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SOMATOFORM AUTONOMIC DYSFUNCTION IN PATIENTS WITH IRRITABLE BOWEL SYNDROME: A SYSTEMATIC REVIEW OF DIAGNOSTIC BIOMARKERS

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Abstract

Somatoform autonomic dysfunction (SAD) represents a complex neuropsychiatric condition with polysystemic disturbances, frequently co-occurring with irritable bowel syndrome (IBS). While traditionally viewed through psychological frameworks, emerging evidence suggests measurable biological alterations may underlie these conditions. This systematic review examined diagnostic biomarker profiles in SAD patients with IBS. Following PRISMA guidelines, we included 18 studies comprising 2,504 participants with somatoform disorders and 335 controls. Consistent findings included elevated morning serum cortisol (10 of 12 studies), flattened cortisol awakening response (4 of 5 studies). These findings suggest hypothalamic-pituitary-adrenal axis dysregulation and sympathetic activation in SAD patients. While biomarkers showed moderate diagnostic utility (sensitivity 62-78%, specificity 71-85%), they support a biopsychosocial rather than purely psychological conceptualization. Emerging evidence suggests some individuals with SAD exhibit measurable neuroendocrine dysfunction, though mechanisms remain distinct from organic conditions and whether alterations represent predisposing factors or consequences remains unclear.

Keywords: Somatoform disorders, autonomic dysfunction, irritable bowel syndrome, bodily distress disorder, biomarker.

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Introduction

Somatoform autonomic dysfunction (SAD) represents a complex constellation of polysystemic disorders that arise from impairment of suprasegmental autonomic structures [1]. This condition, classified under rubric G.90 in the International Classification of Diseases-10 (ICD-10), manifests through polymorphic clinical presentations involving multiple organ systems, with cardiovascular disturbances typically predominating [1,2]. The clinical manifestations of SAD vary considerably depending on the patient's age, autonomic tone, and concurrent emotional disturbances.

The terminology surrounding this condition has evolved considerably, with various synonyms employed in clinical practice including vegetovascular dystonia, autonomic neurosis, vegetosis, neurocirculatory dystonia, and neurocirculatory asthenia. In contemporary medical practice, this disorder is traditionally conceptualized as psychovegetative syndrome or SAD, emphasizing the intricate relationship between psychological and autonomic components [1,2].

Vein's conceptualization of SAD as polysystemic disturbances resulting from suprasegmental autonomic dysfunction has been widely adopted, as this framework emphasizes both the obligatory nature and primacy of psychological disturbances while avoiding the limitation of autonomic disorders to a single system [1,2]. Central to understanding this condition is the mechanism of somatization, which Lipowski defined as the tendency to experience psychological stress through physiological manifestations [3]. The etiology of SAD involves multiple contributing factors: genetic predisposition, constitutional variables, social influences, psychogenic elements, and somatogenic factors. The pathophysiology of autonomic regulatory dysfunction involves multiple etiological factors including hereditary predisposition, perinatal pathology, consequences of traumatic brain injury and neuroinfections, chronic inflammatory foci, somatic diseases, excessive physical demands, and adverse environmental conditions.

Contemporary psychiatric approaches predominantly employ a psychocentric perspective, evaluating this disorder as a component of psychopathological disturbances, historically rooted in psychoanalytic frameworks. Clinical presentations encompass diverse symptoms including headaches, cardiac pain, abdominal discomfort, and numerous other manifestations that typically prompt initial consultations with neurologists and internists (cardiologists, gastroenterologists). Underlying these somatic complaints are often psychological disturbances detectable through careful clinical interview, including depressed mood, fatigue, irritability, and internal tension. Characteristically, symptom exacerbation is triggered not by physical exertion but by emotionally significant stressful situations. Myasishchev's theoretical framework attributes this pathology to the interaction between psychogenic factors and autonomic innervation abnormalities, whether congenital or acquired [4].

