Primary psychogenic depression: a pathogenetic model based on impaired translation of the mobilizing impulse into action

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Abstract

Objective The aim of this paper was to develop a theoretical, clinical, and psychobiological model of the pathogenesis of primary psychogenic depression and to substantiate an etiological approach to psychotherapy.

Method A theoretical analysis of psychodynamic, neurobiological, and clinical studies of depression was conducted, together with an analysis of clinical observations obtained in the course of applying Integrative Causal Psychotherapy to the treatment of depressive disorders.

Results The proposed model identifies the central pathogenetic mechanism of primary psychogenic depression is the chronic inability to translate into action the mobilizing impulse arising during attempts to satisfy fundamental psychological needs. In this context, chronic inhibition of action may contribute to fronto-limbic conflict and mesolimbic dopaminergic hypoactivity, potentially promoting learned helplessness, diminished agency, and the development of a persistent depressive symptom complex. Based on this model, key therapeutic targets are identified and a mechanism-based psychotherapeutic approach is proposed, aimed at restoring access to the mobilizing impulse and the capacity to act.

Conclusions The proposed model integrates psychodynamic and neurobiological perspectives on depression within a unified causal framework and may serve as a theoretical basis for the development of targeted psychotherapeutic interventions for primary psychogenic depression.

Keywords: depression; pathogenesis; psychotherapy; aggression; mobilizing impulse; dopamine.

Introduction

Depressive disorders are among the most prevalent mental illnesses and are among the leading causes of decreased quality of life, impaired social functioning, and occupational disability worldwide. According to the World Health Organization, approximately 280 million people suffer from depression, accounting for about 3.8% of the global population [1]. A substantial proportion of patients experience recurrent depressive episodes rather than a single depressive episode. The risk of recurrence increases with each subsequent episode, and a substantial proportion of the burden of depression is attributable to its chronic and recurrent course [2, 3].

Despite significant advances in psychopharmacology and psychotherapy, the prevention of depressive relapse remains a major challenge. Meta-analyses demonstrate that even after successful treatment the risk of recurrence remains high. Following cognitive behavioral therapy, relapse or recurrence of unipolar depression occurs in approximately 29% of patients within the first year and in more than 54% within two years after treatment completion [4]. After discontinuation of antidepressant therapy, relapse during the subsequent year develops in approximately 56% of patients [5]. Contemporary approaches to relapse prevention in depression demonstrate limited effectiveness and do not eliminate the risk of recurrent depressive episodes [6].

This situation points to a fundamental problem in contemporary depression treatment: despite the large number of therapeutic approaches and theoretical attempts to explain the etiology of depression, there is still no widely accepted causal

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model capable of explaining the core mechanism underlying depressive symptoms and providing a basis for mechanism-based treatment aimed at reducing relapse risk.

An additional challenge is the heterogeneity of depressive disorders themselves. Contemporary diagnostic classifications include a broad range of clinical entities within the depressive spectrum, including recurrent depressive disorder, depressive episode, dysthymia, bipolar affective disorder, mixed anxiety-depressive states, adjustment disorders with depressive symptoms, and a number of other conditions. This suggests that depressive disorders are etiologically heterogeneous and makes the development of a universal model equally applicable to all depressive conditions unlikely.

The aim of the present article is to propose a theoretical and clinical model of the pathogenesis of a specific type of depressive disorder — primary psychogenic depression — and to substantiate a mechanism-based approach to psychotherapy aimed at eliminating the presumed central pathological process underlying this type of depression. The proposed concept was developed within the Integrative Causal Psychotherapy approach* and integrates psychodynamic, neurobiological, and clinical perspectives on the pathogenesis of depressive disorders within a unified causal framework.

Key concepts and definitions

The model conceptualizes depressive disorders as etiologically heterogeneous conditions that can be divided into two groups and four types according to their causal mechanisms.

Group I. Somatogenic depressions — depressions caused by pathological processes in the body.

- **Primary somatogenic (endogenous) depressions** are associated with neurotransmitter dysfunctions.
- **Secondary somatogenic depressions** are associated with pathological conditions of organs and physiological systems that affect neurotransmitter functioning, including vascular, metabolic, chronic pain, substance-induced, and other pathological processes.

Group II. Psychogenic depressions — depressions arising as a maladaptive psychological response and not primarily driven by somatic pathology.

