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A THEORETICAL MODEL OF DIFFERENTIAL PSYCHOCORRECTION IN PANIC DISORDER

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ABSTRACT

Current international guidelines recommend cognitive behavioral therapy (CBT) as the first-line treatment for panic disorder; however, uniform application of a single therapeutic approach fails to account for the heterogeneity of etiological factors underlying the disorder in different patients. This paper proposes an original theoretical model of differential psychocorrection for panic disorder, in which three distinct etiological profiles — cognitive misappraisal, early maladaptive schemas, and neurophysiological predisposition — are systematically matched to three evidence-based therapeutic approaches: CBT, schema therapy, and virtual reality (VR) therapy, respectively. Drawing on a review of international theoretical and empirical literature, the paper presents the conceptual foundations, diagnostic criteria for etiological classification, and the rationale for therapy selection within this model. The proposed model aligns with the emerging paradigm of precision psychiatry and addresses a significant gap in both the international literature and clinical practice in Central Asia.

Keywords: panic disorder, differential psychocorrection, etiological model, cognitive behavioral therapy, schema therapy, virtual reality therapy, precision psychiatry, catastrophic misappraisal, early maladaptive schemas.

1. INTRODUCTION

Panic disorder (PD) is one of the most prevalent and functionally impairing anxiety disorders, affecting approximately 2–3% of the global population across all age groups and cultural contexts (WHO, 2022). Despite the well-established efficacy of cognitive behavioral therapy (CBT) as the current gold-standard treatment — producing panic-free outcomes in approximately 85% of treated patients (Barlow et al., 2000) — a substantial subgroup of patients does not respond adequately to standard CBT protocols. Residual symptoms, high relapse rates in certain subgroups, and the limited applicability of uniform protocols to clinically heterogeneous presentations represent persistent challenges in the field.

One theoretically compelling explanation for this variability lies in the etiological heterogeneity of panic disorder itself. Panic disorder does not arise from a single, universal cause; rather, its development and maintenance involve complex interactions among cognitive, developmental, and neurobiological factors that vary substantially across individuals. A patient whose panic attacks are maintained primarily by catastrophic misinterpretation of bodily sensations presents a fundamentally different clinical picture from one whose panic is rooted in deeply

ingrained early maladaptive schemas formed in childhood, or one whose hyperreactive neurophysiological alarm system drives disproportionate panic responses.

Despite this recognized heterogeneity, the prevailing clinical approach remains largely undifferentiated: the same CBT protocol is applied to patients regardless of the specific etiological factors driving their disorder. This "one-size-fits-all" approach is increasingly challenged by the emerging paradigm of precision psychiatry — the principle that treatment should be individualized based on patient-specific biological, psychological, and social characteristics (McIntyre et al., 2023; Guest et al., 2024).

This paper aims to address this gap by proposing an original theoretical model of differential psychocorrection in panic disorder, in which patients are classified into etiological subgroups and matched to the therapeutic approach most directly targeting their primary maintaining mechanism. The model integrates the Clark (1986) cognitive model, Young's (1994) schema theory, and Barlow's (2004) neurobiological model into a unified classification and treatment-selection framework, with CBT, schema therapy, and VR therapy serving as the corresponding interventions.

2. THEORETICAL BACKGROUND

2.1. The Problem of Etiological Heterogeneity in Panic Disorder

The scientific literature on panic disorder encompasses three well-established, partially competing theoretical models, each of which identifies a distinct primary etiological mechanism. Clark's (1986) cognitive model proposes that panic disorder is maintained by the catastrophic misinterpretation of benign bodily sensations — the tendency to interpret normal physiological arousal (tachycardia, shortness of breath, dizziness) as signs of imminent catastrophe. Barlow's (1988; 2004) neurobiological model emphasizes the role of biological vulnerability, anxiety sensitivity, and conditioned fear responses centered on interoceptive cues. Young's (1994; 2003) schema theory, in contrast, locates the etiological roots of chronic anxiety disorders — including panic disorder — in early maladaptive schemas (EMS) formed during childhood in response to unmet core emotional needs.

Each model has independently generated a corresponding therapeutic approach: Clark's model underpins the Clark panic-focused CBT protocol; Barlow's model informs interoceptive exposure-based interventions; and Young's schema theory underlies schema therapy with its techniques of imagery rescripting, empty chair work, and mode work. Critically, these three approaches have largely been studied and applied in isolation, without a systematic framework for matching the intervention to the specific etiological profile of the individual patient.

A comparative study by Kwak and Lee (2015) found that patients with OCD and panic disorder show distinct patterns of early maladaptive schema activation, suggesting that etiological subtyping within diagnostic categories is both theoretically meaningful and clinically feasible. Similarly, Kyriakoulis and Kyrios

(2023) demonstrated in a narrative review that biological and cognitive theories explain partially non-overlapping aspects of panic disorder, supporting the case for etiological differentiation in clinical practice.