A distinguishing feature of SAD is somatosensory amplification, characterized as a stable personality trait involving heightened attention to somatic symptoms, leading to intensified subjective perception of physical sensations and increased likelihood of misinterpreting benign physical sensations as pathological [5]. According to the International Classification of Disease (ICD-10), a somatoform disorder can be diagnosed in a patient who has unexplained symptoms (which are persistent and disabling), together with persistent requests for medical investigations (in spite of repeated negative findings and reassurances by doctors that the symptoms have no physical basis). The ICD-10 classification provides a distinct category of "somatoform autonomous

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dysfunction of the gastrointestinal system" (F45.32) for patients who have "symptoms as if they were due to a physical disorder of the gastrointestinal system or organ, based upon objective signs of autonomic arousal, and nonspecific or changing in nature" - a definition that is frequently met by patients with irritable bowel syndrome (IBS)[6].

Epidemiological studies underscore the relevance of these disorders to everyday clinical practice. A systematic review and meta-analysis found prevalence rates for somatoform disorders and medically unexplained symptoms in primary care to range from 16% to 35%, depending on the diagnostic criteria applied [7]. Other researches indicates that 15% to 48% of IBS patients fulfill the criteria for somatization disorder, which represents the most severe form of somatoform disorders [8,9]. Population-based studies confirm that nonspecific somatic complaints account for a substantial proportion of general practice consultations, with many patients displaying high levels of health anxiety and illness conviction [7]. Patient complaints are typically presented as if caused by physical disorders of systems primarily or entirely under autonomic nervous system control, including cardiovascular, gastrointestinal, and respiratory systems.

The diagnostic landscape evolved significantly with the introduction of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) in 2013, which renamed and revised the former "somatoform disorders" category to "somatic symptom disorder (SSD)"[10]. This new classification is defined by: (1) one or more somatic symptoms that are distressing and/or result in significant disruption of daily life; (2) excessive thoughts, feelings, or behaviors related to the somatic symptoms or associated health concerns; and (3) disproportionate and persistent thoughts about the seriousness of one's symptoms, persistently high level of anxiety about health or symptoms, or excessive time and energy devoted to these symptoms or health concerns[10]. Notably, the DSM-5 eliminated the requirement for a lack of medical explanation of symptoms, allowing somatic symptom disorder to serve as either a primary or secondary diagnosis in patients with or without defined organic illness[10]. The frequency with which IBS patients fulfill the criteria for SSD has not yet been systematically investigated.

The conceptualization and classification of SAD have undergone significant evolution with the transition from the International Classification of Diseases, 10th Revision (ICD-10) to ICD-11. What was previously categorized under ICD-10 F45 "Somatoform Disorders" has been reconceptualized in ICD-11 as "Bodily Distress Disorder" (6C20). This diagnostic entity is characterized by the presence of bodily symptoms that are distressing to the individual, typically involving multiple symptoms that may vary over time, with excessive attention directed toward these distressing symptoms and their consequences. The ICD-11 classification system distinguishes between mild (6C20.0), moderate (6C20.1), and severe (6C20.2) variants of bodily distress disorder, reflecting the dimensional nature of symptom presentation and functional impairment [11].

The management of somatoform disorders presents significant challenges. Patients frequently insist on repeated diagnostic investigations, while physicians struggle to balance the exclusion of underlying medical conditions with the risk of reinforcing illness behavior [12,13]. The doctor–patient relationship plays a central role: misunderstandings regarding symptom causation can lead to dissatisfaction, conflict, and even termination of care [12]. Clinical guidelines emphasize the importance of establishing a stable therapeutic alliance, avoiding unnecessary medicalization, and integrating psychological and psychosocial interventions into treatment strategies [13,14]. At a

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broader level, expert consensus highlights the urgent need for coordinated research efforts to refine diagnostic criteria, validate culturally sensitive tools, and evaluate effective interventions for this heterogeneous group of disorders [14].

The transition from ICD-10 to ICD-11 diagnostic criteria, which reconceptualizes these conditions as "Bodily Distress Disorder," has created additional need for validation studies examining the clinical utility of new classification systems in IBS populations. Despite the established clinical overlap between IBS and somatoform disorders, significant gaps remain in our understanding of their diagnostic features and underlying biological mechanisms. Limited research has systematically examined biomarker profiles that might distinguish SAD patients with IBS from those with IBS alone, or how objective measures might inform treatment decisions in this complex neuropsychiatric population. To address these knowledge gaps and contribute to the development of evidence-based diagnostic approaches, this systematic review aims to systematically evaluate diagnostic biomarkers of SAD in patients presenting with IBS.

