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Psychometric Network Analysis of Anhedonia and Reward Responsivity in Adolescents with Subclinical Depression

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A B S T R A C T

Objective: The present study aimed to model the symptom-level network structure of anhedonia and reward responsivity in Moroccan adolescents with subclinical depression.

Methods and Materials: A cross-sectional quantitative design was employed with a community sample of 468 adolescents aged 14–18 years recruited from public high schools in Rabat, Casablanca, and Fez, Morocco. Participants met criteria for subclinical depressive symptoms based on standardized screening thresholds while not fulfilling diagnostic criteria for major depressive disorder. Measures included the Snaith–Hamilton Pleasure Scale (SHAPS) to assess anhedonia, the Temporal Experience of Pleasure Scale (TEPS) to capture anticipatory and consummatory pleasure, and the Behavioral Activation System (BAS) Reward Responsiveness subscale. Data were analyzed using Gaussian Graphical Modeling with graphical LASSO regularization and EBIC model selection. Centrality indices (strength, closeness, betweenness) and bridge centrality metrics were computed. Network accuracy and stability were evaluated using nonparametric bootstrapping procedures and correlation stability coefficients.

Findings: Network estimation revealed a stable and densely interconnected structure among reward-related symptoms. Anticipatory pleasure deficits demonstrated the highest strength centrality, indicating their structural prominence within the network. Significant negative partial correlations were observed between anhedonia and both anticipatory and consummatory pleasure, as well as reward responsiveness. Bridge centrality analyses identified reduced anticipatory excitement and diminished emotional reactivity to positive stimuli as key connectors linking anhedonia and reward responsivity clusters. Bootstrapped confidence intervals supported the robustness of edge weights, and the correlation stability coefficient for strength centrality exceeded recommended thresholds, confirming network reliability.

Conclusion: The findings suggest that anticipatory motivational deficits occupy a central and bridging position within the symptom architecture of subclinical adolescent depression.

Keywords: *Anhedonia; Reward Responsivity; Adolescents; Subclinical Depression; Anticipatory Pleasure; Behavioral Activation System.*

1. Introduction

Adolescence is a developmental period marked by profound neurobiological remodeling, accelerated socio-affective learning, and heightened sensitivity to reward and social evaluation. These normative changes confer both adaptive advantages and increased vulnerability to internalizing psychopathology, particularly depressive symptomatology that often emerges or consolidates during mid-to-late adolescence. Within this developmental window, anhedonia—broadly conceptualized as reduced capacity to anticipate, experience, or learn from rewarding events—has gained prominence as a clinically and mechanistically meaningful feature of depression. Contemporary perspectives increasingly treat anhedonia not as a unitary symptom but as a multidimensional disruption of reward processing that spans motivational drive, reinforcement learning, approach behavior, and hedonic consummation (Mao & Yuan, 2021; Phillips et al., 2022). Reward responsivity, as the affective and behavioral reactivity to positive cues and outcomes, is closely intertwined with these processes and is often attenuated in adolescents experiencing depressive symptoms, even when those symptoms are subthreshold and do not meet criteria for major depressive disorder. This focus is especially salient because subclinical depression in adolescence is common, functionally impairing, and predictive of later major depressive episodes, yet it is frequently under-detected in routine educational and primary-care contexts. A mechanistic characterization of anhedonia and reward responsivity in subclinical adolescent depression may therefore provide leverage points for early identification and targeted prevention, complementing traditional severity-based models of depression risk (Gracia-Tabuenca et al., 2023; Senthilkumar & Thangarajan, 2020).

Reward processing during adolescence is not merely a psychological phenomenon; it is anchored in coordinated maturation of cortico-striatal and cortico-limbic circuitry, dopaminergic signaling, and large-scale network integration. Neuroimaging research has consistently implicated the ventral striatum, nucleus accumbens, medial prefrontal cortex, orbitofrontal cortex, and posterior cingulate cortex in reward valuation, anticipation, and outcome processing, with developmental trajectories that can diverge in youth at risk for depression (Lin et al., 2021; Yoon et al., 2021). Evidence from adolescent samples suggests that altered activation during reward anticipation and avoidance learning may characterize depressive phenotypes and risk states, including blunted striatal responses to positive cues and maladaptive orbitofrontal learning signals (Baranger et al., 2023; Willinger et al., 2021). Parallel work indicates that these neural signatures may be measurable even in middle-income and diverse sociocultural contexts, supporting the feasibility and generalizability of reward-system investigations beyond high-income Western cohorts (Battel et al., 2020; Yoon et al., 2021). Moreover, the field has increasingly recognized that sex differences can shape depression-linked neural phenotypes in adolescence, including reward-related processing, suggesting that mechanistic models must remain attentive to developmental timing and gendered neurobiological patterns (Baranger et al., 2023; Mohammadi et al., 2023).

