

Irrational Beliefs, Health Promoting Behaviors, and Pain Perception in Patients With Irritable Bowel Syndrome: The Mediating Role of Behavioral Activation and Inhibition Systems

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ABSTRACT

Irritable bowel syndrome (IBS) is a chronic gastrointestinal disorder in which psychological factors play a significant role in symptom severity and pain perception. The present study aimed to examine the association between irrational beliefs, health promoting behaviors, and pain perception in patients with IBS, with the mediating roles of the Behavioral Activation System (BAS) and Behavioral Inhibition System (BIS). This cross sectional study was conducted among 320 patients diagnosed with IBS who were recruited using convenience sampling from a gastroenterology clinic. Participants completed the McGill Pain Questionnaire, the Health Promoting Lifestyle Profile (HPLP), the Irrational Beliefs Test, and the BIS/BAS scales. Data were analyzed using path analysis within the structural equation modeling framework in AMOS version 24. Path analysis revealed that health promoting behaviors were negatively associated with pain perception ($\beta = -.41, p < .001$), whereas irrational beliefs were positively associated with pain perception ($\beta = .36, p < .001$). Behavioral activation (BAS) showed a significant negative effect on pain perception ($\beta = -.31, p < .001$), while behavioral inhibition (BIS) demonstrated a significant positive effect ($\beta = .35, p < .001$). Health promoting behaviors were significantly related to BAS ($\beta = -.24, p < .001$) and BIS ($\beta = .27, p < .001$). Irrational beliefs were positively associated with BIS ($\beta = .30, p < .001$) but not with BAS ($\beta = -.09, p = .106$). Bootstrapping analyses indicated that BAS and BIS significantly mediated the relationship between health promoting behaviors and pain perception, whereas only BIS mediated the relationship between irrational beliefs and pain perception. The final model explained 50% of the variance in pain perception. These findings highlight the role of cognitive factors and behavioral self regulation systems in the experience of pain among patients with IBS. Interventions targeting irrational beliefs and promoting healthy behaviors may reduce pain perception by influencing motivational systems such as BAS and BIS.

Keywords: Irrational beliefs, Health promoting behaviors, Pain perception, Behavioral activation system, Behavioral inhibition system, Irritable bowel syndrome

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Introduction

Irritable Bowel Syndrome (IBS) is a prevalent functional gastrointestinal disorder characterized by chronic abdominal pain, altered bowel habits, bloating, and recurrent gastrointestinal discomfort in the

absence of identifiable structural abnormalities. Contemporary gastroenterological research increasingly conceptualizes IBS not merely as a peripheral gut disorder but as a complex disorder of the gut–brain axis, in which central nervous system processing, visceral hypersensitivity, and psychological factors interact dynamically to shape symptom expression and severity (1, 2). Neurobiological models emphasize dysregulation in pain processing circuits, including enhanced afferent signaling, altered emotional salience processing, and disrupted top–down inhibitory control, all of which contribute to heightened pain perception in IBS patients (1, 3). In this context, IBS shares substantial overlap with other chronic pain conditions in which pain is amplified by cognitive, emotional, and behavioral mechanisms rather than proportional to peripheral tissue damage (4, 5).

Pain perception in chronic conditions is increasingly understood as a multidimensional construct shaped by sensory input as well as cognitive appraisal, attentional bias, and emotional regulation processes. Empirical evidence suggests that pain is not a passive sensory experience but an active inferential process influenced by expectations, beliefs, and prior learning (6, 7). In IBS, visceral hypersensitivity and central sensitization mechanisms contribute to exaggerated pain responses to normal gastrointestinal stimuli, reinforcing the subjective intensity of symptoms (1, 8). Moreover, attentional biases toward internal bodily sensations and threat-related cues further amplify pain perception and distress, highlighting the cognitive penetrability of pain systems (9). This cognitive–affective amplification is particularly relevant in IBS, where symptom unpredictability increases vigilance and anticipatory anxiety.