- **Primary psychogenic depressions** develop directly in response to life circumstances.
- **Secondary psychogenic depressions** develop as comorbid conditions associated with other mental disorders (personality disorders, posttraumatic disorders, and related conditions).

This article examines the pathogenesis of primary psychogenic depression (hereinafter referred to as PPD). In the proposed model, PPD is conceptualized as an independent disorder arising as a psychological response to life circumstances and not caused by somatic pathological processes or pre-existing mental disorders.

Thus, the proposed model does not claim to explain the pathogenesis of depression as a whole, but rather describes one specific causal type of depressive disorder. The identification of primary psychogenic depression as a distinct etiological category is considered necessary for the development of differentiated mechanism-based psychotherapeutic interventions.

Theoretical foundations

The proposed model of primary psychogenic depression draws on psychodynamic theories of depression, as well as contemporary research on the neurobiology of depressive disorders, motivation, and psychophysiological regulation under conditions of chronic stress.

One of the early theoretical foundations of the model derives from the works of Sigmund Freud and Karl Abraham, who conceptualized depression as a condition associated with difficulties in processing aggressive impulses and redirecting them inward [7, 8]. In *Mourning and Melancholia,* Freud described melancholia as a process in which aggressive impulses originally directed toward an external object become redirected against the self [7]. Abraham further developed this concept by associating depressive states with ambivalence, object loss, and self-directed aggression [8]. Unlike classical psychoanalytic models, however, the present article conceptualizes aggression not as a destructive drive but as a natural adaptive mechanism — the affective component of the mobilizing impulse that enables the transition from motivation to action, facilitates overcoming obstacles, and supports the protection of personal boundaries and the satisfaction of significant needs.

* Integrative Causal Psychotherapy is a psychotherapeutic approach developed by Yu. Tor that focuses on identifying the core pathogenic mechanisms underlying mental disorders and designing therapeutic protocols aimed at modifying these mechanisms.

An important theoretical foundation of the model is provided by contemporary concepts concerning the role of the mesolimbic dopaminergic system in motivation, agency, and anhedonia. Research demonstrates that the nucleus accumbens, ventral striatum, and associated frontal structures are involved not only in the experience of pleasure but primarily in mechanisms of action initiation, goal evaluation, and reward anticipation [9, 10]. Dysfunction of these systems is considered one of the key mechanisms underlying anhedonia, psychomotor retardation, and reduced motivation in depression [11].

Research on motivation and learned helplessness also informs the proposed model. The work of M. Seligman demonstrated that repeated experiences of inability to influence a situation lead to the formation of a stable state of helplessness accompanied by reduced initiative, diminished motivation, and impaired capacity for active behavior [12]. Subsequent studies demonstrated associations between learned helplessness and impairments in dopaminergic reinforcement, motivated behavior, and reward anticipation [13].

Another important theoretical foundation of the model is provided by contemporary research on the neurobiology of chronic stress and emotion regulation. Prolonged suppression of emotional responses has been shown to be associated with dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, amygdala hyperactivation, fronto-limbic dysregulation, and reduced neuroplasticity [14, 15]. Chronic stress is also associated with reduced brain-derived neurotrophic factor (BDNF) expression and impaired dopaminergic transmission, which are considered pathogenetic mechanisms underlying motivational and emotional deficits in depression [16].

Psychological mechanism of the pathogenesis of PPD

Primary psychogenic depression is conceptualized as a dynamic pathological process arising from chronic suppression of the mobilizing impulse, particularly its affective component — aggression. The mobilizing impulse is understood as an internal drive toward action that emerges in response to frustration of core psychological needs, is accompanied by psychophysiological mobilization, and is directed toward overcoming obstacles and enabling effective action.

Unlike models that conceptualize depression primarily as a disorder of cognition or motivation, the proposed model assumes that the central pathogenetic mechanism underlying PPD is a disruption of the translation of an internal mobilizing impulse into action aimed at resolving the chronic frustration of core psychological needs.

1. Blocking the satisfaction of existentially significant needs and the execution of related actions

The initial stage of the pathogenesis is characterized by chronic frustration of basic needs under conditions in which individuals perceive themselves as unable to take the actions necessary to preserve autonomy, psychological integrity, and control over significant life circumstances.