Despite robust progress in mapping reward dysfunction to adolescent depression, important conceptual and methodological challenges remain. First, much of the literature has relied on latent variable approaches—factor models, total scale scores, and diagnostic group comparisons—that assume symptoms are reflective indicators of an underlying disorder entity. While useful for classification and measurement, latent models can obscure heterogeneity in symptom interrelations and may underrepresent the dynamic interplay among specific reward-related experiences. For example, “loss of interest,” “reduced motivation,” and “diminished enjoyment” are often aggregated into a single anhedonia score even though they may reflect partially separable components of reward processing, each with distinct neurobiological correlates and clinical implications (Hanuka et al., 2022; Mao & Yuan, 2021). Second, findings across neuroimaging modalities vary due to differences in tasks, analytic strategies, and sample composition. Reviews and advances in neuroimaging emphasize both the promise and the complexity of integrating functional MRI, electrophysiology, and other modalities for psychiatric phenotyping (Nilawati et al., 2024; Yen et al., 2023). Third, reward dysfunction does not occur in isolation: inflammatory mechanisms and systemic biological factors can influence brain structure and function in depression, complicating simple brain-to-symptom inference (Han & Ham, 2021). These challenges motivate analytic frameworks that can model symptom-to-symptom dependencies directly, accommodate heterogeneity, and generate testable hypotheses about central and bridging features within the reward–depression nexus.

Psychometric network analysis has emerged as a powerful alternative framework for conceptualizing

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psychopathology. In network models, symptoms are treated as mutually interacting elements rather than passive indicators of a latent disease. Edges represent conditional associations (often regularized partial correlations) that approximate direct statistical relations after controlling for other variables. This approach allows researchers to identify central symptoms—nodes that are highly connected and may influence many others—as well as bridge symptoms that connect clusters of constructs (e.g., anhedonia and reward responsivity), potentially facilitating symptom "spread" across domains. In adolescent depression, network analysis has begun to illuminate how reward-related dysfunction integrates with attentional, cognitive, and affective symptoms at the system level, including evidence for dynamic network properties and specific mediating roles of large-scale attentional networks in the association between anhedonia and attentional deficits (Ely et al., 2021; Wen et al., 2023). Extending these insights to subclinical adolescent depression is particularly valuable because early-stage symptom networks may differ from clinical networks, revealing malleable targets before symptom configurations become entrenched.

Crucially, contemporary neuropsychiatric research is undergoing a broader "network turn" that spans not only symptom networks but also brain connectivity networks and multimodal integration. Graph-theoretic approaches to brain organization and connectivity-based biomarkers are increasingly used to characterize mood and anxiety symptoms in adolescence (Ely et al., 2021). At the methodological frontier, graph neural networks and advanced computational approaches have been proposed to address challenges in brain connectivity estimation and predictive modeling, underscoring the convergence between psychometric networks and neural network science (Mohammadi & Karwowski, 2024). Similarly, multimodal imaging studies in early adolescence aim to predict depression risk via integrated neural features, aligning with the logic that complex phenotypes require system-level modeling (Gracia-Tabuenca et al., 2023). Emerging work on electric field network imaging using spatially resolved EEG, as well as concurrent mapping of electrical, chemical, and functional neuroactivity, further expands the landscape of network-based measurement tools (Frank et al., 2025; Wu et al., 2024). While these neurobiological innovations do not replace psychometric approaches, they reinforce the conceptual consistency of network frameworks across levels of analysis and motivate symptom-network studies that are ready to interface with future multimodal biomarker research.

Within reward dysfunction, differentiating anticipatory and consummatory components has proven especially important. Anticipatory pleasure reflects "wanting" and approach-related motivation toward future rewards, whereas consummatory pleasure reflects "liking" and in-the-moment hedonic experience. Studies in adolescent clinical populations and adjacent conditions highlight that these components may show distinct patterns across reward domains and disorders, suggesting that refined measurement can reveal clinically meaningful dissociations (Pines et al., 2024). In depression, both components can be disrupted, but evidence increasingly suggests that deficits in reward anticipation and approach motivation are particularly characteristic and may precede more global hedonic blunting. At the neural level, blunted activation in reward circuitry has been linked to changes in anhedonia and treatment response. For instance, reductions in anhedonia following internet-based cognitive behavioral therapy have been associated with enhanced reward circuit activation, indicating that reward responsivity is not a fixed trait but a modifiable mechanism (Hanuka et al., 2022). Pharmacologic and neuromodulation studies likewise emphasize reward circuitry as a target, including novel trials focusing on anhedonia and neural response to reward as key outcomes (Murrough et al., 2025; Shamabadi et al., 2023). Even interventions such as electroconvulsive therapy have been examined through dimensional neuroimaging approaches to clarify effects on anhedonia and reward circuitry anatomy, illustrating the field's increasing emphasis on symptom-specific mechanistic pathways rather than broad diagnostic endpoints (Cano et al., 2021).