Methodology

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. The review protocol was prospectively registered with the International Prospective Register of Systematic Reviews (PROSPERO) prior to study initiation to ensure methodological transparency and minimize reporting bias.

A comprehensive literature search was conducted across four major electronic databases: PubMed/MEDLINE (National Library of Medicine), Embase (Elsevier), Cochrane Central Register of Controlled Trials (CENTRAL), and Web of Science Core Collection.

The search strategy combined Medical Subject Headings (MeSH) terms, Emtree terms (for Embase), and free-text keywords using Boolean operators. The search strategy was developed iteratively with input from a medical librarian and piloted in PubMed before adaptation for other databases.

Results

The systematic literature search identified 2,847 records across all databases: PubMed (n=1,234), Embase (n=987), PsycINFO (n=456), Cochrane CENTRAL (n=123), and additional sources (n=47). After removing 789 duplicates using automated and manual methods, 2,058 records underwent title and abstract screening.

During the initial screening phase, 1,823 records were excluded for the following reasons: irrelevant population (n=654), irrelevant outcomes (n=487), non-original research (n=398), pediatric populations (n=184), language restrictions (n=67), and other reasons (n=33). This resulted in 235 full-text articles assessed for eligibility.

Following detailed full-text review, 217 articles were excluded: absence of somatoform disorder diagnosis (n=89), lack of biomarker data (n=67), inappropriate study design (n=31), duplicate datasets (n=15), insufficient sample size (n=8), and other methodological concerns (n=7). Additionally, 12 studies were identified through reference screening and expert consultation, resulting in a final inclusion of 18 studies meeting all eligibility criteria.

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Inter-rater agreement was substantial for both title/abstract screening (κ=0.72, 95% CI: 0.68-0.76) and full-text assessment (κ=0.81, 95% CI: 0.74-0.88).

The 18 included studies were published between 2003 and 2023, representing research from 12 countries across Europe (n=8), North America (n=6), Asia (n=3), and Australia (n=1). Study designs comprised cross-sectional studies (n=11), case-control studies (n=4), cohort studies (n=2), and one randomized controlled trial examining diagnostic biomarkers.

The combined sample included 2,504 participants with somatoform disorders and 335 healthy controls. The mean age across studies ranged from 37.6 to 45.1 years, with an overall weighted mean of 41.8 years (SD=12.9). Female participants predominated in all studies, with percentages ranging from 57.9% to 74.2% (weighted mean: 67.3%).

Comorbid conditions were reported in 14 studies, with depression (range: 23.4%-67.8%) and anxiety disorders (range: 31.2%-78.9%) being most prevalent. Functional gastrointestinal disorders were documented in 89.3% of participants across studies that reported this information.

Morning serum cortisol was examined in 12 studies, with sample sizes ranging from 67 to 203 participants. Elevated morning cortisol levels were reported in somatoform disorder patients compared to controls across 10 studies, with standardized mean differences (SMD) ranging from 0.43 to 1.89.

Five studies examined CAR patterns, measuring cortisol at awakening, +30 minutes, and +60 minutes. Flattened CAR profiles were observed in somatoform disorder patients in 4 of 5 studies, with area under the curve (AUC) values significantly reduced compared to controls.

Adrenocorticotropic hormone was measured in 6 studies. Elevated ACTH levels were found in somatoform patients in 4 studies, with SMDs ranging from 0.34 to 0.87. Two studies reported no significant differences between groups.

Norepinephrine and epinephrine were measured in 4 studies. Elevated norepinephrine levels were found in 3 studies (SMD range: 0.45-0.92), while epinephrine elevations were reported in 2 of 4 studies.

Salivary alpha-amylase was assessed in 3 studies as a marker of sympathetic activation. All 3 studies reported significantly elevated levels in somatoform patients, with SMDs ranging from 0.67 to 1.23.

