters are correlated with factors of covariation of symptoms derived from exploratory factor analysis (10,42–44). Compared with this approach, network analysis provides a richer description of how external factors interact with computational parameters by also considering how the factors interact with each other. Thus, network analysis provides an opportunity to investigate how behaviors (learned helplessness) relate to self-reported measures of apathy, hedonic capacity, and symptoms of depression. Here, we quantify the edge strengths and the nodes' predictability to measure how strongly related nodes are and the amount of variance of a specific node explained by all other nodes in the network, respectively.

In a community sample of participants, we aimed to characterize the relationship between our behavioral measure of helplessness and 1) a network of the symptoms that make up the DSM-5 diagnosis criteria for depression; 2) a network of several measures of apathy, appetitive and aversive motivation, emotional reactivity, and hedonic capacity; and 3) a network including both depressive symptoms and the measures of apathy and motivation.

METHODS AND MATERIALS

Participants

A community sample of 550 participants was recruited from the online participant database prolific.com. Participants were collected in 2 different sessions, where the blocking of the tasks differed slightly (see Procedure). For a full description of the samples and detailed characteristics, see Supplemental Methods and Table S1. When collapsed, the final sample consisted of 521 participants (47.8% female, 52.2% male; sample 1 = 273, sample 2 = 248) with a mean age of 27.10 (SD = 5.00) self-reporting 47 different nationalities. A post hoc sensitivity analysis carried out with G*power version 3.1.9.7 (45) showed that this sample size afforded us 80% power to detect correlations with effect sizes as small as $r = 0.108$ with an alpha level set at 0.05.

The study complies with all relevant ethical requirements and was approved by the Swedish Ethical Review Authority (ref. no.: 2021-04906). All participants provided informed consent before participating in the study. Participants were compensated with £10 if they agreed to participate and completed the experiment.

Procedure

To estimate a task-invariant prior belief for active avoidance, participants performed 2 blocks of 2 different binary choice tasks (the Robber task and the Factory task, collectively called the Variable ShuttleBox Generalization tasks), each block consisting of 60 trials. In these tasks [described in detail in (32)], participants decided between an active and passive option. If they chose the passive option, they would incur a negative consequence of varying size (the offer). The offer on each trial (o_t) was sampled from a set of numbers:

$$o_t \in \frac{50}{[0.10, 0.15, \ldots, 0.95, 1, 2]} \tag{1}$$

which was rounded to the nearest integer. All offers in this set were encountered 3 times within one block of the task in a randomized order. If they chose the active option instead, participants paid an action cost (cost) but had a varying probability of avoiding the negative outcome. The action cost was constructed as the sum of a percentage of the offer and a set cost (see Figure 1 for the details of tasks). In both tasks, this success probability was linked to the stimulus that participants encountered. In each block, participants encountered 12 different stimuli, each with a unique success probability ranging from 7.69% to 92.31%. Participants made 4 to 8 repeated decisions with the same stimulus. The order of the success probabilities was randomized for each participant. Crucially, participants were not told the success probability of each stimulus. Thus, to increase their success rate, participants had to learn the success probability associated with each stimulus from successive interactions with it, such that their learning reflected their prior belief over the distribution of success probabilities that they expected to encounter.

The structure of the blocks differed between the 2 samples. The first sample was the subject of analysis in Granwald *et al.* (32). The same tasks and a reformatted version of that model are used in the current article. However, the second sample, which was not analyzed in Granwald *et al.* (32), suffered a coding error that caused an unintended change in the task design. Instead of separating the blocks of the tasks (e.g., robber 1, factory 1, robber 2, factory 2), participants performed all the trials of one task before starting the next task without a break between the blocks of the task (e.g., robber 1, robber 2, factory 1, factory 2).

Questionnaires

Three different motivation, apathy, and anhedonia questionnaires and one depression questionnaire were used in this study. The scales included the Apathy Motivation Index (AMI),