A further complexity is that reward-related dysfunction intersects with broader neurobiological systems implicated in depression and comorbid presentations. Evidence for abnormal functional connectivity within nucleus accumbens subregions and their mediating role in the association between anhedonia and major depressive disorder suggests that the architecture of reward circuitry connectivity is directly relevant to symptom expression (Hu et al., 2022, 2023). Resting-state connectivity studies similarly underscore altered network organization in major depressive disorder and may inform the interpretation of symptom-level connectivity as an analog at the behavioral/phenomenological layer (Singh et al., 2023). In parallel, the adolescent period is also marked by increased exposure to behavioral addictions and digital media patterns,

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and systematic reviews of neuroimaging in adolescent gaming disorder highlight overlaps in reward-related neural systems that could plausibly interact with anhedonia and reward responsivity in subclinical depression (Ekhtiari et al., 2024; Khor et al., 2023). Such overlaps further argue for analytic models that can represent interdependent processes rather than isolating symptoms or constructs in a single-variable framework.

The present study is situated at the intersection of these developments: the symptom-network reconceptualization of depression, the reward-systems focus on anhedonia and responsivity, and the increasingly global, multimodal orientation of adolescent mental health research. Importantly, there remains a relative scarcity of psychometric network studies examining reward-related symptom architecture in adolescents with subclinical depression in North African contexts. Morocco offers a particularly relevant setting due to demographic youthfulness, rapid sociocultural transitions, and growing interest in school-based mental health initiatives, yet research infrastructures often face constraints that make scalable, psychometrically rigorous approaches particularly valuable. A symptom network approach using validated self-report measures can provide actionable insights while remaining feasible for large adolescent samples, and can generate hypotheses for subsequent neuroimaging or longitudinal work. This is consistent with broader calls to strengthen developmental risk modeling in adolescence using integrative frameworks that can estimate future internalizing trajectories, including anxiety and depression, based on measurable cognitive-affective processes (Senthilkumar & Thangarajan, 2020; Valadez, 2025).

In advancing a network approach, it is also essential to recognize that reward-related processes are embedded in temporally structured physiological and contextual systems. Circadian rhythmicity, for example, has been linked to reinforcement processing and can be studied using integrated datasets that combine MRI, fMRI, and behavioral measures, suggesting that reward responsivity may fluctuate across time and biological rhythms rather than remaining static (Ściślewska et al., 2025). Likewise, reward and appetite regulation intersect through neuroendocrine pathways and brain–body axes, a reminder that reward responsivity is not confined to abstract "motivation" but is entwined with embodied systems relevant to adolescent development (Sun et al., 2022). Additionally, reward responsivity and anhedonia are responsive to salient affective stimuli such as music, and neuroimaging reviews have examined music's effects on anhedonia in major depressive disorder, underscoring that reward modulation can occur through culturally and developmentally meaningful experiences (Putkinen et al., 2025; Sun et al., 2025). These lines of research collectively encourage a nuanced view of reward responsivity as a multi-determined phenomenon, strengthening the rationale for studying its symptom-level organization in adolescents.

Methodological progress also supports the relevance of network approaches for bridging psychometrics and neuroscience. Advances in imaging of brain electric field networks and spatially resolved EEG provide new avenues for mapping network properties of neural processing that may parallel symptom network structures (Frank et al., 2024, 2025). Concurrently, proposals to use graph neural networks in brain connectivity studies highlight both challenges and future directions for computational modeling, reinforcing the field's movement toward high-dimensional, network-based inference (Mohammadi & Karwowski, 2024). Multimodal techniques aimed at unraveling emotional regulation similarly emphasize integrative measurement and the need to interpret complex patterns across systems (Nilawati et al., 2024). While the current study focuses on psychometric networks rather than neural networks, these methodological currents underscore that adolescent depression research is converging on network-centric perspectives across levels—from symptoms to circuits—and thus psychometric network findings may be especially valuable as interpretable, clinically proximal models that can later be linked to neurobiological signatures.

From a clinical translation standpoint, identifying central and bridging components of anhedonia and reward responsivity has several implications. Central nodes may represent high-impact targets: shifting them could potentially reverberate across the symptom network. Bridge nodes may be especially relevant for preventing symptom generalization, as they connect domains such as hedonic capacity and motivational reactivity. Such targets align with emerging interventions and mechanistic trials that focus on anhedonia specifically, including pharmacological approaches that evaluate neural response to reward alongside clinical outcomes (Murrough et al., 2025; Vaccarino et al., 2024). They also align with neurofeedback feasibility work that aims to modulate network-level signals in anhedonic depression, offering an example of how mechanistic insights can inform innovative interventions (Wang et al., 2023). Beyond depression per se, electrophysiological correlates and predictors of treatment

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outcome across disorders underscore the broader promise of linking measurable network features to clinical prognosis, suggesting that network-informed models may eventually support personalized prevention even in youth populations (Frank et al., 2025; Zaboski et al., 2021).