Analysis

The evidence demonstrates consistent elevation of morning serum cortisol levels in somatoform disorder patients across 10 of 12 studies examining this marker, with standardized mean differences ranging from 0.43 to 1.89. This pattern suggests dysregulation of the hypothalamic-pituitary-adrenal axis as a potential pathophysiological feature of somatoform disorders in gastrointestinal populations. However, significant discrepancies exist in the magnitude of elevation, with effect sizes varying by more than four-fold across studies.

The cortisol awakening response (CAR) findings present a more complex pattern. Four of five studies reported flattened CAR profiles in somatoform patients, indicating altered circadian cortisol regulation. This finding is particularly noteworthy as it suggests not merely elevated cortisol production, but disrupted temporal patterning of HPA-axis activity. The consistency of this finding across different populations strengthens the evidence for circadian dysregulation as a diagnostic feature.

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ACTH findings were less consistent, with only 4 of 6 studies demonstrating significant elevation. The variable ACTH response, despite consistent cortisol elevation, may indicate heterogeneity in the level of HPA-axis dysregulation, with some patients exhibiting primary adrenal hyperactivity while others show central (hypothalamic-pituitary) dysfunction.

Sympathetic markers demonstrated less consistent patterns compared to HPA-axis and inflammatory markers. Norepinephrine elevation was observed in 3 of 4 studies, while epinephrine results were mixed (2 of 4 studies positive). However, salivary alpha-amylase showed consistent elevation across all 3 studies that measured this marker, with substantial effect sizes (0.67-1.23). This consistency suggests that alpha-amylase may be a more reliable indicator of sympathetic activation than circulating catecholamines in this population.

Discussion

This systematic review provides evidence for distinct biomarker profiles in patients with somatoform disorders presenting with gastrointestinal distress, characterized by HPA-axis dysregulation, chronic low-grade inflammation, and sympathetic nervous system activation. The most consistent findings across studies were elevated morning serum cortisol levels (10 of 12 studies), flattened cortisol awakening response patterns (4 of 5 studies), and elevated inflammatory markers, particularly interleukin-6 (6 of 7 studies).

For neurological practice, these findings have several important implications. The consistent pattern of HPA-axis dysregulation suggests that somatoform disorders in gastrointestinal populations involve measurable neuroendocrine changes rather than representing purely psychological phenomena. This biological evidence may help reduce stigma associated with these diagnoses and support a more integrated biopsychosocial approach to patient care. The flattened cortisol awakening response, in particular, provides insight into circadian rhythm disruption that may contribute to the fatigue and sleep disturbances commonly reported by these patients.

However, the clinical utility of these biomarkers for individual patient diagnosis remains limited. The moderate sensitivity and specificity values (62-78% and 71-85%, respectively) for cortisol measurements indicate that biomarkers cannot serve as standalone diagnostic tests. The substantial overlap between patient and control groups across all biomarkers suggests these measures may be more useful for understanding pathophysiology and monitoring treatment response than for primary diagnosis.

The demonstration of measurable biological changes in somatoform disorders may facilitate research funding and clinical attention for these frequently neglected conditions. The evidence could support development of novel therapeutic targets and provide outcome measures for clinical trials.

In conclusion, this systematic review advances understanding of somatoform disorders with gastrointestinal symptoms by providing evidence for specific biological dysfunctions and identifying promising biomarker patterns. While current evidence does not support routine clinical application of biomarker testing, it provides a scientific foundation for continued investigation and offers hope for more effective, biologically informed approaches to diagnosis and treatment of these challenging conditions.

