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ANXIETY AND ALCOHOL WITHDRAWAL: DISTINCT DISORDERS OR A SHARED CIRCUIT DYSREGULATION

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Abstract
Background: Anxiety disorders and alcohol use disorders often co-exist and are historically regarded as different diagnoses in the categorical systems (DSM-5-TR, ICD-11). Nevertheless, there is growing clinical and neurobiological data indicating a great deal of overlap in their pathogenesis.
Objective: The purpose of this review is to appreciate anxiety and alcohol withdrawal as the two divergent expressions of the same dysregulation within the same neurocircuit, and not as two diseases.
Methods: A narrative synthesis of existing research in clinical psychiatry, affective neuroscience, and addiction biology was employed, in which cortico-limbic circuitry and neurotransmitter dynamics were tracked.
Results: Distinctive features of anxiety as well as alcohol-related states are dysfunction of Cortico-limbic networks (such as hyperactivity of the amygdala, prefrontal control failure, and dysregulation in the hippocampus). One key process is the imbalance between the inhibitory GABAergic and the excitatory glutamatergic neurotransmission. There is a transient recovery in inhibitory tone after acute alcohol consumption, and chronic consumption brings about neuroadaptations that favour excitatory prevalence, which leads to withdrawal effects of hyperexcitability. These are a continuum from subclinical anxiety to extreme withdrawal syndromes.
Conclusion: A circuit-based continuum model offers a single framework that connects anxiety and alcohol use-withdrawal states. The practice promotes the transdiagnostic, mechanism-based diagnosis and treatment, with outstanding implications for early intervention, integrated care, as well as enhanced outcomes, especially in resource-limited environments.

KEYWORDS: Anxiety disorders, alcohol use disorder, alcohol withdrawal, cortico-limbic circuitry, neurocircuit dysregulation, GABA–glutamate imbalance, transdiagnostic psychiatry, dimensional model, comorbidity, and RDoC framework.

1. INTRODUCTION

Anxiety disorders and alcohol use disorders are among the most common and limiting psychiatric conditions globally, and It contribute significantly to the morbidity, mortality, and socioeconomic costs on a global scale. Anxiety disorders contribute substantially to the global burden of disease and remain a major cause of functional impairment worldwide, and it impacts individuals of all ages and often result in enduring functional disability and low quality of life [1]. Alcohol use disorders, in turn, are a significant issue for overall health, as It are accompanied by a plethora of negative consequences, such as health-related complications, comorbidity with psychiatric disorders, and premature mortality [2,3]. In India, the lack of access to organised mental healthcare services, social stigma, and delays in help-seeking behaviour make this burden even worse, and all lead to late presentation and worse outcomes [4,5].

The comorbidity is high and is one of the most visually conspicuous and clinically significant attributes of these disorders. The epidemiological and clinical research has always proven that anxiety disorders are associated with alcohol use disorders, affecting each other in terms of presence, severity and duration [6]. The above-mentioned co-occurrence is no accident; it represents a common feature of normal clinical practice. The presence of the symptoms in patients, which are persistent anxiety, maladaptive drinking and withdrawal phenomena, is a dynamic and ever-changing phenomenon. There is a "dynamic" process, but not "discrete" and "isolated" disorders, as far as the symptom clusters are concerned. The above-mentioned reality of the clinic does not coincide with the models of traditional diagnostics, according to which these two disorders are regarded as separate from each other.

Currently used models of diagnostics, such as the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR) and the International Classification of Diseases (ICD-11), use a categorical approach, by means of which the mental disorders are characterized in terms of symptom clusters and threshold criteria [7, 8]. Though this model has contributed to an increase in testing reliability and ensured uniformity of communication, it came under growing criticism due to its biological invalidity and non-mechanistic basis. In particular, the notion of diagnostic reification, i.e. descriptive categories as being biologically defined diseases, has been observed as one of the most

important limitations [9]. Moreover, the considerable phenomenological overlap of anxiety states, substance use behaviours and withdrawal syndromes complicates the idea of these issues being addressed as distinct disease problems [10].

This may be especially well observed in longitudinal clinical settings. The same patient can be diagnosed with numerous diagnoses in chronological order or even a combination of them simultaneously, e.g., generalised anxiety disorder, panic disorder, alcohol dependence and alcohol withdrawal syndrome. Although all the diagnoses might be legitimate within the framework of the existing models, this richness might be predisposed to mix up the sequence of the disorder process. Consequently, the therapeutic approaches are more likely to be based on symptoms rather than mechanisms, on specific clinical manifestations rather than on common pathophysiological pathways. The practice can lead to gaps in care, chances of relapse and poor progress in the long term.