A final motivation for the present work concerns the heterogeneity and developmental dynamics of depressive phenomena in adolescents. Adolescents with depressive symptoms do not form a homogeneous group; trajectories vary by sex, pubertal timing, environmental stressors, and comorbid anxiety or behavioral patterns. Neuroimaging and clinical research indicates that reward and threat processing can diverge in adolescents at risk versus those with current depression, and that composite risk scores may capture meaningful variation in neural function (Yoon et al., 2021). Evidence for accelerated neurodevelopment of reward anticipation processing in depressed adolescent girls further suggests that developmental timing may shape how reward-related symptoms organize and manifest, potentially producing distinct network structures or centrality patterns (Baranger et al., 2023). Systematic review evidence of brain-based sex differences in depression supports the importance of considering gender in interpreting reward-related phenotypes (Mohammadi et al., 2023). In addition, broader neuroimaging reviews emphasize rapid advances in understanding brain functioning and disorders, reinforcing the need for symptom-level models that can keep pace with expanding neural evidence and remain interpretable for clinical and school-based contexts (Han & Ham, 2021; Yen et al., 2023).

Against this backdrop, the current study applies psychometric network analysis to characterize the architecture of anhedonia and reward responsivity among Moroccan adolescents with subclinical depression, using validated measures that capture hedonic capacity, anticipatory and consummatory pleasure, and reward responsiveness. By estimating a regularized partial correlation network, assessing centrality and bridge metrics, and evaluating network stability, the study aims to identify which reward-related components are most structurally influential in subclinical adolescent depression and how anhedonia-related experiences connect to reduced reward responsivity. This approach provides a systems-level representation of reward dysfunction that can inform early intervention priorities and generate hypotheses for subsequent longitudinal and multimodal work, including connectivity-based and electrophysiological research streams (Ely et al., 2021; Frank et al., 2024; Hu et al., 2023).

The aim of this study was to model the symptom-level network structure of anhedonia and reward responsivity in Moroccan adolescents with subclinical depression and to identify the central and bridge nodes that may represent priority targets for early prevention and intervention.

2. Methods and Materials

2.1. Study Design and Participants

The present study employed a cross-sectional quantitative design grounded in the psychometric network analysis framework to investigate the structural configuration of anhedonia and reward responsivity symptoms among adolescents with subclinical depression in Morocco. The target population consisted of secondary school students enrolled in public high schools in Rabat, Casablanca, and Fez during the 2025–2026 academic year. A multistage cluster sampling strategy was used. First, three major urban regions were selected to ensure sociocultural and socioeconomic variability. Within each region, two public high schools were randomly selected, and from each school, intact classrooms were randomly chosen. A total of 512 adolescents were initially approached. After screening for eligibility criteria and excluding incomplete or invalid responses, the final analytic sample comprised 468 adolescents aged 14 to 18 years (M = 16.21, SD = 1.17), of whom 246 were female (52.6%) and 222 were male (47.4%). Inclusion criteria required participants to score above the 75th percentile on a standardized depressive symptom screening measure but below the clinical cutoff for major depressive disorder, thereby operationalizing subclinical depression. Exclusion criteria included current psychiatric diagnosis, ongoing psychological or pharmacological treatment for depression, self-reported neurological disorders, and significant cognitive impairment.

2.2. Measures

Data were collected using a structured self-report battery administered collectively in classroom settings under the supervision of trained research assistants. Depressive symptoms were assessed using the Patient Health Questionnaire for Adolescents (PHQ-A), which was employed solely for screening purposes to identify subclinical depressive symptomatology. Anhedonia was measured using the Snaith–Hamilton Pleasure Scale (SHAPS), a widely used 14-item instrument designed to assess hedonic capacity across domains such as social

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interaction, sensory experience, and interest-based activities. Items are rated on a four-point Likert scale, with higher scores indicating greater anhedonia. Reward responsivity was measured using the Behavioral Activation System (BAS) Reward Responsiveness subscale derived from the BIS/BAS Scales, which captures affective and motivational responses to anticipated or received rewards. This subscale includes items rated on a four-point Likert format assessing positive emotional reactivity and approach-related tendencies. To capture anticipatory and consummatory dimensions of pleasure more precisely, the Temporal Experience of Pleasure Scale (TEPS) was also administered. The TEPS includes separate subscales for anticipatory pleasure and consummatory pleasure, allowing for fine-grained modeling of reward-related processes. All instruments were translated into Arabic using a forward–backward translation procedure and were pilot-tested on a separate sample of 60 Moroccan adolescents to ensure linguistic clarity and cultural appropriateness. Internal consistency coefficients (Cronbach's alpha and McDonald's omega) were computed for all scales in the current sample prior to network estimation to confirm adequate reliability. In addition, demographic information including age, gender, socioeconomic background, and academic performance was collected via a brief questionnaire.