Unlike frustration arising from unmet situational or secondary desires, the blocking of existentially significant needs is experienced as a threat to psychological survival and is accompanied by the activation of aggression as a mobilizing affect directed toward overcoming the obstacle.

The pathogenic factor is not the stressful or adverse life situation itself, but its subjective experience as a chronic inability to satisfy critically important needs, maintain autonomy, or alter significant life circumstances. Such situations may include prolonged controlling or dependent relationships, chronic psychological pressure, emotional suppression, restrictions on social autonomy, and other persistent forms of restriction of personal agency.

Markers of this stage include:

- chronic frustration of needs experienced as essential for identity, autonomy, and psychological integrity;
- a persistent sense of diminished autonomy and inability to influence one's life circumstances;
- subjective experience of threat to core aspects of psychological existence.

2. Activation of aggression as a mobilizing affect

The key condition for the transition to the next stage is the persistence of the need despite chronic inability to satisfy it. This state activates aggression as a mobilizing mechanism directed toward overcoming the obstacle.

At the clinical level, the mobilization stage may manifest as:

- the emergence of an urge to change a subjectively intolerable situation;
- a sense of internal mobilization;
- the emergence of psychophysiological tension.

3. Repression of aggression and inhibition of the mobilizing impulse

However, in the presence of stable inhibitions against experiencing and expressing aggression, the mobilizing impulse and its affective component become subject to systematic suppression or repression. The sources of such inhibitions may include early introjects, fear of object loss, prior experiences of punishment for expressions of

aggression, strong dependence on external approval, or internalized beliefs regarding the unacceptability of conflict.

Clinical markers of the stage of suppression of the mobilizing impulse include:

- avoidance of confrontation and open conflict;
- rationalized explanations for why the situation cannot be resolved;
- reduced energy and mood associated with cortical inhibition of sympathetic activation.

4. Development of learned helplessness

The transition to the next stage is mediated by repeated experiences of failure to translate the mobilizing impulse into action. The individual repeatedly encounters situations in which an internal impulse toward action arises, yet its translation into action remains blocked. Over time, this leads to the formation of a stable sense that one's own actions are ineffective and contributes to the development of learned helplessness [13, 16].

At this stage, the following manifestations are observed:

- the development of a belief that one's own actions are ineffective in changing a situation of persistent frustration;
- the abandonment of attempts to change the situation or their rapid cessation;
- a subjective sense of losing the ability to influence events.

5. Development of depressive syndrome

Chronic blocking of the transition from mobilization to action results in an inability to complete the adaptive behavioral cycle. As a consequence, unresolved arousal that is neither behaviorally expressed nor affectively processed becomes consolidated as persistent internal tension and progressive depletion of regulatory and motivational resources.

At the psychological level, a sustained state of unresolved internal activation develops, accompanied by reduced agency, loss of behavioral initiative, and consolidation of helplessness. The prolonged absence of successful action and of reinforcement of behavioral efficacy contributes to the generalized expectation that one's efforts are ineffective.

At the neurobiological level, dysfunction emerges within motivational, reward, and stress-regulatory systems, including mesolimbic dopaminergic hypoactivity, reduced reward sensitivity, and chronic dysregulation of the stress response associated with HPA axis hyperactivation. A functional imbalance in prefrontal-limbic regulation develops, characterized by chronic hyperactivation of cortical inhibitory mechanisms that suppress the mobilizing impulse and persistently prevent its translation into action.

Clinically, this stage is manifested by:

- chronic diffuse psychophysiological tension;
- persistent exhaustion accompanied by an impaired capacity for recovery;
- anhedonia, reduced motivational energy, and decreased self-worth.

A self-perpetuating pathological cycle develops in which the chronic inability to translate the mobilizing impulse into action reduces agency and reinforces helplessness, which in turn further restricts active coping and contributes to the consolidation of the depressive syndrome into a persistent psychopathological state.

Neurobiological mechanism of the pathogenesis of primary psychogenic depression

Within the proposed model, psychological and neurobiological processes are viewed as interconnected components of a unified causal mechanism operating as an integrated dynamic system. They are discussed separately here solely for analytical clarity. Chronic inability to meet core psychological needs and systematic suppression of the mobilizing impulse are accompanied by simultaneous alterations in motivational reinforcement systems, emotional regulation, and stress adaptation mechanisms.