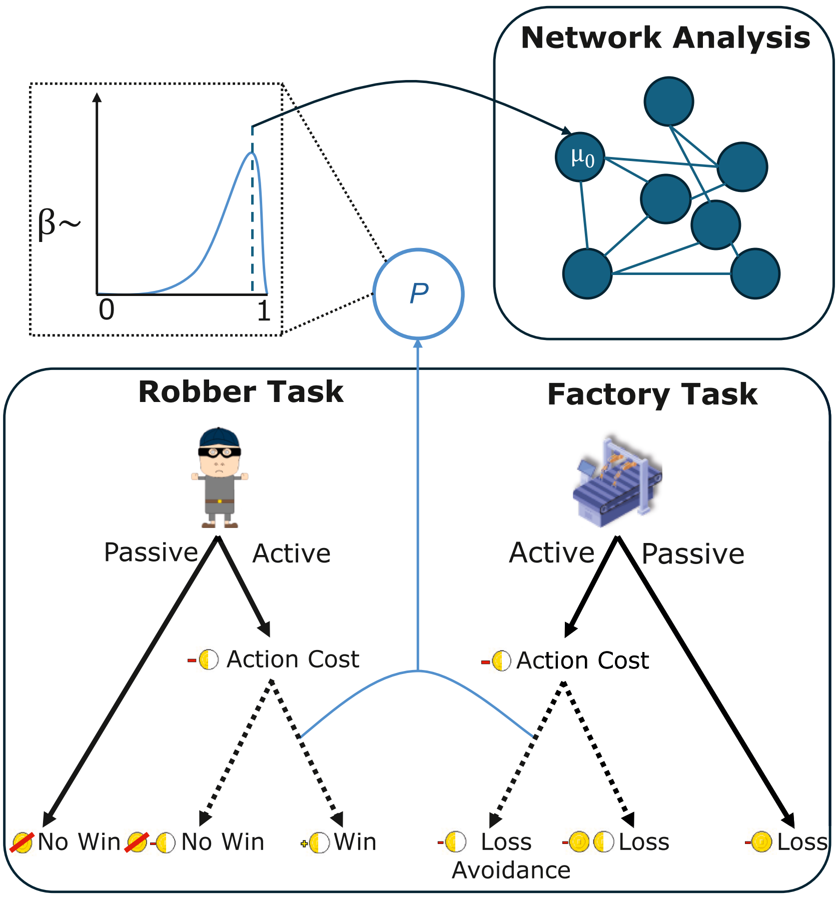

Figure 1. Task outline and method. The figure shows the schematic outline of the robber and factory tasks used to quantify participants' subjective prior beliefs about the probability of successfully avoiding a negative outcome given an action. The 2 tasks have different framing and outcome valence. In the robber task, the negative outcome participants are trying to avoid is missing out on a win. In this task, participants were asked to imagine that they had been working late as a server at a bar and collected the tips for the evening. While on the way home, they encounter a robber and decide whether to try to fight off the robber or relinquish their tip. When making these decisions, participants had to consider the cost of the active option, presented as losing their phone or ripping their shirt in the scuffle, as well as the amount of money they were carrying and could gain if they made it home safe (the offer). In this task, the set costs were 10, 40, or 75 points, and the associated percentages of the offers were 15%, 5%, and 0%. In the factory task, the negative outcome that participants are trying to avoid is a loss. In this task, participants were asked to imagine that they were working in a factory. They were the foreperson of this factory, and they made decisions about whether to try to repair machinery that is breaking down. When making these decisions, they had to consider the cost of the active option, which was presented as the cost to try to repair the machine and the cost that they would incur if the machine broke (the offer; $-o_t$ as described by equation 1). The action costs associated with the active option here were the sum of 0, 50, or 100 points and 10%, 5%, and 0% of the offer. The mean of prior belief, quantified with a Bayesian learner model, is used as a node in the network analysis to explore its relationship to multiple self-report measures of depression, apathy, and anhedonia.

the Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) scales, the Snaith-Hamilton Pleasure Scale (SHAPS), and the Patient Health Questionnaire-9 (PHQ-9). For the AMI subscales and the SHAPS, higher scores indicated lower levels of apathy and anhedonia, respectively. For the BIS/BAS, higher scores indicated higher levels of behavioral inhibition and behavioral activation, respectively. These questionnaires are reliable and valid in nonclinical samples (46–48) and are known to correlate with measures of depression [AMI, (46); BIS/BAS, (49,50); SHAPS, (47)]. For information about each scale, scoring, and reliability, see Supplemental Methods.

Statistical Analysis

Statistical analysis, including network analysis and correlations, was carried out in R version 4.3.0. Computational models were fitted in MATLAB (version 9.19.0.2105380, R2022b, The MathWorks, Inc.). Traditional inference criteria were used for the analyses ($p < .05$ and $BF_{10} > 3$) (see Supplemental Methods for inference criteria).