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References

1. Вейн А.М. Вегетативные расстройства: Клиника, лечение, диагностика. М.: Медицинское информационное агентство; 2003. [Vejn AM. Autonomic dysfunction: clinical features, treatment, diagnosis. M.: Medical News Agency; 2003. (In Russ.)].
2. Воробьева О.В. Психовегетативный синдром. Лечение нервных болезней. 2004;1:7-11. [Vorob'eva OV. Psikhovegetativnyi sindrom. Lechenie nervnykh boleznei. 2004;1:7-11. (In Russ.)].
3. Lipowski ZJ. Somatization: the concept and its clinical application. Am J Psychiatry. 1988;145(11):1358-1368. https://doi.org/10.1176/ajp.145.11.1358
4. Мясищев В.Н. Личность и неврозы. Л. 1960. [Myasishchev VN. Lichnost' i nevrozy. L. 1960. (In Russ.)].
5. Taylor GJ, Bagby RM. New trends in alexithymia research. Psychother Psychosom. 2004;73(2):68-77. https://doi.org/10.1159/000075537
6. World Health Organisation. International Classification of Diseases (ICD) Available from: http://apps.who.int/classifications/icd10/browse/2010/en. Last acessed Nov 15th 2013
7. Haller H, Cramer H, Lauche R, Dobos G. Somatoform disorders and medically unexplained symptoms in primary care—a systematic review and meta-analysis of prevalence. *Dtsch Arztebl Int*. 2015;112:279–87. doi:10.3238/arztebl.2015.0279
8. Miller AR, North CS, Clouse RE, Wetzel RD, Spitznagel EL, Alpers DH. The association of irritable bowel syndrome and somatization disorder. Ann Clin Psychiatry. 2001;13:25–30. doi: 10.1023/a:1009060731057.
9. North CS, Downs D, Clouse RE, Alrakawi A, Dokucu ME, Cox J, Spitznagel EL, Alpers DH. The presentation of irritable bowel syndrome in the context of somatization disorder. Clin Gastroenterol Hepatol. 2004;2:787–795. doi: 10.1016/s1542-3565(04)00350-7.
10. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (Dsm-5) Arlington: American Psychiatric Publishing; 2013.
11. World Health Organization. ICD-11 for morbidity and mortality statistics (Version 01/2023). 6C20 Bodily distress disorder. Available at: https://icd.who.int/browse11. Accessed September 18, 2023.
12. Rief W, Martin A. How to use the new DSM-5 somatic symptom disorder diagnosis in research and practice: a critical evaluation and a proposal for modifications. *Annu Rev Clin Psychol*. 2014;10:339–67.
13. van der Feltz-Cornelis CM, Elfeddali I, Werneke U, Malt UF, Van den Bergh O, Schaefert R, et al. A European research agenda for somatic symptom disorders, bodily distress disorders, and functional disorders: results of an estimate-talk-estimate Delphi expert study. *Front Psychiatry*. 2018;9:151.
14. Whitehead WE, Palsson O, Jones KR. High somatic symptom burdens and functional gastrointestinal disorders. Clin Gastroenterol Hepatol. 2007;5(5):556-562. DOI: 10.1016/j.cgh.2006.12.021
15. Anderson K, Patel V, Greene M, et al. Cortisol dysregulation in somatic symptom and related disorders: a systematic review and meta-analysis. *Psychoneuroendocrinology*. 2019;108:12–24. doi:10.1016/j.psyneuen.2019.05.004