Key neurobiological mechanism: fronto-limbic conflict and suppression of the mobilizing impulse

The central neurobiological mechanism of PPD is a chronic fronto-limbic conflict associated with systematic suppression of the emotional and behavioral components of the mobilizing impulse. It is assumed that stable inhibitions against experiencing and expressing aggression enhance cortical inhibition of emotional activation and neural systems involved in action initiation and behavioral control, leading to a chronic fronto-limbic conflict between persistent mobilization and the inability to translate it into action [15].

Under normal conditions, emotional and physiological mobilization culminates in action directed toward overcoming an obstacle or restoring disrupted equilibrium. However, in the presence of aggression-inhibiting attitudes, the mobilizing impulse arising in a conflict situation undergoes cortical inhibition and remains unresolved both emotionally and behaviorally. As a result, a state of unresolved motivational activation develops, in which emotional activation systems remain engaged while its translation into action remains blocked.

Over time, this inhibitory state may lead to consolidation of neural patterns of passive responding, in which activation associated with the need for action becomes automatically suppressed even prior to behavioral expression. As a result, the gap between the subjective experience of the need to change the situation and the capacity to initiate corresponding action progressively increases. Clinically, this may manifest as chronic internal tension, difficulty recognizing one's own needs, conflict avoidance, psychomotor inhibition, experiences of helplessness, and a perceived inability to influence ongoing events.

An additional role in maintaining this condition is presumably played by the basal ganglia and associated mechanisms of action inhibition. Chronic suppression of the mobilizing impulse may be accompanied by increased activity of the indirect inhibitory pathway of the basal ganglia, resulting in enhanced inhibition of thalamocortical transmission [18, 19]. As a consequence, the impulse toward action fails to result in overt motor behavior and remains confined to subcortical-striatal circuits, thereby sustaining chronic internal tension and psychomotor retardation.

Indirect support for the proposed model may be found in experimental studies of learned helplessness and chronic stress in animals. These studies demonstrate that repeated inability to engage in avoidance or defensive behavior is accompanied by the development of behavioral passivity, reduced motivational activity, and states resembling depressive behavior [12, 17]. These observations are consistent with the assumption that chronic suppression of the mobilizing impulse may gradually impair systems responsible for action initiation and motivational reinforcement.

Mesolimbic dopaminergic hypoactivity and disruption of the action–reward system

One of the presumed key consequences of chronic suppression of the mobilizing impulse is the development of mesolimbic dopaminergic hypoactivity. In contemporary neuroscience, dopamine is regarded not merely as a neurotransmitter of pleasure, but as a central component of the systems involved in reward prediction and action initiation. Dopaminergic neurons of the ventral tegmental area respond primarily to the discrepancy between expected and actual action outcomes — the so-called reward prediction error. Under normal conditions, the mobilizing impulse proceeds through a sequential cycle: emergence of a fundamental psychological need, motivational mobilization, action, and dopaminergic reinforcement of successful action outcomes.

During the premorbid stage of PPD, this cycle becomes systematically disrupted: an impulse toward action arises, yet it cannot be translated into action due to chronic suppression and inhibition of action. As a result, the action cycle remains incomplete, and dopaminergic reinforcement of action does not occur. Repeated inability to perform subjectively significant actions may gradually alter dopaminergic reward prediction dynamics. The brain begins to anticipate failure even before action is initiated. Consequently, basal dopaminergic activity decreases, goals lose motivational salience, and the capacity to initiate action becomes impaired even when awareness of its necessity is preserved [20].

From this perspective, anhedonia in PPD may be conceptualized not merely as loss of the capacity to experience pleasure, but as a disruption of the action–reward system in which the individual no longer experiences action as likely to be effective [21]. This leads to reduced initiative, psychomotor retardation, experiences of helplessness, and a perceived inability to initiate activity.

Mesolimbic dopaminergic hypoactivity is viewed not as a primary biochemical deficit but as a consequence of a chronically incomplete mobilization cycle, in which repeated failure to translate the mobilizing impulse associated with fundamental psychological needs into action gradually leads to a weakening of motivational reinforcement mechanisms and the development of a persistent depressive state.

Chronic activation of the stress-response system and HPA axis dysregulation

The third key component of the neurobiological mechanism of PPD is chronic activation of the stress-response system associated with prolonged maintenance of incomplete mobilization. Chronic inability to carry out meaningful action in the presence of persistent internal tension is accompanied by sustained activation of the sympathetic nervous system and the HPA axis [14].