Within this biopsychosocial framework, irrational beliefs represent a critical cognitive vulnerability factor influencing pain perception. Irrational beliefs—defined as rigid, absolutistic, and maladaptive thought patterns such as catastrophizing, overgeneralization, and low frustration tolerance—have been consistently linked to heightened psychological distress and symptom amplification in chronic conditions (10, 11). In IBS populations, maladaptive cognitive appraisals intensify symptom monitoring and exacerbate emotional reactivity, thereby increasing perceived pain severity (12). Cognitive theories of pain suggest that such beliefs distort threat appraisal mechanisms, leading individuals to interpret benign visceral sensations as harmful or uncontrollable, which in turn triggers maladaptive coping responses (13, 14).

Experimental and clinical studies have further demonstrated that cognitive biases and irrational interpretations of bodily signals are strongly associated with increased pain interference and reduced cognitive flexibility (7, 15). These maladaptive cognitive patterns are also linked to attentional biases toward pain-related stimuli and heightened emotional distress, which together reinforce chronic symptom cycles (8, 9). Additionally, trauma-related cognitive processing has been identified as an important modifier of pain experiences, suggesting that irrational belief systems may be rooted in broader emotional and developmental vulnerabilities (16). Collectively, these findings position irrational beliefs as central cognitive determinants of pain perception in IBS.

Alongside cognitive factors, behavioral determinants—particularly health-promoting behaviors—play a protective role in modulating pain experiences. Health-promoting behaviors include physical activity, nutrition regulation, stress management, sleep hygiene, and health responsibility, all of which contribute to improved physiological regulation and psychological resilience (17). Evidence indicates that individuals who engage in consistent health-promoting lifestyles demonstrate lower pain intensity and improved quality of life even in chronic pain conditions (18, 19). Lifestyle-related factors are now considered central in the

perpetuation or mitigation of chronic pain syndromes, including IBS, through their effects on inflammation, autonomic regulation, and psychological well-being (20).

In IBS specifically, lifestyle interventions have been associated with improved symptom management and reduced visceral hypersensitivity, suggesting that behavioral regulation can influence gut–brain axis functioning (21, 22). Health-promoting behaviors may also enhance self-efficacy and perceived control, thereby reducing catastrophic interpretations of symptoms and strengthening adaptive coping mechanisms (23, 24). Furthermore, social support and interpersonal resources have been shown to mediate the relationship between health behaviors and psychological distress in chronic pain populations, emphasizing the broader psychosocial context of behavioral engagement (24, 25).

A growing body of research suggests that cognitive and behavioral factors do not operate in isolation but interact through underlying motivational systems. The Behavioral Activation System (BAS) and Behavioral Inhibition System (BIS) provide a neuropsychological framework for understanding how individuals regulate approach and avoidance behaviors in response to environmental and internal cues (26, 27). BAS is associated with reward sensitivity, goal-directed behavior, and approach motivation, whereas BIS reflects sensitivity to threat, punishment, and avoidance behavior. In chronic pain populations, dysregulation of these systems has been linked to maladaptive coping patterns and altered pain processing (26, 28).

Empirical evidence indicates that elevated BIS activity is associated with increased pain sensitivity, heightened anxiety, and avoidance behaviors that contribute to functional disability (27, 29). Conversely, stronger BAS functioning is associated with engagement in adaptive activities, greater resilience, and reduced pain interference (28, 30). These motivational systems are particularly relevant in IBS, where anticipatory anxiety and avoidance of gastrointestinal triggers often exacerbate symptom persistence and reduce quality of life (1, 8).

Importantly, BIS and BAS may serve as mediating mechanisms linking cognitive beliefs and health behaviors to pain perception. For instance, maladaptive cognitive appraisals such as irrational beliefs may heighten BIS activation, increasing vigilance to internal threat signals and reinforcing avoidance behaviors (7, 14). Similarly, engagement in health-promoting behaviors may enhance BAS functioning by increasing reward sensitivity, mastery experiences, and behavioral engagement, thereby reducing perceived pain intensity (23, 30). This dual-process motivational framework provides a more integrative explanation of pain modulation than purely cognitive or behavioral models.

Neurobiological evidence also supports the interaction between cognitive biases and motivational systems in pain processing. Early life stress and emotional dysregulation have been shown to alter neural circuits involved in visceral hypersensitivity and emotional comorbidity, particularly within limbic regions such as the amygdala (3). Similarly, pain catastrophizing has been associated with altered reward processing and reduced neural responsiveness to positive stimuli, suggesting impaired BAS-related functioning (14). These findings highlight the neurocognitive overlap between pain, emotion, and motivation systems.