2.2. The Precision Psychiatry Paradigm

The concept of precision psychiatry — adapting treatment to the individual's specific biological, psychological, and social profile — has gained considerable momentum in recent years (McIntyre et al., 2023). In psychotherapy research, this has been operationalized through the stratified care model, in which treatment intensity and type are matched to patient characteristics such as symptom severity, comorbidity, and response history (Papola et al., 2022).

The proposed model of differential psychocorrection in panic disorder represents an application of this precision paradigm to the specific domain of treatment selection. Rather than stratifying by severity alone, the model stratifies by etiological profile, matching each patient to the therapeutic approach most directly targeting their primary maintaining mechanism. This approach is consistent with the theoretical principle that therapeutic change is maximally efficient when the treatment mechanism directly addresses the disorder-specific maintenance factor.

3. THE DIFFERENTIAL PSYCHOCORRECTION MODEL

3.1. Three Etiological Profiles

The proposed model identifies three distinct etiological profiles in panic disorder, each defined by a primary maintaining mechanism, a corresponding theoretical model, and a clinical presentation pattern.

Profile 1 — Cognitive Misappraisal Etiology: The primary maintaining mechanism is the catastrophic misinterpretation of bodily sensations, as described by Clark (1986). Clinically, these patients present with prominent cognitive distortions about the meaning of physiological arousal, high scores on the Agoraphobic Cognitions Questionnaire (ACQ), and elevated cognitive subscale scores on the Anxiety Sensitivity Index-3 (ASI-3). There is typically no significant childhood trauma history and no active early maladaptive schemas on the Young Schema Questionnaire (YSQ-S3). Avoidance behavior is present but secondary to cognitive misappraisal.

Profile 2 — Schema-Based Etiology: The primary maintaining mechanism is the activation of early maladaptive schemas formed in childhood — most prominently the Vulnerability to Harm schema, Functional Incompetence/Dependency schema, and Emotional Inhibition schema (Hedley et al., 2001; Kwak & Lee, 2015). These patients typically present with a history of unmet childhood emotional needs, chronic or recurrent panic disorder that has been resistant to standard CBT, and elevated scores on multiple YSQ-S3 schema domains. Cognitive misappraisal is present but secondary to deeper schematic patterns.

Profile 3 — Neurophysiological Etiology: The primary maintaining mechanism is a hyperreactive neurophysiological alarm system, characterized by elevated interoceptive sensitivity, low threshold for physiological arousal, and strong conditioned fear responses to bodily sensations (Barlow, 2004). Clinically, these patients present with elevated physical subscale scores on ASI-3, high baseline physiological reactivity, and panic attacks that appear to arise with minimal or no cognitive antecedent. Schema activation and catastrophic cognition are minimal.

3.2. Diagnostic Classification Protocol

Etiological classification within the proposed model proceeds through a structured, multi-instrument assessment protocol. All patients first receive a DSM-5-TR diagnosis of panic disorder using the Structured Clinical Interview (SCID-5-CV) and complete the Panic Disorder Severity Scale (PDSS) as a baseline severity measure.

Etiological profiling is then conducted using three instruments administered in combination. The YSQ-S3 identifies the presence and intensity of early maladaptive schemas. The ASI-3, with its three subscales (physical, cognitive, and social concerns), distinguishes between primarily interoceptive/biological and primarily cognitive anxiety sensitivity. The ACQ measures the frequency and conviction of catastrophic misappraisals. The pattern of elevations across these instruments determines etiological profile assignment according to the dominant mechanism principle: the profile whose corresponding instruments show the most elevated scores is assigned as primary.

3.3. Therapy Matching Rationale

The core theoretical proposition of the differential psychocorrection model is that treatment efficacy is maximized when the therapeutic mechanism directly targets the primary etiological maintaining factor. This proposition is grounded in the theoretical principle of mechanism-target alignment, which holds that therapeutic change is most efficient when the intervention's active mechanism corresponds to the disorder's primary maintenance mechanism.

For Profile 1 (cognitive misappraisal etiology), CBT is the indicated treatment because its primary mechanisms — cognitive restructuring, behavioral experiments, and interoceptive exposure — directly target catastrophic misinterpretation, the primary maintaining factor. Clark's panic-focused CBT protocol (16 sessions) provides the standardized framework, with Socratic dialogue and behavioral experiments as the core cognitive change mechanisms.

For Profile 2 (schema-based etiology), schema therapy is indicated because its mechanisms — imagery rescripting, empty chair work, and mode work — operate at the level of early maladaptive schemas, directly modifying the deeper cognitive-emotional structures that maintain the disorder. Standard CBT is insufficient for this profile because it addresses surface-level cognitions without reaching the underlying schematic structures. Schema therapy's limited reparenting

relationship additionally addresses the relational deficit that contributed to schema formation. Peeters et al. (2022), in a systematic review of schema therapy for anxiety disorders, found preliminary evidence of beneficial effects on both disorder-specific symptoms and early maladaptive schemas.