Computational Model. To estimate the participants' prior belief for active avoidance, we fitted a Bayesian learner model with 5 parameters to participants' choice behavior. The model was fitted across both blocks of both tasks and across tasks, in contrast to Granwald *et al.* (32), where the model was only fitted within blocks of the tasks. This model assumed a shared prior belief between the 2 tasks defined as a beta distribution. The model has previously been established as the best-fitting model for these tasks (32). For correlations between model agnostic task-relevant measures and self-report measures, see Supplemental Results and Table S8.

In this model, the prior belief is defined as a beta distribution with the parameters μ_0 and ρ_0, where $0 \le \mu_0 \le 1$ is the mean of the beta distribution, and $\rho_0 \ge 0$ is the relative precision of the distribution. Thus, on each trial t, the belief of the participant about the success probability associated with each stimulus j can be described as a beta distribution with stimulus-specific parameters $\mu_{t,j}$ and $\rho_{t,j}$ that are initialized to the respective parameters of the prior belief described above, and they are then updated on each trial when the corresponding stimulus was presented and the active option chosen. The focus of the current study is on μ_0, which directly impacts the utility function of both tasks (through $\mu_{t,j}$). Specifically, the value of the active option [Q(go)] on trial t and the value of the passive option [Q(ng)] are calculated as follows:

$$\text{Robber}: \begin{cases} Q_t(\text{go}) = -\,c_t + \mu_{t,j}\,o_t \\ Q_t(\text{ng}) = 0 \end{cases} \qquad (2)$$
$$\text{Factory}: \begin{cases} Q_t(\text{go}) = -c_t - (1-\mu_{t,j})o_t \\ Q_t(\text{ng}) = -o_t \end{cases}$$

Beyond the parameters of the prior belief, this model had 3 additional parameters, which were implemented in the choice function of the model. These included separate temperature parameters for the SoftMax choice function for each task and a bias/lapse parameter. For details on the updating of the $\mu_{t,j}$ parameter and the implementation of the other parameters, see Supplemental Methods.

Parameters were estimated with the hierarchical Bayesian inference algorithm implemented in the CBM toolbox version from 04/2019 (51) in MATLAB (for details, see Supplemental Methods). Parameters were fit with weakly informative zero-centered Gaussian priors with SD = 6.25. This is based on recommendations by the authors of the toolbox (51). Parameters were then transformed to their respective parametrization within the model.

We fitted the model separately for the 2 samples because the hierarchical structure of the model fitting would otherwise assume shared variance across the 2 samples. This is not guaranteed because of the difference in the block design between the 2 samples. The model fitted the data in both samples well and recovered comparable parameters of the prior belief across samples, and so for all subsequent analyses, the samples were combined (see Supplemental Methods for details).

We performed the same modeling analysis as in Granwald *et al.* (32) to both samples. Overall, the results were replicated, but the second sample was better fit with a 2-prior belief model when the 2 tasks and blocks were modeled together. However, the results below were also robust to fitting only the first block of each task in both samples, where the results from Granwald *et al.* (32) were fully replicated (see Supplemental Methods for the alternative analysis and Tables S2 and S3 for the replication in the full sample).

Network Analysis. Undirected partial correlation networks were estimated for different constellations of the items of the PHQ-9, the sum score of the PHQ-9, μ_0, and the sum scores of subscales of the apathy and anhedonia questionnaires. These capture the conditional relationships between each of these constructs.

Gaussian copula graphical models (GCGMs) (52) were used to estimate the networks. These models were estimated with an unregularized Bayesian approach for mixed data. This method estimates the posterior partial correlation (ρ) between each node in the network. The GCGM enabled treating the total scores of the scales and individual items as ordinal while treating the mu of the prior belief as continuous within the same network. This was implemented using the R package *BGGM* version 2.1.5 (53).

Matrix-F priors were used for the precision matrices used to estimate the partial correlations (54,55) with a hyperparameter delta value corresponding to an SD of 0.3 in a beta distribution multiplied by 2. This prior belief implies the approximate expected deviation from 0 that each partial correlation would have in an unconstrained model (55). The use of the matrix-F prior belief enabled testing of conditional dependency as well as independence by quantifying the evidence in favor of dependence or independence with a Bayes factor (BF), thereby providing exploratory hypothesis testing (55). The suggested threshold of $BF_{10} > 3$ has high specificity in determining the presence of edges and a low false inclusion rate (55,56). However, it suffers from low sensitivity (55,56). As a result, the absence of edges should be interpreted with caution when evidence is inconclusive. The networks were estimated with 20,000 posterior samples.