Recent advances in experimental pain research further demonstrate that cognitive biases for pain can interfere with attentional and cognitive processing, particularly under conditions of anticipated pain threat (9). Such findings align with broader evidence indicating that cognitive rigidity and reduced flexibility exacerbate symptom persistence in IBS and other chronic pain conditions (15). Moreover, behavioral

activation-based interventions have shown promise in reducing pain interference by enhancing engagement in meaningful activities and reducing avoidance behaviors (30).

From a methodological perspective, structural equation modeling (SEM) has been increasingly used to examine complex multivariate relationships in pain research, enabling simultaneous testing of direct and indirect pathways among cognitive, behavioral, and emotional variables (31). This analytic approach is particularly suitable for examining integrated models of IBS symptomatology, where multiple interacting variables contribute to pain perception.

Despite substantial progress, existing literature remains fragmented. Most studies have examined either cognitive factors (such as irrational beliefs or catastrophizing) or behavioral/lifestyle factors in isolation, without integrating motivational systems as mediating mechanisms. Furthermore, limited research has specifically focused on IBS populations using comprehensive models that incorporate BIS/BAS alongside cognitive and behavioral predictors. Given the high prevalence and clinical burden of IBS, as well as its strong association with psychological distress and impaired quality of life (4, 19), there is a clear need for integrative models that explain how cognitive and behavioral factors jointly influence pain perception.

In summary, the literature suggests that pain perception in IBS is shaped by an interplay of maladaptive cognitive beliefs, health-related behaviors, and underlying motivational systems that regulate approach and avoidance tendencies. However, the mediating mechanisms through which these variables interact remain insufficiently understood, particularly in the context of IBS-related pain. Addressing this gap is essential for developing more precise psychological and behavioral interventions targeting both cognitive restructuring and motivational system modulation.

This study aimed to examine the mediating roles of behavioral activation and behavioral inhibition systems in the relationships between irrational beliefs, health-promoting behaviors, and pain perception in patients with Irritable Bowel Syndrome.

Methods and Materials

Study Design and Participants

This study used a cross-sectional correlational design and employed structural equation modeling (SEM) to examine the relationships among irrational beliefs, health-promoting behaviors, behavioral activation and inhibition systems (BAS/BIS), and pain perception in patients with Irritable Bowel Syndrome (IBS). Data were collected during 2024 from patients who had been referred to the Masoud Gastrointestinal and Liver Clinic in Iran.

Participants were recruited using a convenience sampling procedure. Patients who attended the clinic during the data-collection period were screened for eligibility and were invited to participate in the study after receiving an explanation of the research objectives and procedures. Individuals were considered eligible if they had received a clinical diagnosis of IBS based on the Rome IV criteria confirmed by a gastroenterologist, were between 20 and 45 years of age, had no diagnosed severe psychiatric disorders and were not using psychiatric medications, and expressed willingness to participate in the research. Participants who withdrew during the study process or returned incomplete questionnaires were excluded from the final dataset.

A total of 320 patients with IBS participated in the study. The adequacy of the sample size was determined according to Kline's (2023) recommendation for sample size in structural equation modeling, which suggests that path models should include approximately 10 to 20 participants per estimated parameter. Based on the number of free parameters in the proposed structural model, a sample size of 320 was considered sufficient to ensure stable estimation of model parameters and adequate statistical power.

Prior to data collection, the research protocol was approved by Islamic Azad University, and permission to conduct the study was obtained from the medical center. Participants were informed about the purpose of the study, confidentiality of their responses, voluntary nature of participation, and their right to withdraw from the research at any stage without any consequences. Ethical principles, including protection of participants' privacy, confidentiality of data, avoidance of harm, and respect for participant autonomy, were carefully maintained throughout the study.

In addition, demographic characteristics such as age, gender, and educational level were collected and examined in the statistical analyses to assess their potential influence on the relationships among the study variables and to ensure that the structural model results were not substantially confounded by demographic factors.