Prolonged elevation of cortisol levels and persistent activation of stress-response systems gradually lead to a state of allostatic overload accompanied by impaired neuroplasticity, reduced BDNF signaling and depletion of adaptive resources [16]. Simultaneously, chronic activation of the stress-response system maintains underlying anxiety, internal tension, and a persistent sense of incomplete recovery.

It is proposed that fronto-limbic inhibition, together with mesolimbic dopaminergic hypoactivity and chronic activation of the stress-response system, forms a stable symptom complex characteristic of depression.

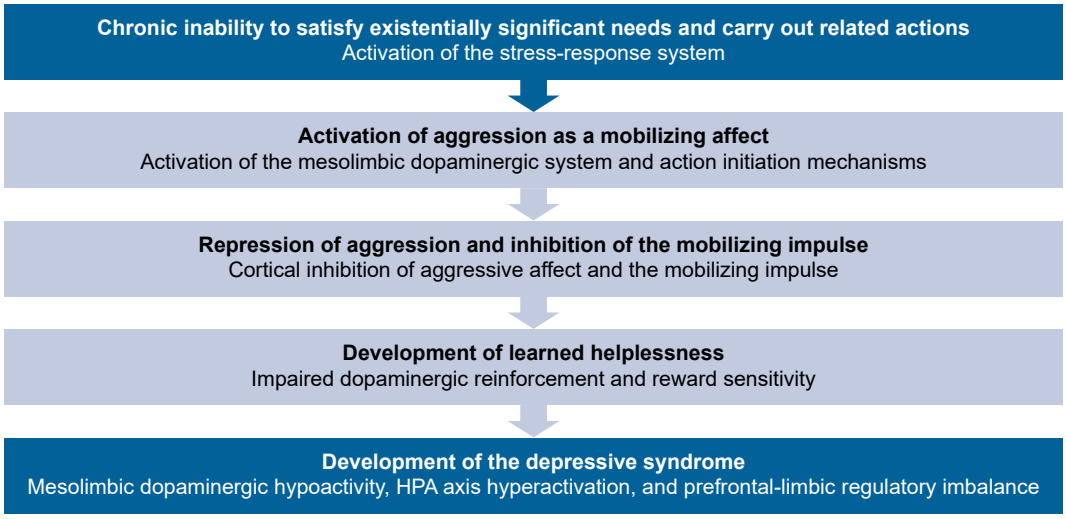


Figure 1. Model of the pathogenesis of primary psychogenic depression

Thus, from a neurobiological perspective, PPD may be conceptualized as a state of chronic suppression of the mobilizing impulse in which systems involved in emotional activation, action initiation, and the stress response remain chronically activated, while translation of the mobilizing impulse into action remains blocked. Persistence of such a state gradually leads to the formation of fronto-limbic conflict, mesolimbic dopaminergic hypoactivity, and chronic stress dysregulation underlying anhedonia, psychomotor retardation, reduced agency, and the sense of inability to initiate action. As the process becomes chronic, this pathological system acquires a self-sustaining character.

Causally targeted psychotherapy of PPD

The proposed pathogenetic model of primary psychogenic depression makes it possible to identify the core pathological mechanism of the disorder and define therapeutic targets whose modification may disrupt the core pathological cycle maintaining this type of depression. The core pathogenetic mechanism of PPD is conceptualized as chronic suppression of the mobilizing impulse arising in response to chronic inability to fulfill basic psychological needs and initiate meaningful action. Accordingly, targeted psychotherapeutic intervention should aim to eliminate the mechanisms that maintain chronic inhibition of action.