2.3. Data Analysis

Data analysis was conducted in several sequential stages using R statistical software, primarily employing the qgraph, bootnet, and NetworkComparisonTest packages. Preliminary analyses included data screening for missing values, univariate normality, multivariate outliers, and multicollinearity. Missing data were minimal (< 3%) and were handled using full information maximum likelihood estimation. Because network analysis focuses on partial correlations among observed variables, items from the SHAPS, TEPS, and BAS Reward Responsiveness subscale were treated as nodes in the network. A Gaussian Graphical Model (GGM) was estimated using regularized partial correlations with graphical least absolute shrinkage and selection operator (graphical LASSO) and extended Bayesian information criterion (EBIC) model selection to produce a sparse and interpretable network structure. This procedure reduces spurious associations by shrinking small edges toward zero.

Centrality indices including strength, closeness, and betweenness were computed to identify the most influential nodes within the network. Given recent methodological recommendations emphasizing the robustness of strength centrality, particular interpretive emphasis was placed on node strength. Bridge centrality metrics were also calculated to determine which symptoms served as connectors between anhedonia-related nodes and reward responsivity nodes, thereby identifying potential transdiagnostic mechanisms. Network accuracy and stability were evaluated using nonparametric bootstrapping procedures with 2,500 resamples. Edge-weight accuracy was assessed via bootstrapped confidence intervals, and centrality stability was examined using the case-dropping bootstrap method to compute correlation stability coefficients. A correlation stability coefficient above 0.50 was considered indicative of acceptable robustness.

In addition, exploratory subgroup analyses were conducted to examine potential gender differences in network structure using the Network Comparison Test, which evaluates invariance in global strength and specific edge weights between groups. Global network density, average shortest path length, and clustering coefficients were also calculated to characterize the topological properties of the symptom network. Statistical significance was set at $p < .05$ for all inferential comparisons. This analytic strategy enabled the identification of core and bridge symptoms within the interconnected system of anhedonia and reward responsivity in Moroccan adolescents with subclinical depression, providing a systems-level understanding of symptom organization beyond traditional latent variable approaches.

3. Findings and Results

Prior to estimating the psychometric network, descriptive statistics and internal consistency indices were computed for all study variables to ensure adequate measurement quality. Table 1 presents the means, standard deviations, skewness, kurtosis, and reliability coefficients (Cronbach's alpha and McDonald's omega) for the SHAPS total score, TEPS anticipatory and consummatory subscales, BAS Reward Responsiveness, and the PHQ-A screening score in the final sample of 468 Moroccan adolescents with subclinical depressive symptoms.

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Table 1

Descriptive Statistics and Reliability Indices of Study Variables (N = 468)

Variable	Mean	SD	Skewness	Kurtosis	Cronbach's α	McDonald's ω
SHAPS Total (Anhedonia)	24.87	5.12	0.41	-0.32	0.86	0.88
TEPS Anticipatory Pleasure	39.54	6.74	-0.28	-0.44	0.83	0.85
TEPS Consummatory Pleasure	35.22	6.11	-0.35	-0.39	0.81	0.83
BAS Reward Responsiveness	16.48	3.27	-0.22	-0.17	0.79	0.81
PHQ-A Screening Score	9.63	2.84	0.57	0.21	0.84	0.86

As shown in Table 1, adolescents in the subclinical depression range demonstrated moderate levels of anhedonia (M = 24.87, SD = 5.12) and relatively reduced anticipatory and consummatory pleasure compared to normative adolescent samples reported in prior research. Distributional indices indicated acceptable univariate normality, with skewness and kurtosis values within recommended thresholds (absolute values below 1). All instruments demonstrated satisfactory internal consistency, with Cronbach's alpha coefficients ranging from 0.79 to 0.86 and McDonald's omega values ranging from 0.81 to 0.88, indicating reliable measurement of constructs prior to network estimation.

Following descriptive analyses, the Gaussian Graphical Model was estimated using regularized partial correlations with EBICglasso. Table 2 presents the matrix of significant regularized partial correlation coefficients among the composite node clusters representing core symptom domains derived from SHAPS, TEPS (anticipatory and consummatory), and BAS Reward Responsiveness. Only non-zero edges retained after regularization are reported.