Data Collection

McGill Pain Questionnaire (MPQ): The McGill Pain Questionnaire (MPQ), developed by Melzack (1987), consists of 20 sets of descriptive words designed to assess the multidimensional nature of pain perception. Scoring is performed on a dichotomous scale (0–1); if the respondent selects a descriptor within a set, a score of 1 is assigned, whereas no selection yields a score of 0. Exploratory and confirmatory factor analyses have supported a four-component structure comprising Sensory, Affective, Evaluative, and Miscellaneous dimensions. Kuder-Richardson coefficients for these dimensions have been reported as .77, .91, .75, and .89, respectively. In the Iranian context, Naderipour et al. (2023) reported reliability using the Kuder-Richardson coefficient, with values ranging from .79 to .85 across dimensions, and a content validity index of .87. In the present study, the internal consistency of the scale was calculated using Cronbach's alpha, yielding a coefficient of .73.

Behavioral Inhibition/Activation Systems (BIS/BAS) Scales: The Behavioral Inhibition/Activation Systems (BIS/BAS) Scales is a self-report inventory developed by Carver and White (1994) to assess the two behavioral systems of inhibition and activation. This scale comprises 24 items and two main scales: the Behavioral Inhibition System scale and the Behavioral Activation System scale. The researchers examined the psychometric properties of this scale and reported an internal consistency of .74 for the BIS. For the BAS subscales—including Reward Responsiveness (5 items), Drive (4 items), and Fun Seeking (4 items)—internal consistency coefficients were reported as .73, .76, and .66, respectively. In Iran, the validity of this questionnaire was reported using the test-retest method as .78 for the BAS scale and .81 for the BIS subscale. In the present study, the internal consistency of this scale was calculated using Cronbach's alpha, yielding .78 for behavioral activation and .81 for behavioral inhibition.

Health-Promoting Lifestyle Profile (HPLP): The Health-Promoting Lifestyle Profile (HPLP), developed by Walker et al. (1987) based on Pender's Health Promotion Model, assesses lifestyle behaviors focused on maintaining or enhancing well-being, self-actualization, and individual satisfaction. This

instrument comprises 52 items distributed across six subscales: Nutrition, Physical Activity, Stress Management, Health Responsibility, Interpersonal Relations, and Spiritual Growth. Responses are rated on a 4-point Likert scale ranging from Never to Always. Scoring is assigned as follows: Never (0), Sometimes (1), Often (2), and Always (3). The original authors reported a total reliability coefficient of .94, with subscale reliabilities ranging from .79 to .94. The psychometric properties of this instrument have been evaluated in Iran; Mousavi et al. (2021) confirmed its validity and reported a Cronbach's alpha of .82 for the total scale, with subscale alphas ranging from .79 to .91. Although the Cronbach's alpha for the HPLP was .69, this value is considered acceptable for research purposes in large-sample correlational studies. In the present study, the internal consistency of the scale was calculated using Cronbach's alpha, yielding a coefficient of .69.

Irrational Beliefs Test (IBT): To assess irrational beliefs, the short form of the Irrational Beliefs Test was employed. This 40-item inventory was developed and validated by Ebadi and Motamedin for the Iranian population based on Jones's original 10-factor structure using factor analysis. The scale measures four factors: Helplessness for Change, Demand for Approval, Problem Avoidance, and Emotional Irresponsibility. Items are rated on a 5-point Likert scale ranging from 1 (Strongly Disagree) to 5 (Strongly Agree). Regarding psychometric properties, Ebadi and Motamedin evaluated the reliability using Cronbach's alpha and split-half methods, reporting a reliability coefficient of .76 for the total scale. In the present study, the internal consistency of the scale was calculated using Cronbach's alpha, yielding a coefficient of .77.

Data Analysis

The collected data were analyzed using path analysis within a structural equation modeling (SEM) framework employing IBM SPSS AMOS version 24. Prior to model testing, the assumptions underlying SEM were systematically examined. First, descriptive statistics (means, standard deviations, skewness, and kurtosis) were calculated for all study variables (Table 1). Univariate normality was assessed based on skewness and kurtosis values, with values within the range of ± 2 considered acceptable. As shown in Table 1, all variables satisfied this criterion, indicating approximate univariate normality.

To evaluate multivariate normality, Mardia's multivariate kurtosis coefficient was computed. The obtained value (3.41) was below the recommended threshold of 5, suggesting that the assumption of multivariate normality was adequately met. Additionally, multivariate outliers were examined using Mahalanobis distance (d^2). No cases exceeded the critical chi-square value at $p < .05$, indicating the absence of multivariate outliers. To assess potential multicollinearity among predictor variables, Tolerance and Variance Inflation Factor (VIF) indices were calculated. Tolerance values were close to 1 and all VIF values were below 2, demonstrating that multicollinearity was not a concern in the present model.