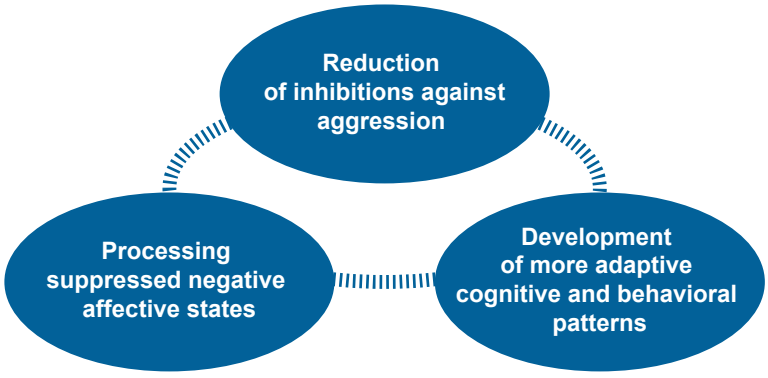


Figure 2. Key therapeutic targets of primary psychogenic depression according to the pathogenetic model

From the perspective of the proposed model, targeted interventions involve helping patients recognize aggressive feelings as adaptive and psychologically meaningful, as well as removing inhibitions against their experience and expression, thereby restoring agency. Reestablishing contact with the mobilizing impulse creates the conditions for successful action, an increased sense of self-efficacy, and a restored ability to shape one's life circumstances.

It is assumed that such an intervention simultaneously affects both the psychological and neurobiological mechanisms of PPD. At the psychological level, this is accompanied by a reduction in the internal conflict between the mobilizing impulse and the inability to translate it into action. At the neurobiological level, restoration of the ability to translate mobilizing impulses into action may potentially contribute to a reduction in chronic fronto-limbic inhibition, attenuation of stress-system hyperactivation, and normalization of dopaminergic reward processes associated with effective action.

A second important therapeutic target is the processing of chronically suppressed negative affective states associated with inhibition of aggressive impulses and the experience of learned helplessness.

Another therapeutic direction is the development of more adaptive cognitive and behavioral patterns that become accessible after disruption of the core pathological process — suppression of the mobilizing impulse.

Thus, therapeutic improvement is associated with patients regaining the capacity to engage in meaningful action and disengaging from the pathological cycle of chronically suppressed mobilization. Schematically, this process may be represented as follows:

ACCEPTANCE OF AGGRESSIVE IMPULSES AS LEGITIMATE → DEVELOPMENT OF THE ABILITY TO TRANSLATE THE MOBILIZING IMPULSE INTO ACTION → RESTORATION OF PERSONAL AGENCY → SUSTAINED REMISSION OF DEPRESSIVE SYMPTOMS

This sequence reflects disruption of the pathological cycle: recognizing aggression as adaptive restores access to the mobilizing impulse, thereby enabling effective action and promoting normalization of dopaminergic reward processing associated with motivation, subjective self-efficacy, and the capacity to experience satisfaction.

Clinical observations in the context of the model

Clinical observations obtained during the application of a 20-session protocol of Integrative Causal Psychotherapy in the treatment of depression generally correspond with the proposed model of PPD [22].

In patients, depressive symptoms were associated with pronounced inhibitions against experiencing and expressing aggression, fear of conflict, chronic suppression of anger and resistance, subjective inability to defend personal boundaries, and experiences of internal helplessness. The therapeutic dynamics observed during treatment corresponded to the proposed causal model of PPD: reduction of inhibitions against aggression was accompanied by a gradual restoration of the capacity for meaningful action and personal agency.

At the initial stages of therapy, patients predominantly demonstrated anhedonia, reduced initiative, psychomotor retardation, feelings of helplessness, and difficulties in recognizing their own aggressive emotions. A substantial part of the therapeutic work was aimed at helping patients recognize aggression as an adaptive mobilizing affect and revise beliefs that inhibited the experience and expression of aggression.

As inhibitions against aggression weakened during therapy, patients initially demonstrated increased emotional activation, emotional engagement, renewed motivation, and an increasing tendency to take an active role in shaping their life circumstances. During subsequent stages of therapy, a gradual restoration of agency was observed: patients reported increased self-confidence, improved ability to make decisions, defend personal boundaries, carry out meaningful actions, and take a more active role in shaping life circumstances. Simultaneously, the severity of anhedonia, helplessness, and depressive symptoms decreased. Such dynamics may reflect restoration of the previously suppressed mobilizing impulse.

In addition to remission of depression, psychotherapy outcomes included statistically significant improvements in self-confidence, professional functioning, enjoyment of life, and environmental mastery*, consistent with the hypothesis that the capacity for mobilized action had been restored [23].

* Environmental mastery refers to a dimension of psychological well-being in Ryff's model describing the perceived ability to effectively manage life circumstances.