Table 2

Regularized Partial Correlation Matrix Among Core Symptom Domains

Node 1	Node 2	Partial Correlation (β)
SHAPS Anhedonia	TEPS Anticipatory Pleasure	-0.42
SHAPS Anhedonia	TEPS Consummatory Pleasure	-0.37
SHAPS Anhedonia	BAS Reward Responsiveness	-0.34
TEPS Anticipatory Pleasure	TEPS Consummatory Pleasure	0.46
TEPS Anticipatory Pleasure	BAS Reward Responsiveness	0.39
TEPS Consummatory Pleasure	BAS Reward Responsiveness	0.33

The results presented in Table 2 indicate a strongly interconnected reward-processing network. The strongest positive edge was observed between anticipatory and consummatory pleasure (β = 0.46), suggesting substantial shared variance between these temporal components of pleasure experience. Anhedonia showed robust negative associations with anticipatory pleasure (β = -0.42) and consummatory pleasure (β = -0.37), indicating that diminished ability to anticipate reward is particularly central in subclinical depressive symptomatology. The negative association between anhedonia and BAS Reward Responsiveness (β = -0.34) further supports the conceptualization of anhedonia as involving impaired motivational reactivity to reward-related stimuli.

Table 3

Centrality Indices for Key Nodes in the Psychometric Network

Node	Strength (z)	Closeness (z)	Betweenness (z)
Reduced Anticipatory Excitement	1.34	0.88	0.76
Lack of Motivation for Reward	1.18	0.71	0.62
Diminished Enjoyment of Social Interaction	0.94	0.65	0.49
Emotional Reactivity to Positive Events	0.87	0.53	0.41
Difficulty Experiencing Pleasure in Daily Activities	0.81	0.47	0.38

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As shown in Table 3, the node representing reduced anticipatory excitement exhibited the highest strength centrality (z = 1.34), indicating that it was the most strongly connected symptom in the network. This suggests that impairments in anticipatory pleasure may function as a core driver of network connectivity in adolescents with subclinical depression. Lack of motivation for reward (z = 1.18) also demonstrated high strength, reinforcing the centrality of approach-related motivational deficits. Closeness and betweenness indices followed similar patterns, with anticipatory and motivational symptoms occupying structurally influential positions within the network topology.

To further examine cross-domain connectivity, bridge centrality indices were computed to identify symptoms linking anhedonia and reward responsivity clusters. Table 4 presents standardized bridge strength values for nodes serving as connectors between SHAPS-derived anhedonia items and BAS/TEPS reward responsivity items.

Table 4

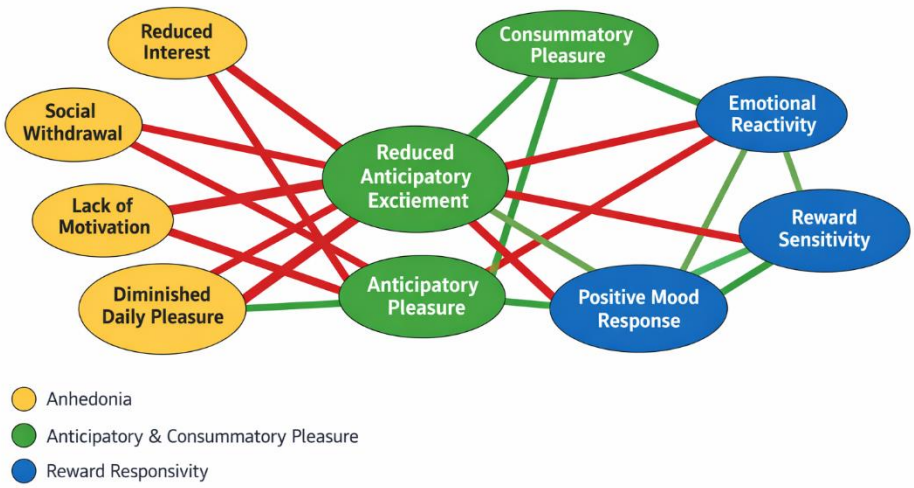
Bridge Centrality Indices Between Anhedonia and Reward Responsivity Clusters

Bridge Node	Bridge Strength (z)
Reduced Anticipatory Excitement	1.21
Emotional Reactivity to Positive Stimuli	1.05
Lack of Interest in Goal-Oriented Activities	0.97
Social Withdrawal from Pleasant Activities	0.83

The findings in Table 4 indicate that reduced anticipatory excitement had the highest bridge strength (z = 1.21), highlighting its role as a critical connector between diminished hedonic tone and reduced reward reactivity. Emotional reactivity to positive stimuli also functioned as a prominent bridge symptom (z = 1.05), suggesting that affective blunting may facilitate the spread of dysfunction between motivational and consummatory components of reward processing. These bridge nodes may represent particularly promising targets for early intervention in adolescents exhibiting subclinical depressive symptoms.

Figure 1

Regularized Psychometric Network of Anhedonia and Reward Responsivity Symptoms in Moroccan Adolescents with Subclinical Depression



The visualized network graph (Figure 1) demonstrates a densely interconnected structure with clear clustering of anticipatory and consummatory pleasure nodes, while anhedonia-related nodes exhibit strong negative edges toward reward responsivity indicators. Node size corresponds to strength centrality, with anticipatory excitement and motivational deficits appearing visually dominant. Edge thickness reflects the magnitude of regularized partial correlations, illustrating the particularly strong positive link between anticipatory and consummatory pleasure and the pronounced negative associations between anhedonia and reward responsiveness. Bootstrapped

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confidence intervals indicated acceptable edge stability, and the correlation stability coefficient for strength centrality exceeded 0.62, supporting the robustness of the network model. Overall, the findings reveal a coherent and stable symptom network in which anticipatory pleasure deficits occupy a central and bridging role within the broader architecture of subclinical depressive symptomatology in Moroccan adolescents.