Findings and Results

The sample comprised 320 patients diagnosed with Irritable Bowel Syndrome (IBS), including 101 males (31.6%) and 219 females (68.4%). In terms of age distribution, 198 participants (61.9%) were between 20 and 30 years old, whereas 122 participants (38.1%) were between 31 and 45 years old. Regarding educational attainment, 114 participants (35.6%) had a high school diploma or associate degree, 129 (40.3%) held a bachelor's degree, and 77 (24.1%) possessed a master's degree or higher. The means and standard deviations of the study variables are presented in Table 1.

Table 1. Descriptive Statistics of Study Variables

Variable	<i>M</i>	<i>SD</i>	Skewness	Kurtosis
Health-Promoting Behaviors	125.44	19.72	0.06	-1.08
Irrational Beliefs	128.46	20.59	0.25	-0.58
Behavioral Inhibition (BIS)	14.93	3.40	-0.24	0.45
Behavioral Activation (BAS)	37.41	9.60	-0.94	-0.47
Pain Perception	49.33	8.04	-0.43	0.16

Table 2. Correlation Matrix of Study Variables

Variable	1	2	3	4	5
1. Health-Promoting Behaviors	1				
2. Irrational Beliefs	-.39**	1			
3. Behavioral Inhibition (BIS)	-.24**	.28**	1		
4. Behavioral Activation (BAS)	.30**	-.11	-.41**	1	
5. Pain Perception	-.43**	.39**	.36**	-.29**	1

Pearson correlation coefficients among the study variables are presented in Table 2. The results indicated significant associations in the expected directions among health-promoting behaviors, irrational beliefs, BIS, BAS, and pain perception ($p < .01$), supporting the suitability of proceeding with path analysis.

Table 3. Goodness-of-Fit Indices for the Proposed Model

Model	χ^2/df	GFI	PCFI	PNFI	CFI	IFI	RMSEA
Research Model	2.689	0.923	0.785	0.798	0.924	0.919	0.067

Note. Acceptable thresholds: $CMIN/DF < 3$; $RMSEA < .08$; $CFI, GFI, IFI > .9$; $PCFI, PNFI > .5$

The hypothesized conceptual model was evaluated using multiple goodness-of-fit indices, including χ^2/df , RMSEA, GFI, CFI, IFI, PCFI, and PNFI (Table 3). The results demonstrated an acceptable to good model fit ($\chi^2/df = 2.69$, $RMSEA = 0.067$, $CFI = 0.924$, $GFI = 0.923$, $IFI = 0.919$), all of which met commonly accepted thresholds.

The coefficient of determination (R^2) for pain perception was .50, indicating that 50% of the variance in pain perception among patients with IBS was explained by irrational beliefs, health-promoting behaviors, and the mediating roles of BAS and BIS, this represents a moderate to substantial explanatory power.

Table 4. Direct Path Coefficients Among Study Variables in the Structural Model

Outcome Variable	Predictor Variable	Effect	Unstandardized Coefficient	Standardized Coefficient (β)	t	Sig.
Pain Perception	Health-Promoting Behaviors	Direct	-0.70	-0.41	-6.45	0.001
Pain Perception	Irrational Beliefs	Direct	0.67	0.36	5.51	0.001
Pain Perception	Behavioral Activation	Direct	-0.65	-0.31	-5.17	0.001
Pain Perception	Behavioral Inhibition	Direct	0.19	0.35	5.48	0.001
Behavioral Inhibition	Health-Promoting Behaviors	Direct	0.39	0.27	4.50	0.001
Behavioral Activation	Health-Promoting Behaviors	Direct	-0.43	-0.24	-4.39	0.001
Behavioral Inhibition	Irrational Beliefs	Direct	0.17	0.30	5.16	0.001
Behavioral Activation	Irrational Beliefs	Direct	-0.22	-0.09	-1.27	0.106

As presented in Table 4, the standardized path coefficients indicated that health-promoting behaviors had a significant negative direct effect on pain perception ($\beta = -0.41$, $p < 0.001$), whereas irrational beliefs showed a significant positive direct effect on pain perception ($\beta = 0.36$, $p < 0.001$). Regarding motivational

systems, behavioral activation (BAS) was negatively associated with pain perception ($\beta = -0.31, p < 0.001$), while behavioral inhibition (BIS) demonstrated a significant positive association with pain perception ($\beta = 0.35, p < 0.001$).