The networks were plotted with a force-embedded layout using the Fruchterman-Reingold layout algorithm (57) implemented in the R package *qgraph* version 1.9.8 (58) through the R package *SNA*.

We also estimated the predictability of each node in each network. This was calculated as Bayesian R^2 (59) with the method outlined in the study by Williams (60) through BGGM (53). This measure enables an estimation of the extent to which a node can be predicted by other nodes in the network and provides critical information about the practical usefulness of the edges connected to the node. The predictability was estimated with 20,000 posterior samples.

RESULTS

The Link Between the Prior Belief and Depression

We first correlated the mean of each participant's prior belief (μ_0) with their PHQ-9 total score. The PHQ-9 total score serves as a general measure of participants' depression symptomatology, where a higher score indicates a higher severity of depressive symptoms. There was no significant correlation between these measures (rs_{519} = 0.029, p = .51). Next, we investigated whether μ_0 was linked to specific symptoms by estimating the network structure of depressive symptoms and μ_0. This network (see Supplemental Results and Figure S4) did not detect any association between μ_0 and any individual symptom of depression (all $BF_{10} < 3$), indicating that when controlling for all other symptoms measured in the PHQ-9, there was no link between μ_0 and specific symptoms of depression.

The Link Between the Prior Belief, Apathy, and Anhedonia

Next, we explored how μ_0 correlated with self-rated motivation, apathy, and anhedonia (a total of 6 measures from AMI, BIS/BAS, and SHAPS scales). Table 1 reports the pairwise rank-order correlations between the subscales and μ_0. Only the correlation between μ_0 and the behavioral activation subscale (BA) of the AMI survived familywise error correction (rs_{519} = 0.121, p = .0056, Holm's adjusted p = .033). Here, a more optimistic prior belief correlated with participants' rating lower levels of behavioral apathy.

To further explore the interconnectedness between the different apathy and anhedonia constructs and their link to the prior belief, we estimated the network structure among all

Table 1. Correlations Between μ_0 of the Prior Belief and the Subscales of Anhedonia and Apathy

Scale	rs	*p* Value	Holm-Corrected *p* Value
BIS	0.021	.640	1
BAS	0.102	.020	.101
AMI: Emotional Sensitivity	0.016	.717	1
AMI: Social Motivation	−0.014	.746	1
AMI: Behavioral Activation	0.121	.0056	.0334
SHAPS	0.034	.435	1

AMI, Apathy Motivation Index; BAS, Behavioral Activation System; BIS, Behavioral Inhibition System; SHAPS, Snaith-Hamilton Pleasure Scale.

these measures. Here, we replicated the link between BA scores and μ_0 ($\rho = 0.135$, SD = 0.044, $BF_{10} = 12.9$), indicating that this link remained even when controlling for all other apathy and anhedonia measures (see Supplemental Results and Figure S5B for the full network).

The Link Between the Prior Belief, Anhedonia, Apathy, and Depression

To investigate how apathy and anhedonia relate to symptoms of depression, we estimated a network with the subscales of anhedonia, apathy, the sum score of the PHQ-9, and μ_0 (see Figure 2B and Table S14).

In this network, we replicated the link between μ_0 and BA scores ($\rho = 0.145$, SD = 0.043, $BF_{10} = 29.7$). We also saw evidence for dependence between depression severity and BA scores ($\rho = -0.212$, SD = 0.044, $BF_{10} = 7962$). However, the strongest edge between depression severity and the apathy and anhedonia scales was the negative edge from SHAPS ($\rho = -0.355$, SD = 0.040, $BF_{10} = 3.54 \times 10^{13}$), indicating strong dependence between hedonic capacity and depression severity. We also found evidence for a dependence between depression severity and BIS scores ($\rho = 0.258$, SD = 0.043, $BF_{10} = 2.06 \times 10^6$). Between these 3 nodes, we found a negative edge between BA and BIS scores ($\rho = -0.168$, SD = 0.044, $BF_{10} = 143$) but independence between BIS and SHAPS scores ($BF_{01} = 7.47$) and inconclusive evidence for an edge between BA and SHAPS scores, very weakly favoring dependence ($\rho = 0.099$, SD = 0.045, $BF_{10} = 1.441$). Together, this indicates that hedonic capacity, BA scores, and BIS scores independently explained the variance in depression severity, with the BIS and BA scores having opposing effects and being dependent on each other.