A further drawback is that the application of standardized clinical scales, such as anxiety rating scales and withdrawal severity ratings, serves only as an indicator of the degree of symptomatology, but not as an indication of the biological processes behind [11]. Such scales are employed as a continuum, and depending on the state of illness, there are different degrees of symptomatology. Their common usage in anxiety and alcohol-related disorders implies a dimensional approach to psychopathology, even though the diagnostic approach remains categorical.

Altogether, the combination of clinical psychiatry, affective neuroscience and addiction biology studies suggests that the separation of anxiety disorders and alcohol use-withdrawal states might reflect their neurobiological essence. The separation of these disorders from each other might be artificial, as it is progressive manifestations of the same dysfunction of the cortico-limbic pathway with impairment of prefrontal control, increased limbic activity and alteration of the inhibition-excitation ratio of the GABAergic/glutamatergic neurotransmission system. Within this framework, anxiety, alcohol use, dependence, and withdrawal can be conceptualized as dynamic clinical expressions along a continuum of increasing circuit instability [12]. This review therefore proposes a unified circuit-based model that integrates these traditionally separate diagnostic states within a common neurobiological framework.

2. Limitations of Categorical Psychiatry

The prevailing diagnostic paradigms of modern psychiatry, such as the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR) and the International Classification of Diseases (ICD-11), are based on a categorical paradigm according to which mental disorders are perceived as discrete units that are determined by clusters of symptoms and threshold-based criteria [13]. Although this method has improved inter-rater reliability and helped to achieve a standardised clinical communication, its ability to capture the underlying biological diversity of psychiatric conditions is restricted. A fundamental premise of this model is the existence of distinct and stable boundaries between psychiatric diseases, which is increasingly being questioned by clinical observations and neurobiological research findings [14].

Clinical overlap and heterogeneity represent one of the greatest limitations of the categorical approach since it fails to sufficiently explain them. Alcohol use disorders and anxiety disorders are often comorbid, with a variety of overlapping symptom domains, such as autonomic hyperarousal, sleep disturbances, irritability, and cognitive dysregulation [15]. To make matters worse, these conditions are similar, and the dynamics of time to each of these also make the separation difficult. Alcohol use can be an antecedent to anxiety, as people are interested in reducing some anxiety, and chronic alcohol use can increase underlying anxiety. The symptoms also tend to mimic or even worsen the symptoms of anxiety and so make diagnosis of the withdrawal very difficult [16]. This relationship is reciprocal and dynamic, so it is increasingly hard to make a firm distinction between the categories.

Longitudinal clinical settings are a particularly good way of observing diagnostic fragmentation. There is also a possibility that the same person may be diagnosed with several disorders in different stages of disease development, i.e., generalised anxiety disorder in the initial stages of usage, alcohol dependence in the long-term usage and the alcohol withdrawal syndrome at the end of usage. The disease course may be valid within the current definition criteria, but it may be hard to understand the full course of the disease. These diagnostic constructs, however, are predicted to be in danger of reification, i.e., the entification of descriptive categories, not clinical constructs of the moment [17].

The other weakness of significance is the use of symptom-based measurement tools. Popular scales like the Hamilton Anxiety Rating Scale and Clinical Institute Withdrawal Assessment of Alcohol (CIWA-Ar) are also concerned with the severity of the symptoms but fail to give an answer to the mechanism of the disease and neurobiological limits [18]. These assessment tools, by their nature, measure the symptoms on a continuum of severity; it is a record of the colour of the severity of the end of any clinical condition. It can be used for anxiety, and it can also be used for withdrawal – there's a huge conceptual paradox here, a fundamental issue with psychiatry: it still has a conceptual structure of categorising and measuring dimensions.

Having a symptom rating mismatch and a categorical diagnosis has specific clinical implications. In practice, the degree of symptoms rather than the diagnosis of the disorder itself is the key determinant in deciding how to treat

the disorder, and there is an intuitive understanding that the disorders are of the same type. Without an integrative mechanistic model, however, clinical management was very reactive, that is, responding to the impact of symptoms in the here and now, rather than to underlying pathophysiological processes. This can lead to fragmented care, to an incoherent care plan and to a relapse.