In terms of predictor–mediator relationships, health-promoting behaviors were significantly associated with both BIS ($\beta = 0.27, p < 0.001$) and BAS ($\beta = -0.24, p < 0.001$). Additionally, irrational beliefs were positively associated with BIS ($\beta = 0.30, p < 0.001$); however, their relationship with BAS was not statistically significant ($\beta = -0.09, p = 0.106$). All direct effects and standardized path coefficients are reported in Table 4.

The mediating roles of BAS and BIS were examined using bootstrapping with 2,000 resamples and a 95% confidence interval. The results indicated that the indirect effect of health-promoting behaviors on pain perception through BAS was significant (95% CI [-0.13, -0.05]). Likewise, the indirect effect through BIS was also significant (95% CI [-0.12, -0.04]), suggesting partial mediation.

Furthermore, BIS significantly mediated the relationship between irrational beliefs and pain perception (95% CI [0.09, 0.19]). In contrast, BAS did not significantly mediate this relationship, as the confidence interval included zero. Overall, these findings support a model in which health-promoting behaviors influence pain perception both directly and indirectly through BAS and BIS, whereas irrational beliefs exert their indirect effect primarily through BIS.

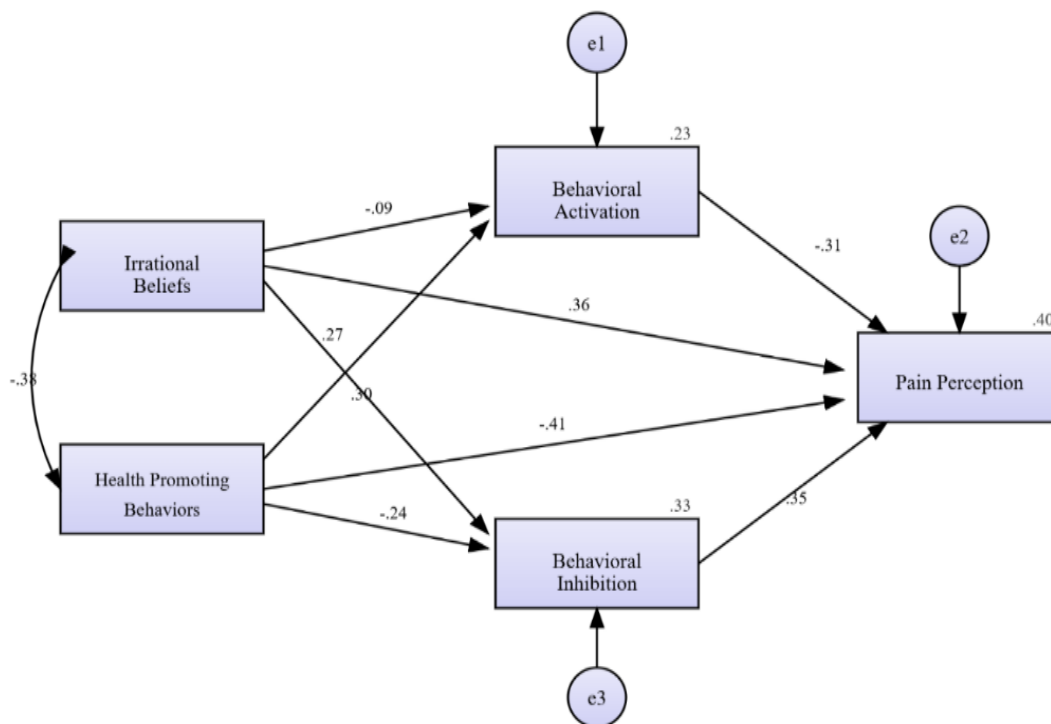


Figure 1. Path Analysis Model of the Research in Standardized Coefficients Mode

Discussion and Conclusion

The present study investigated an integrated cognitive–behavioral–motivational model of pain perception in patients with Irritable Bowel Syndrome (IBS), focusing on the relationships between irrational beliefs, health-promoting behaviors, behavioral activation (BAS), behavioral inhibition (BIS), and pain perception.