We also observed independence between depression severity and BAS scores ($BF_{01} = 7.51$), social motivation ($BF_{01} = 6.86$), and μ_0 ($BF_{01} = 3.165$), indicating that these measures were not directly related to depression severity. Lastly, there was inconclusive evidence for dependence between depression severity and emotional sensitivity, very weakly favoring dependence ($BF_{10} = 1.095$).

The node with the highest predictability in this network was depression severity (post $R^2 = 0.470$; 95% CI = [0.410, 0.532]) followed by the BA score (post $R^2 = 0.397$; 95% CI = [0.340, 0.457]). The node with the lowest predictability was μ_0 (post $R^2 = 0.051$; 95% CI = [0.021, 0.087]). See Figure 2A and Table S15 for the predictability of all nodes.

DISCUSSION

Our data show that the mean of the prior belief for active avoidance, a behavioral index of helplessness, is neither related to the severity of depression nor to any individual depressive symptom as measured with the PHQ-9 in a sample of participants without a self-reported psychiatric diagnosis. However, the data show that the mean of the prior belief is related to behavioral activation as measured with the AMI subscale BA. Finally, the network adding the total depression score to the motivational and apathy subscales confirmed the link between the mean of the prior belief for active avoidance and behavioral activation, which itself is linked to the total depression score along with hedonic capacity and the BIS scores.

The link between the prior belief and behavioral activation supports our interpretation that the prior belief is a behavioral measure of helplessness. This link is evident in both pairwise correlations and replicated across two networks. Because we reverse-coded the scale to ease the interpretation within the networks, the positive correlation between the prior belief and behavioral activation suggests that the passive behaviors observed in learned helplessness depend on this prior belief being pessimistic (32). It is also consistent with previous work by Hezemans *et al.* (61), where a prior estimate for action-outcome contingencies estimated in a single visuomotor task was selectively correlated with behavioral activation. This subscale has also been correlated with other behavioral

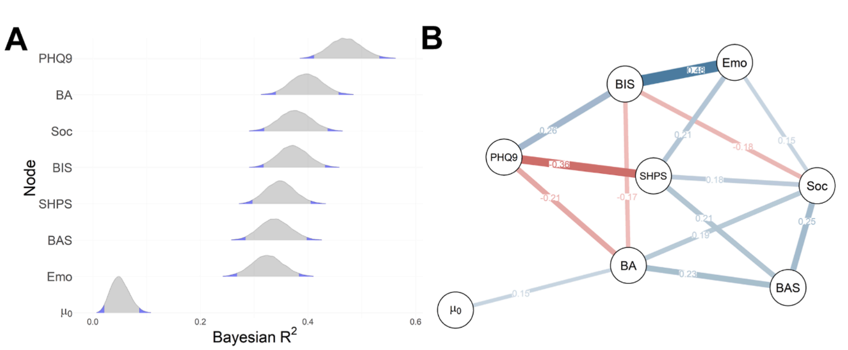

Figure 2. Network with apathy, anhedonia, and depression severity and an active avoidance prior belief. **(A)** Plot of the estimated predictability of each node as Bayesian R^2. The gray parts of the distributions signal the 95% credibility interval. **(B)** The Gaussian copula graphical network model estimated for the motivation subscales of the Behavioral Inhibition System (BIS)/Behavioral Activation System (BAS), apathy subscales of Apathy Motivation Index (AMI), and the hedonic capacity scale of the Snaith-Hamilton Pleasure Scale (SHAPS), as well as the total scores of the Patient Health Questionnaire-9 (PHQ-9) and the μ_0 of the active avoidance prior belief. The numbers shown are the mean posterior partial correlation for each edge. The color of the edge signals the direction of the partial correlation, and the thickness indicates the strength of the association. The network is plotted with the repulse radius at 8^3 and the cooling exponent set at 1.9 to improve legibility of all edges. BA, behavioral activation subscale (of the AMI); Emo, emotional sensitivity; Soc, social motivation.

measures, such as higher physical (62) and cognitive (63) effort discounting, concepts closely related to vigor (64,65).