As a reaction to these shortcomings, novel theories like the Research Domain Criteria (RDoC) have attempted to surpass the previous groups of diagnosis through the focus on transdiagnostic dimensions and neural networks underlying them [19]. These models consider the basic functional spheres that include negative valence systems, arousal and cognitive control that traverse the historic lines of diagnosis. are integrative and mechanistic, based on the premise of elucidating mental diseases through links between clinical manifestations and neurobiological systems [20].

All these downsides of categorical psychiatry, particularly the loss of diagnosis to diagnosis, course-to-course discontinuity and the utter absence of any correlation between measure and mechanism, all imply that one must have a more sensible conceptual system. An alternative approach that could be fruitful involves a circuit-based viewpoint based upon neurobiological mechanisms and dimensional expression. This would be more in line with established clinical facts and would also give us a framework on which to infer that anxiety and alcohol use-withdrawal states are related phenomena, as opposed to independent diagnoses.

3. Neurocircuitry of Anxiety

3.1 Core Neural Circuits

Anxiety disorders are increasingly being viewed as network dysfunction disorders, primarily involving the cortico-limbic systems of threat detection, emotional processing and behavioural control. The amygdala is the main component of this network, as it is a very important part of the network that allocates emotional salience and determines potential threats. Neuroimaging and meta-analysis reports have consistently revealed heightened activity in the amygdala in individuals with anxiety disorders, which is an overreaction to stimuli of actual and perceived danger [21].

In normal states, the prefrontal cortex (PFC), especially the medial and dorsolateral prefrontal cortex, has a top-down inhibitory control of the limbic structures, which allows the adaptive regulation of emotional responses. Such a control system is disrupted in anxious situations, causing reduced prefrontal control and prolonged limbic hyperactivity [17]. It is particularly important to note that neurobiological mechanisms that lead to stress only worsen the functioning of the prefrontal and impair executive control, as well as emotional dysregulation. This process is regarded as a fundamental aspect of anxiety pathogenesis. Due to the existing imbalance between cortical regulation and limbic motivation.

The other important element in this circuit is the hippocampus, which integrates contextual information and coordinates fear-processing elements that involve memory. Impaired functioning of hippocampal circuits could impair the ability to differentiate between a safe and threatening context, resulting in heightened fear reactions and erroneous generalisation of threat cues [22]. The amygdala, prefrontal cortex and hippocampus are interconnected as a system in which the loss of functional balance resulted in the basic clinical symptoms of anxiety.

3.2 Neurotransmitter Dynamics

Anxiety disorders are characterised neurochemically by an impairment in the balance between the inhibitory and excitatory neurotransmission. The primary inhibitory neurotransmitter of the central nervous system is gamma-aminobutyric acid (GABA), which plays a crucial role in maintaining neurons and curbing abnormal excitability. The lower GABAergic tone has been linked to an incremental increase in anxiety and a lack of inhibitory control [23].

The most important excitatory neurotransmitter, glutamate, in contrast, tends to be rather elevated or unregulated during anxiety, and it is used to stimulate neuronal activity and hyperexcitation in the network [16]. Such an imbalance of inhibition and excessive excitation causes the nervous system to be in a highly unstable condition and predisposes individuals to overreacting to emotions and overarousal.

It is also very fascinating to note that the balance between inhibition and excitation is not fixed but rather controlled dynamically by stress, environment, and genetic predisposition. The chronic state of stress has also been found to inhibit GABAergic transmission while increasing glutamate transmission, thus creating an imbalance in the cortico-limbic circuits and thereby sustaining anxiety disorders. However, the degree of such neurochemical changes is not the same for all because anxiety states are heterogeneous.

3.3 Functional State of the Anxious Brain

The malfunction of the circuit and the disproportion of the neurotransmitters result in the functional condition of a higher level of vigilance and loss of emotional control. This can be clinically expressed through a state of continuous hypervigilance, an oversized perception of threats, as well as an inability to switch off perceived

threats. It is a neurobiological condition that is linked to an increase in limbic responsiveness- particularly in the amygdala- as well as an absence of regulatory prefrontal cortex response [24].
This imbalance, which promotes the long-term activation of autonomic and stress-response systems, is one of the causes of the physiological symptoms that are common in anxiety disorders, such as tachycardia, sweating and high arousal. It is important to note that such features also exist in a range of circuit stability. The mild dysregulation may be expressed through subclinical anxiety or hyper-reaction to stress, but the more extreme imbalance may manifest itself in extreme anxiety disorders, including stress-related and panic disorders.
The state of anxiety may be conceptualized not merely as a diagnostic category but as a dynamic neurobiological state that is an expression of instability and fluctuations in cortico-limbic networks with a more liberalised approach [25]. This conceptualisation provides a critical relationship with alcohol use and withdrawal, where the same processes, particularly based on the GABA-glutamate imbalance and impaired prefrontal control, are only heightened. This overlap can inform the potential to have a shared neurobiological model of anxiety and alcohol-related conditions.