Overall, the findings supported a multidimensional explanatory framework in which both cognitive distortions and lifestyle behaviors exerted significant direct effects on pain perception, while also operating indirectly through motivational systems (BAS/BIS). The structural model demonstrated acceptable fit indices and explained a substantial proportion of variance in pain perception, indicating the robustness of the proposed theoretical model in capturing the complexity of IBS-related pain processing.

One of the central findings was the significant positive relationship between irrational beliefs and pain perception in IBS patients. This result is consistent with cognitive models of chronic pain, which emphasize that maladaptive beliefs such as catastrophizing, rigid thinking, and emotional irresponsibility amplify the subjective experience of pain by altering attentional focus and emotional appraisal processes (10, 13). Individuals with stronger irrational belief systems are more likely to interpret visceral sensations as threatening or uncontrollable, which intensifies perceived pain severity and emotional distress. This finding aligns with prior evidence indicating that cognitive biases and maladaptive interpretations of bodily sensations contribute to heightened pain sensitivity and interference in daily functioning (7, 9). In IBS specifically, such cognitive distortions may exacerbate visceral hypersensitivity through heightened anticipatory anxiety and maladaptive symptom monitoring, consistent with the broader neurocognitive model of IBS pathophysiology (1, 8).

The present findings also demonstrated that health-promoting behaviors were significantly and negatively associated with pain perception. This result is consistent with extensive literature highlighting the protective role of lifestyle behaviors in chronic pain conditions. Engagement in adaptive health behaviors, including physical activity, stress management, and dietary regulation, has been shown to reduce pain severity and improve quality of life across chronic pain populations (18, 19). From a mechanistic perspective, health-promoting behaviors may reduce pain perception through physiological pathways such as decreased inflammation, improved autonomic regulation, and enhanced gut–brain axis functioning in IBS (20, 21). Additionally, behavioral engagement enhances psychological resilience by increasing self-efficacy and perceived control over symptoms, which reduces catastrophic thinking and emotional amplification of pain experiences (23, 24). These findings reinforce the importance of lifestyle regulation as a central component in the management of IBS-related pain.

A key contribution of the present study lies in clarifying the mediating role of behavioral activation (BAS) and behavioral inhibition (BIS) systems in the relationship between health-promoting behaviors and pain perception. The results indicated that both BAS and BIS significantly mediated this relationship, suggesting that lifestyle behaviors influence pain not only directly but also through motivational systems that regulate approach and avoidance behavior. This is consistent with reinforcement sensitivity theory, which posits that BAS facilitates reward-driven engagement in adaptive activities, whereas BIS governs sensitivity to threat and avoidance tendencies (26, 27). Individuals who engage in health-promoting behaviors are likely to experience increased behavioral activation, which enhances goal-directed behavior, positive reinforcement, and adaptive coping, ultimately reducing pain perception (28, 30). Conversely, reduced behavioral inhibition may lower avoidance behaviors and hypervigilance toward visceral sensations, thereby attenuating pain amplification processes.

The mediating role of BIS further highlights the importance of threat processing in IBS-related pain. Elevated BIS activity is associated with heightened sensitivity to potential harm, increased anxiety, and

avoidance-oriented coping strategies, all of which are known to exacerbate chronic pain experiences (26, 29). In IBS patients, heightened BIS may lead to excessive monitoring of gastrointestinal sensations and avoidance of daily activities due to fear of symptom exacerbation, thereby reinforcing disability and pain persistence. These findings align with prior research suggesting that behavioral inhibition plays a central role in chronic pain maintenance through fear-avoidance mechanisms and attentional bias toward somatic threat cues (6, 9). Therefore, interventions aimed at reducing BIS activation or modifying threat sensitivity may be particularly beneficial in this population.