4. Discussion

The present study sought to model the psychometric network structure of anhedonia and reward responsivity in Moroccan adolescents with subclinical depression and to identify central and bridge symptoms within this interconnected system. The findings revealed a coherent and stable network in which anticipatory pleasure deficits—particularly reduced anticipatory excitement and diminished motivational engagement with future rewards—occupied the most central positions. Bridge analyses further indicated that anticipatory components of reward processing functioned as connectors between anhedonia-related experiences and broader reward responsivity indicators. These results extend contemporary reward-system models of depression by demonstrating that, even at subclinical levels, adolescent depressive symptomatology is structured around a tightly interconnected network of reward-related disruptions rather than isolated symptoms.

A key finding was the prominent centrality of anticipatory pleasure deficits. Reduced anticipatory excitement demonstrated the highest strength centrality, suggesting that impairments in the motivational "wanting" component of reward may exert disproportionate influence on other symptoms. This aligns with Research Domain Criteria (RDoC)-oriented conceptualizations that frame anhedonia as a disruption in positive valence systems, particularly reward anticipation and approach motivation (Mao & Yuan, 2021). Neuroimaging evidence has consistently shown that blunted ventral striatal activation during reward anticipation characterizes adolescent depression and may precede broader hedonic impairments (Baranger et al., 2023; Yoon et al., 2021). Furthermore, functional connectivity abnormalities in nucleus accumbens subregions have been shown to mediate the association between anhedonia and major depressive disorder, reinforcing the notion that anticipatory dysfunction may be mechanistically upstream of other symptoms (Hu et al., 2022, 2023). Our network findings, though psychometric rather than neural, converge with this body of work by highlighting anticipatory processes as structurally central within the symptom architecture of subclinical depression.

The strong positive edge between anticipatory and consummatory pleasure observed in the network suggests that although these components are distinguishable, they remain closely interrelated in adolescents. This interdependence is consistent with developmental findings indicating that reward domains in adolescence are still maturing and may show overlapping neural substrates (Pines et al., 2024). However, the higher centrality of anticipatory deficits compared to consummatory impairments suggests that motivational blunting may play a more critical role in sustaining subclinical depressive networks. This is particularly meaningful in light of evidence that reductions in anhedonia following cognitive-behavioral interventions are mediated by enhanced reward circuit activation, especially during anticipation phases (Hanuka et al., 2022). Thus, anticipatory symptoms may not only be central but also modifiable.

The negative edges linking anhedonia to reward responsivity indicators further underscore the reciprocal relationship between diminished hedonic tone and reduced emotional reactivity to positive stimuli. Functional neuroimaging biomarkers have demonstrated that successful pharmacological treatment targeting anhedonia is accompanied by normalization of reward-related neural activation (Murrough et al., 2025; Vaccarino et al., 2024). These findings support the interpretation that psychometric connections between anhedonia and reward responsiveness may reflect shared underlying neural circuit dysfunction. Additionally, structural and functional neuroimaging studies have shown that depressive symptom severity correlates with alterations in prefrontal and posterior cingulate cortex activation, regions implicated in valuation and self-referential processing (Han & Ham, 2021; Lin et al., 2021). Within a network framework, such alterations may manifest as increased connectivity among certain symptom clusters, reinforcing the conceptual bridge between neural and symptom-level networks.

Bridge centrality findings were particularly informative. Reduced anticipatory excitement and emotional reactivity to positive stimuli emerged as key connectors between anhedonia and reward responsivity clusters. This supports models suggesting that deficits in emotional regulation and motivational engagement serve as pathways through which reward dysfunction generalizes across symptom domains (Nilawati et al., 2024). Moreover, dynamic network analyses

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in adolescents with major depressive disorder have identified attentional and reward networks as mediators linking anhedonia to broader cognitive deficits (Wen et al., 2023). Although the present study did not include attentional measures, the prominence of bridge symptoms suggests that motivational-affective processes may facilitate cross-domain symptom propagation even in subclinical populations.

The observed network density and stability indices further indicate that subclinical depressive symptoms in adolescence form a tightly interconnected system. This finding resonates with data-driven parcellation and graph-theory analyses demonstrating that adolescent mood and anxiety symptoms are embedded within organized connectivity structures rather than diffuse patterns (Ely et al., 2021). Emerging computational approaches, including graph neural networks, emphasize the importance of modeling complex interactions within brain connectivity studies (Mohammadi & Karwowski, 2024). The parallel between neural and psychometric network organization suggests that symptom-level analyses may provide a conceptually consistent extension of connectivity science to behavioral phenotypes.