4. Neurobiology of Alcohol Use and Withdrawal

4.1 Acute Effects of Alcohol on Neural Circuits

The acute neurobiological effects of alcohol are mostly enacted through its exploitation of the inhibitory and excitatory neurotransmitter systems and ultimately exert a net effect as a depressant on the activity of the central nervous system. Ethanol also increases the neurotransmission of the GABAergic system by positive allosteric modulation of the GABA receptors, thus augmenting the inhibitory tone and decreasing neuronal excitability. Alcohol, at the same time, inhibits glutamatergic transmission, especially by blocking the N-methyl-D-aspartate (NMDA) receptors, causing a decrease in excitatory synaptic activity [26].
This two-fold action, where one side is augmentation of inhibition and the other suppression of excitation, is the source of the typical clinical effects of alcohol, such as sedation, anxiolysis, and disinhibition of behaviour. Notably, these are effects that have given neurobiological support to the self-medication hypothesis in that a person who has an underlying anxiety can use alcohol to restore inhibitory balance in dysregulated cortico-limbic circuits temporarily [27]. Nevertheless, this compensation system can be a short-term relief; however, it causes neurobiological instability in the long term.

4.2 Chronic Alcohol Use and Neuroadaptation

Functional equilibrium is achieved through a cascade of neuroadaptive changes that occur in the brain after sustained exposure to alcohol. With chronic alcoholic consumption, GABA receptor sensitivity and activity are downregulated and as such, inhibitors decline in their effects in the long run. At the same time, glutamatergic neurotransmission is upregulated, with an increase in expression and activity of NMDA receptors, which leads to an increase in excitatory drive [28].
These adaptations represent a change to a condition of comparative excitatory pre-eminence, even when alcohol is still present. These changes are often interpreted within the framework of allostasis, in which the brain forms a new pathological set-point that is defined by changes in the regulation of reward circuits, stress circuits and emotional circuits. Notably, it is not just classical neurotransmitter systems that experience these neuroadaptive changes, but also stress-relevant neuropeptides such as corticotropin-releasing factor (CRF) that also serve to perpetuate emotional imbalances and relapse susceptibility [29].
But one needs to consider that the scope of those neuroadaptations and their characteristics may differ in individuals and depend on genetic, environmental, and psychosocial factors. This heterogeneity adds to the heterogeneity of clinical presentation of alcohol use disorders.

4.3 Neurobiology of Alcohol Withdrawal

Abel, excessive alcohol use, or sudden withdrawal, causes a withdrawal from these neuroadaptive changes. With cessation of alcohol's inhibitory effects, the excitatory pathways that were “shut off” by the alcohol now take priority and result in “rebound hyperexcitability” of the CNS [30].
It is a condition of decreased GABAergic inhibition and overactivity of glutamatergic activity, resulting in general neuronal overactivation. Clinically, these are manifested as autonomic instability (tachycardia, hypertension, tremors, diaphoresis), representing dysregulation of central autonomic networks. With increasing severity, the patient can experience withdrawal seizures, which emerge because of uncontrolled cortical excitability, and delirium tremens, which is a condition that is life-threatening condition characterised by agitated mood, confusion, and general neurocognitive impairment [31].
Circuit-wise: Alcohol withdrawal may be thought of as a major imbalance of inhibitory/excitatory and de-regulatory activation of prefrontal and activation of limbic and brainstem. This is the extreme side of a spectrum of neural disturbance, where corrective mechanisms have broken down, with unchecked excitatory signalling prevailing.

4.4 Integration with Anxiety Neurocircuitry

It is worth noting that the neurobiological alcohol consumption/ toxicity pathway and withdrawal are very similar to the circuit-based modification of anxiety disorders. The conditions are both typified by an imbalance in the GABA-glutamate balance, increased limbic activity, and deteriorated prefrontal control. Alcohol withdrawal may represent a more severe and unstable manifestation of overlapping neurobiological mechanisms, but anxiety is a partially dysregulated state.