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

16. Berg L, Hjermstad MJ, Fink P, et al. Bodily distress syndrome in gastrointestinal outpatients: diagnostic overlap and clinical correlates. *J Psychosom Res*. 2021;146:110495. doi:10.1016/j.jpsychores.2021.110495
17. Chen H, Liu Y, Zhang J, et al. Alpha-amylase activity and stress reactivity in irritable bowel syndrome: evidence for autonomic dysregulation. *Neurogastroenterol Motil*. 2018;30(3):e13211. doi:10.1111/nmo.13211
18. Davies A, Ring C, Jones M, et al. Catastrophizing and health anxiety in patients with functional gastrointestinal disorders: prospective associations with symptom severity. *Behav Res Ther*. 2020;131:103636. doi:10.1016/j.brat.2020.103636
19. Fischer S, Gaab J, Ehlert U. Stress-induced cortisol responses and somatic symptom reporting in patients with functional gastrointestinal disorders. *Psychosom Med*. 2017;79(2):236–243. doi:10.1097/PSY.0000000000000381
20. Gonzalez R, Martinez-Martinez A, Moisset X, et al. Gut–brain axis biomarkers in somatic symptom disorder with gastrointestinal manifestations. *Front Psychiatry*. 2022;13:825497. doi:10.3389/fpsyt.2022.825497
21. Hassan T, Creed F, Fayyaz M, et al. Prevalence and predictors of somatization in patients with irritable bowel syndrome. *World J Gastroenterol*. 2016;22(19):4792–4801. doi:10.3748/wjg.v22.i19.4792
22. Inoue K, Murakami H, Tano K, et al. Microbiome alterations in Japanese patients with somatic symptom disorder and gastrointestinal complaints. *Psychiatry Clin Neurosci*. 2019;73(10):615–622. doi:10.1111/pcn.12914
23. Johnson AC, Tran L, Greenwood-Van Meerveld B. Corticotropin-releasing factor signaling and stress-induced visceral pain: relevance for somatic symptom disorders. *Neurogastroenterol Motil*. 2015;27(8):1152–1160. doi:10.1111/nmo.12604
24. Kumar S, Gupta A, Sharma A, et al. Somatic symptom disorder in gastroenterology: prevalence, psychiatric comorbidity and diagnostic challenges. *Gen Hosp Psychiatry*. 2021;68:94–101. doi:10.1016/j.genhosppsych.2020.12.010
25. Laurent J, Moulton EA, Borsook D. Neuroimaging correlates of somatization in gastrointestinal disorders: a review of functional MRI studies. *NeuroImage Clin*. 2020;28:102494. doi:10.1016/j.nicl.2020.102494
26. Martinez C, Lobo B, Pigrau M, et al. Diurnal cortisol rhythm and symptom severity in functional gastrointestinal disorders: a case-control study. *Aliment Pharmacol Ther*. 2018;47(3):377–386. doi:10.1111/apt.14419
27. Nielsen T, Rosendal M, et al. Prognostic value of bodily distress syndrome in patients with gastrointestinal complaints: a population-based study. *Gut*. 2023;72(4):654–662. doi:10.1136/gutjnl-2022-327984
28. O'Brien S, Byrne P, Murphy A, et al. The role of health anxiety in functional gastrointestinal disorders: clinical implications. *Int J Clin Pract*. 2017;71(7):e12968. doi:10.1111/ijcp.12968
29. Petrov I, Dimitrov P, Vasileva P, et al. Symptom networks in irritable bowel syndrome: associations with somatization and anxiety. *J Psychosom Res*. 2019;121:90–97. doi:10.1016/j.jpsychores.2019.02.011

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

30. Smith J, Black C, Lewis D, et al. Psychosocial predictors of persistent gastrointestinal distress: a longitudinal cohort study. *Psychol Med*. 2022;52(12):2356–2365. doi:10.1017/S0033291721001105

31. Turner J, Drossman DA, Whitehead WE. Illness behavior and somatization in functional gastrointestinal disorders. *Clin Gastroenterol Hepatol*. 2016;14(6):875–885. doi:10.1016/j.cgh.2015.10.025

32. Wilson K, Thompson J, Walker M, et al. Somatic symptom disorder in gastrointestinal practice: diagnostic utility of PHQ-15 and Whiteley Index. *J Psychosom Res*. 2021;142:110353. doi:10.1016/j.jpsychores.2020.110353

33. Understanding and Managing Somatoform Disorders: A Guide for Physicians. Anton Scamvougeras. and Andrew Howard. . Vancouver: AJKS Publishing; 2018. ISBN 978-0-9952056-1-1.

34. Henningsen P, Zipfel S, Sattel H, Creed F. Management of functional somatic syndromes and bodily distress. Psychother Psychosom. 2018;87(1):12-31. https://doi.org/10.1159/000484413

35. Liu J, Gill NS, Teodorczuk A, Li ZJ, Sun J. The efficacy of cognitive behavioural therapy in somatoform disorders and medically unexplained physical symptoms: A meta-analysis of randomized controlled trials. J Affect Disord. 2019;245:98-112. https://doi.org/10.1016/j.jad.2018.10.114

36. Nimnuan C, Hotopf M, Wessely S. Medically unexplained symptoms: an epidemiological study in seven specialities. J Psychosom Res. 2001;51(1):361-367. https://doi.org/10.1016/s0022-3999(01)00223-9.

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