Another important finding was the significant mediating role of BIS in the relationship between irrational beliefs and pain perception, whereas BAS did not significantly mediate this association. This suggests that irrational beliefs primarily influence pain perception through threat-based cognitive-emotional pathways rather than reward-driven motivational systems. Individuals with stronger irrational beliefs are more likely to engage in catastrophic interpretations and rigid thinking patterns, which activate BIS-related processes such as anxiety, vigilance, and behavioral avoidance (11, 14). This pathway aligns with cognitive-affective models of pain, which emphasize that maladaptive beliefs increase pain perception by enhancing negative emotional arousal and attentional bias toward internal sensations (7, 8). The lack of mediation through BAS suggests that irrational beliefs may not significantly impair approach-oriented motivation, but rather primarily intensify avoidance and threat sensitivity mechanisms.

The findings of this study are also consistent with emerging evidence highlighting the central role of cognitive and behavioral integration in IBS symptomatology. IBS has been increasingly conceptualized as a disorder of brain-gut interaction, where psychological processes significantly modulate gastrointestinal functioning and pain perception (1, 22). Neurobiological studies have demonstrated that early life stress and emotional dysregulation can alter neural circuits associated with visceral hypersensitivity, particularly in limbic structures involved in emotional processing and pain modulation (3). These alterations may predispose individuals to heightened BIS activation and maladaptive cognitive interpretations of bodily sensations. The present findings extend this neurobiological framework by demonstrating that cognitive beliefs and lifestyle behaviors interact with motivational systems to shape subjective pain experiences.

Furthermore, the results support prior findings indicating that behavioral activation-based approaches can reduce pain severity by increasing engagement in rewarding and meaningful activities, thereby counteracting avoidance tendencies (30). Similarly, cognitive-behavioral interventions targeting maladaptive beliefs have been shown to improve cognitive flexibility and reduce symptom severity in IBS patients (15). The present study integrates these findings by demonstrating that both cognitive restructuring and behavioral activation may exert their therapeutic effects through modulation of BIS and BAS systems, offering a more comprehensive explanation for treatment efficacy in chronic pain populations.

Overall, the findings highlight the importance of an integrated biopsychosocial framework for understanding pain perception in IBS. Rather than being driven solely by peripheral gastrointestinal dysfunction, pain in IBS appears to be shaped by a complex interaction between cognitive appraisals, behavioral engagement, and underlying motivational systems. This integrated model is consistent with broader literature emphasizing the multidimensional nature of chronic pain and the need for interventions that simultaneously target cognitive, behavioral, and emotional domains (19, 20).

Despite its theoretical and empirical contributions, the present study has several limitations. First, the cross-sectional design limits the ability to draw causal inferences regarding the relationships among irrational beliefs, health-promoting behaviors, BIS/BAS, and pain perception. Longitudinal or experimental designs would be necessary to establish temporal and causal pathways among these variables. Second, the reliance on self-report questionnaires may introduce response biases such as social desirability and subjective reporting error, potentially affecting the accuracy of measured constructs. Third, the sample was drawn from a single clinical center, which may limit the generalizability of findings to broader IBS populations across different cultural or healthcare contexts. Finally, the study did not incorporate biological markers or neurophysiological measures, which could have provided additional insight into the underlying mechanisms of pain perception.

Future research should employ longitudinal designs to examine the temporal dynamics of cognitive, behavioral, and motivational variables in IBS-related pain. Experimental and intervention-based studies are also recommended to determine whether modifying irrational beliefs and enhancing health-promoting behaviors can causally influence BIS/BAS functioning and pain outcomes. Additionally, integrating neurobiological measures such as neuroimaging or hormonal markers could provide a more comprehensive understanding of the brain–gut–behavior interaction. Future studies should also consider cross-cultural comparisons to evaluate whether these mechanisms operate similarly across different populations and healthcare systems.

Clinical interventions for IBS should adopt an integrated cognitive–behavioral approach that targets both maladaptive beliefs and lifestyle behaviors. Cognitive restructuring techniques can be used to reduce irrational beliefs and catastrophic thinking patterns, while behavioral activation strategies can encourage engagement in rewarding and meaningful activities. Health-promoting behavior programs focusing on physical activity, nutrition, and stress management may further reduce pain perception by enhancing adaptive motivational processes. Additionally, clinicians should consider assessing BIS/BAS profiles in patients to tailor interventions that reduce avoidance behaviors and strengthen approach-oriented coping strategies, ultimately improving pain management outcomes in IBS populations.

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Authors' Contributions

All authors equally contributed to this study.

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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