For Profile 3 (neurophysiological etiology), VR therapy is indicated because its primary mechanism — graduated immersive exposure to physiological arousal-inducing contexts — directly targets conditioned interoceptive fear through habituation and inhibitory learning (Craske et al., 2022). VR enables precise, controlled exposure to the specific physiological and environmental cues that trigger panic responses, without the logistical constraints and unpredictability of in vivo exposure. Recent meta-analytic evidence confirms VR therapy's efficacy for panic disorder and agoraphobia (Gonçalves et al., 2025).

Table 1. The Differential Psychocorrection Model: Etiological Profiles and Matched Interventions

Criterion	Profile 1 (CBT)	Profile 2 (Schema Therapy)	Profile 3 (VR Therapy)
Primary mechanism	Catastrophic misappraisal	Early maladaptive schemas	Neurophysiological hyperreactivity
Theoretical basis	Clark (1986)	Young (1994; 2003)	Barlow (2004)
Childhood history	Not prominent	Central — unmet needs	Variable
Core technique	Cognitive restructuring	Imagery rescripting	Graduated VR exposure
Change mechanism	Belief disconfirmation	Schema modification	Inhibitory learning

Source: Clark (1986); Young (1994); Barlow (2004); Peeters et al. (2022); Gonçalves et al. (2025); compiled by author.

4. SCIENTIFIC CONTRIBUTION AND NOVELTY

The proposed differential psychocorrection model makes several distinct contributions to the existing scientific literature. First, it provides the first systematic theoretical framework for matching panic disorder patients to one of

three evidence-based therapeutic approaches based on etiological profile, rather than clinical severity or symptom count alone. While individual models (Clark, Young, Barlow) have generated distinct therapeutic protocols, no prior theoretical work has integrated these models into a unified differential treatment-selection framework.

Second, the model operationalizes precision psychiatry principles in the domain of psychological treatment for panic disorder. The dominant etiological mechanism principle offers a theoretically grounded, clinically applicable decision rule that goes beyond the current trial-and-error approach to treatment selection.

Third, the model generates empirically testable predictions. If the model is theoretically sound, patients assigned to etiologically matched therapy should demonstrate superior outcomes compared to patients receiving etiologically mismatched therapy. This prediction forms the basis of the planned empirical study in which the model will be tested through a quasi-experimental design with three etiologically defined treatment groups.

Fourth, the model addresses a significant gap in the literature for Central Asian populations. A systematic search of PubMed, Scopus, and Web of Science databases identified no randomized controlled trial or quasi-experimental study examining etiology-based differential treatment selection for panic disorder conducted in Central Asia or with Uzbek-speaking populations. The present model provides the theoretical framework for filling this gap through original empirical research.

5. LIMITATIONS AND FUTURE DIRECTIONS

The proposed model carries several important theoretical and methodological limitations that must be acknowledged. First, etiological profiles in clinical populations are rarely pure; most patients with panic disorder present with features of multiple etiological profiles simultaneously. The dominant mechanism principle addresses this issue by assigning treatment based on the primary profile while acknowledging secondary factors, but the theoretical basis for this prioritization requires further empirical validation.

Second, the assessment instruments proposed for etiological classification (YSQ-S3, ASI-3, ACQ) have not been validated in Uzbek-speaking populations. Psychometric validation of these instruments in the local clinical context is a prerequisite for the empirical testing of the model and represents a distinct research contribution in itself.

Third, the model currently identifies three etiological profiles, but future research may identify additional profiles — such as a trauma-based profile — that would require additional therapeutic matching. The model is therefore presented as a developmental framework subject to empirical revision rather than a final taxonomy.

Future research should test the model's core prediction through a quasi-experimental controlled study in which patients are etiologically classified and randomly assigned to matched versus mismatched therapy conditions. Secondary

research questions should examine whether etiological profile moderates treatment outcomes within each therapy type, and whether etiological profiling improves treatment efficiency compared to standard clinical assessment.

6. CONCLUSION

This paper has proposed a theoretically grounded model of differential psychocorrection in panic disorder, systematically linking three etiological profiles to three evidence-based therapeutic approaches. The following conclusions are drawn:

- The etiological heterogeneity of panic disorder — spanning cognitive misappraisal, early maladaptive schemas, and neurophysiological hyperreactivity — provides a theoretically sound basis for differential treatment selection.
- CBT, schema therapy, and VR therapy each possess distinct therapeutic mechanisms that directly target one of the three identified etiological profiles, maximizing mechanism-target alignment.
- The proposed model operationalizes precision psychiatry principles in psychotherapy and generates empirically testable predictions that can be tested through a quasi-experimental clinical study.
- The model addresses a significant gap in both the international theoretical literature and clinical practice in Central Asia, where etiology-based differentiation of panic disorder treatment has not previously been systematically studied.

The proposed model is presented as a theoretical framework requiring empirical validation, and the planned clinical study is designed to provide the first direct test of its core predictions in an Uzbek clinical population.

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