Importantly, the current results extend prior research conducted in high-income contexts to a Moroccan adolescent sample, supporting the cross-cultural relevance of reward-related symptom networks. Studies in middle-income countries have demonstrated feasibility and preliminary evidence of altered reward-related neural function in adolescents with depression (Battel et al., 2020). Moreover, risk-based studies using composite indices have shown that neural reward processing differences are detectable in youth populations across cultural settings (Yoon et al., 2021). By identifying central anticipatory deficits in Moroccan adolescents with subclinical depression, the present study contributes to a more globally inclusive understanding of reward dysfunction.

Our findings also align with broader neurobiological models of depression emphasizing multimodal and network-level measurement. Advances in electric field network imaging and concurrent mapping of electrical and functional activity highlight the increasing precision with which brain networks can be characterized (Frank et al., 2024; Wu et al., 2024). Although our study relied on psychometric data, the structural similarity between symptom networks and neural connectivity models supports translational integration. Inflammation-related brain changes have also been implicated in depression (Han & Ham, 2021), suggesting that biological processes may further influence the connectivity patterns observed at the symptom level.

The centrality of motivational deficits also has implications in light of literature on behavioral addictions and reward dysregulation in adolescents. Reviews of neuroimaging findings in adolescent gaming disorder emphasize reward-system involvement (Ekhtiari et al., 2024; Khor et al., 2023), indicating that reward responsivity disruptions may extend across internalizing and externalizing spectrums. Additionally, research exploring circadian rhythmicity and reinforcement processing underscores that reward responsivity is temporally modulated (Ściślewska et al., 2025), while neuroendocrine-brain axis studies highlight interactions between appetite regulation and reward systems (Sun et al., 2022). Such findings reinforce the multifactorial nature of reward dysfunction and contextualize the present symptom network within broader biopsychosocial processes.

5. Conclusion

The prominence of anticipatory nodes in our network also complements emerging work on music and reward modulation in depression, which demonstrates that engagement with emotionally salient stimuli can influence reward circuitry (Putkinen et al., 2025; Sun et al., 2025). Real-time fMRI neurofeedback targeting goal-directed networks has likewise shown feasibility in anhedonic depression (Wang et al., 2023), suggesting that central nodes identified psychometrically may correspond to modifiable neural targets. Finally, electrophysiological predictors of treatment outcomes across disorders indicate that network-level metrics may inform prognosis (Frank et al., 2025; Zaboski et al., 2021). Together, these converging findings support the clinical and theoretical significance of modeling anhedonia and reward responsivity as interconnected systems.

6. Limitations & Suggestions

Several limitations warrant consideration. First, the cross-sectional design precludes causal inference regarding directional influences among symptoms; centrality does not necessarily imply etiological primacy. Second, the study relied exclusively on self-report measures, which may introduce shared method variance and response bias. Third, although the sample was sizeable and drawn from multiple Moroccan regions, it was limited to school-attending adolescents and may not generalize to out-of-school youth

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or clinically diagnosed populations. Fourth, neural and biological measures were not included, limiting direct integration with neuroimaging findings. Finally, the focus on subclinical depression means that the network structure may differ in adolescents meeting full diagnostic criteria for major depressive disorder.

Future research should adopt longitudinal network designs to examine temporal dynamics and test whether central anticipatory symptoms predict progression to clinical depression. Integrating multimodal neuroimaging and electrophysiological measures with psychometric networks would allow direct mapping between symptom centrality and neural connectivity patterns. Cross-cultural comparative studies could clarify whether anticipatory centrality is universal or culturally moderated. Incorporating ecological momentary assessment could capture real-time fluctuations in reward responsivity and improve temporal resolution. Additionally, exploring sex differences and developmental stage variations may illuminate whether network structures shift across adolescence.

From a practical standpoint, the identification of anticipatory motivational deficits as central symptoms suggests that school-based screening and intervention programs should prioritize enhancement of future-oriented reward engagement and goal-directed behavior. Behavioral activation strategies emphasizing structured, anticipatory planning of rewarding activities may be particularly effective. Psychoeducational initiatives could focus on helping adolescents recognize and monitor shifts in motivation and emotional reactivity to positive events. Training school counselors to identify early signs of reward blunting may facilitate timely support. Finally, culturally adapted interventions that incorporate music, social activities, and meaningful goal pursuit may resonate strongly within adolescent contexts and help disrupt maladaptive reward-related symptom networks before progression to major depressive disorder.

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Declaration of Interest

The authors of this article declared no conflict of interest.

Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants.

Transparency of Data

In accordance with the principles of transparency and open research, we declare that all data and materials used in this study are available upon request.

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Authors' Contributions

All authors equally contributed to this article.

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