The lack of association between the prior belief and the total depression score or any individual symptoms of depression was surprising considering the tight theoretical link between helplessness and depression (11,15,66). These negative results could stem from a floor effect and lack of variability in the levels of depressive symptoms in our sample, which was a community sample without explicit self-report of psychiatric diagnoses. However, the motivation network including the total depression score shows an indirect link between the mean of the prior belief and depression via behavioral activation. In this network, 3 nodes are directly connected to total depression scores—hedonic capacity as measured with the SHAPS scale, AMI BA, and BIS—with hedonic capacity showing the strongest connection.

The strong link between depression and hedonic capacity is unsurprising because the SHAPS scale was designed to identify reductions in hedonic tone in psychiatric conditions such as depression (27) and is concordant with the notion that anhedonia is central to depression (20,23). Based on several small studies showing preserved pleasantness ratings in response to sucrose solutions (67,68), it has been argued that depression is not related to deficits in hedonic sensitivity to rewarding outcomes (24). Instead, it has been suggested that the deficits in hedonic capacity reflect disrupted dopamine-dependent reinforcement learning leading to attenuated anticipation of the value of actions (20,24). Interestingly, the items of the SHAPS intend to quantify the ability to enjoy certain activities (i.e., consuming food, meeting friends) but likely measure the ability to anticipate them.

As expected, AMI BA and BIS are anticorrelated and are negatively and positively associated with the total depression score, respectively (69–71). Because network analysis builds on partial correlations, our results suggest that hedonic capacity, behavioral activation, and behavioral inhibition independently contribute to total depression scores. Instead, helplessness and anhedonia may independently contribute to depression severity through potentially distinct mechanisms, which may account for the heterogeneity in treatment outcomes observed in depression (2).

Limitations

A central limitation of this study is the assessment of motivation with questionnaires, which rely on self-report and may not fully capture the nature of motivation. While self-report measures provide valuable insights into subjective experience, they lack specificity when quantifying behavioral and cognitive processes (26,72) underlying psychopathology (73). This relates to a more general limitation in psychiatry, namely the diagnostic system as constellations of self-reported symptoms (23,74). The DSM diagnostic criteria for depression are known to be imperfect (7,75–77). For example, the symptoms included as criteria are not the most central symptoms across patients (75) [although see (78) for issues with centrality]. Similarly, transcultural studies have shown that highly frequent symptoms are omitted from the diagnosis criteria (79). Therefore, it is likely that some critical aspects of the depression symptomatology are not captured by the PHQ-9, since this scale only consists of items from the DSM-IV diagnosis criteria (80). Because of this heterogeneity in symptoms and descriptions of depression, we cannot make strong conclusions from the lack of association between the prior belief and depression. However, our results provide evidence that they are not directly related but indirectly linked through the tendency to engage in goal-directed actions.

Considering these limitations, our network analyses should be seen as an attempt at understanding how the prior belief relates to established motivation indices and depression symptoms. To more precisely target core motivational constructs underlying psychopathology in depression, future research should focus on multiple measures inferred from observed behavior such as the prior belief. Examples of these could be measures of effort (28,81), the ability to anticipate rewards and punishments (82–84), and the hedonic response when consuming rewards (67,68). Integrating multiple neurobehavioral measures would enhance our understanding of how prior beliefs for active avoidance influence motivation and relate to broader constructs such as reward responsiveness, learning, cost-sensitivity, and vigor. This approach would be consistent with the Research Domain Criteria (8) and computational psychiatry program (9,85).

Finally, we acknowledge that this study was not preregistered and is exploratory. While the findings offer valuable insights, they should be interpreted with caution and need confirmation in future studies.

Conclusions

Our data show that our newly developed behavioral measure of helplessness is indirectly related to the expression of depressive symptoms through the tendency to engage in goal-directed actions and independently from hedonic capacity, suggesting that helplessness is not related to anhedonia.

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TG, FT, and MG-M contributed to conceptualization. TG contributed to investigation. MG-M contributed to funding acquisition and supervision. TG and MG-M wrote the original draft of the article. TG, FT, ML, PD, and MG-M contributed to methodology and reviewing and editing the article.

Anonymized data are openly available at https://doi.org/10.5281/zenodo.13745011. All code used to analyse the data and create the figures is openly available at OSF (https://osf.io/fhrc5/). Experiment code used to quantify the prior belief is openly available at https://github.com/Granwald/healthyPriors and OSF (https://doi.org/10.17605/OSF.IO/8GBSU).

The authors report no biomedical financial interests or potential conflicts of interest.

ARTICLE INFORMATION

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