Additional indirect support for the theoretical and clinical validity of the proposed model may be found in the absence of depressive relapses among patients who achieved remission during the one-year follow-up period [22]. From the perspective of the proposed concept, this may be explained by the fact that psychotherapeutic intervention aimed at reducing inhibitions against aggression directly targets the central pathogenetic mechanism of PPD — suppression of the mobilizing impulse. It is assumed that after patients become aware of and process these inhibitions, such inhibitions are unlikely to re-emerge because patients develop a stable understanding of aggression as an adaptive mechanism supporting self-protection, autonomy, and the satisfaction of core psychological needs. Such psychotherapy may not only reduce symptoms but also decrease the likelihood of reactivation of the core pathological mechanism underlying PPD.

Discussion

The proposed model conceptualizes primary psychogenic depression as a disorder associated with chronic suppression of the mobilizing impulse arising in response to the inability to fulfill core psychological needs and engage in meaningful action. Unlike approaches that conceptualize depression primarily as a state of motivational deficit, negative affect, or cognitive distortion, the present model proposes that the key pathogenetic mechanism of PPD is a chronic disruption of the transition from mobilization to action.

Anhedonia, psychomotor retardation, helplessness, and a reduced sense of agency are therefore viewed not as manifestations of a primary motivational deficit, but as consequences of a chronically impaired ability to act resulting from suppression of the mobilizing impulse. The proposed model also integrates psychodynamic concepts regarding the role of suppressed aggression in depression with contemporary findings on fronto-limbic conflict, dopaminergic dysregulation, and chronic activation of the stress-response system. The PPD model does not exclude the involvement of cognitive, personality-related, social, and biological factors in the formation of depressive states; however, it conceptualizes them primarily as variables influencing the degree of inhibition of the mobilizing impulse and the ability to act.

Clinical observations obtained during the application of Integrative Causal Psychotherapy are generally consistent with this model. Particularly significant is the absence of relapse among patients who achieved remission during the one-year follow-up period [22]. Within the framework of the proposed model, this may be explained by the fact that psychotherapeutic intervention is directed not only toward symptom reduction, but also toward modification of the central pathogenetic mechanism — chronic suppression of the mobilizing impulse.

At the same time, the proposed model remains hypothetical and requires further empirical validation. The small sample size, absence of a control group, and limited availability of neurobiological data do not allow firm conclusions regarding the validity of the proposed mechanism. In addition, the model is primarily applicable to primary psychogenic depression and does not claim to explain all forms of depressive disorders.

Future research should focus on validating the proposed model in larger samples and investigating the neurobiological correlates of restoration of personal agency.

Conclusion

The proposed model of primary psychogenic depression conceptualizes the depressive state as resulting from chronic suppression of the mobilizing impulse that arises in response to the inability to fulfill fundamental psychological needs and initiate meaningful action. Within this framework, the central pathogenetic mechanism of PPD is assumed to consist of chronic suppression of the mobilizing impulse and blocking of its translation into action, accompanied by impaired dopaminergic reinforcement processes, reduced agency, helplessness, and the development of a persistent depressive symptom complex. Depression in PPD may be understood not only as an affective disorder, but also as a state characterized by a chronically impaired capacity for action.

The model integrates psychodynamic concepts concerning the role of suppressed aggression in depression. It also incorporates contemporary findings on fronto-limbic conflict, dopaminergic dysregulation, and chronic activation of the stress-response system. From this perspective, aggression is conceptualized primarily as a mobilizing affect that facilitates overcoming obstacles and translating the mobilizing impulse into action.

Based on the proposed pathogenetic model, a causally targeted psychotherapeutic approach to PPD was developed to eliminate inhibitions against experiencing the mobilizing impulse and translating it into action. Clinical observations obtained during application of the Integrative Causal Psychotherapy protocol are generally consistent with the proposed model and suggest the potential value of a mechanism-based approach to the treatment of primary psychogenic depressive.

Thus, within the proposed model, depression is conceptualized as a state of unresolved mobilization and impaired translation of the mobilizing impulse into action in situations perceived by the individual as threatening fundamental psychological needs. Accordingly, the regained ability to shape one's life circumstances may be viewed not merely as a contributor to symptom reduction, but as a potential central therapeutic mechanism for primary psychogenic depression. Although the proposed model requires further empirical validation, it provides a preliminary pathogenetic framework for understanding the mechanisms underlying the development of PPD and for guiding its psychotherapeutic treatment.

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