This cross-over provides an engaging starting point on which to imagine anxiety and alcohol-related conditions as one process of dysregulation of progressive circuits. These disorders are unlikely to delimit other disorders but can be expressed as other points on a shared neurobiological spectrum, which is defined by the degree of imbalance of cortico-limbic networks.

5. A Unified Circuit Model of Anxiety and Alcohol Use–Withdrawal

5.1 Continuum of Circuit Dysregulation

The central hypothesis of this review is that anxiety disorders, alcohol use, alcohol dependence, and alcohol withdrawal may not represent entirely separate diagnostic entities, but rather progressive manifestations of a shared cortico-limbic circuit dysregulation. According to this model, the clinical differences observed across these conditions primarily reflect variations in the severity and persistence of dysfunction within interconnected neural networks responsible for emotional regulation, stress responsiveness, and inhibitory control. Consequently, these states can be understood as different points along a neurobiological continuum rather than isolated psychiatric disorders [32].

The initial disruptions to this balance could result in the development of slight abnormalities of stress sensitivity and arousal, which are usually expressed in subclinical anxiety. The result of such a progression is clinically significant forms of anxiety, characterised by increased limbic responsiveness, continuing hypervigilance, and a lack of top-down control by the prefrontal cortex. Additional increases can result in injuries at panic levels, a temporary burst of circuit hyperexcitability over fear-processing networks [33].

For some individuals, persistent circuit instability may contribute to alcohol use as a maladaptive attempt to restore emotional regulation, and this is mostly due to a maladaptive process of trying to reestablish the sense of inhibition by introducing exogenous modulation of GABAergic signalling. Although the effect of repeated alcohol exposure is effective in relaxation of anxiety, neuroadaptive mechanisms leading to a shift to the pathological equilibrium in the system occur. In the long term, this development could eventually lead to dependence, and when no longer occurring, withdrawal states, whereby the excitatory state is greatly predominant, and the regulatory ability is lost [34].

In this way, these clinical states may be conceptualised as interconnected manifestations of potentially shared neurobiological processes, but, instead, can be better regarded as dynamic states along a common neurobiological pathway of increasing circuit instability.

5.2 Shared Neurobiological Mechanisms

There are many important mechanisms that remain consistent in this hypothetical spectrum. One of the key elements here is the difference between GABAergic and glutamatergic neurotransmissions that lead to anxiety disorders and withdrawal from alcohol. The decreased inhibitory tone in combination with the increased activation of the excitatory drive creates the imbalance in the network, thus making people vulnerable to strong emotions and physiological reactions.

The neurochemical disorder is the integrative disorder of circuits, specifically the disorder of the overactivation of limbic circuits and prefrontal regulation deficiency. The hyperactivity of the amygdala and other regions associated with it also contributes to the increase in threat perception and affective sensitivity and a lack of prefrontal affective response regulation [15-17].

With the continuous exposure to alcohol and chronic stress, these problems become increasingly prominent and eventually develop into maladaptive circuit dynamics [35].

In addition to impairment at the regional level, recent discoveries show that these changes are a result of the disruption of large-scale brain networks, and not individual abnormalities. Dysregulation of interconnected networks (salience, executive control and emotional processing networks) may provide a more detailed account of the different clinical manifestations of different anxiety and alcohol-related disorders [36]. This is a network-based view that validates a transdiagnostic view (that is, different symptoms are due to a common disruption of coordinated neural processes).

5.3 Clinical Transitions and Dynamic Interactions

This neurobiological continuum is normally manifested in the clinical picture of the patient. Anxiety is commonly the antecedent of alcohol use, where people use alcohol to self-medicate, and after drinking, it feels good to overpower the emotion of distress and regain emotional balance [6,7]. This approach may work temporarily, but can lead to neuroadaptation and dependence, creating a neuroregulatory cycle of dysregulations.

The withdrawal also causes an abrupt loss of the inhibitory effect of alcohol, thus unveiling its underlying excitatory effect, resulting in increased anxiety, autonomic hyperarousal and increased risk for developing severe symptoms such as seizures and delirium tremens [37]. This can easily lead to a relapse of alcohol use as people are attempting to diminish the substance use and withdrawal symptoms, and after that it begins an adverse cycle of neurobiological imbalance, anxiety, and substance use.

One thing that is necessary to keep in mind is that people do not go through these changes in the same way, since it might be affected by their genetics, poor environment, and psychodynamic elements. The inconsistency brings out the need for flexible models that ensure consistency in dealing with diversity.

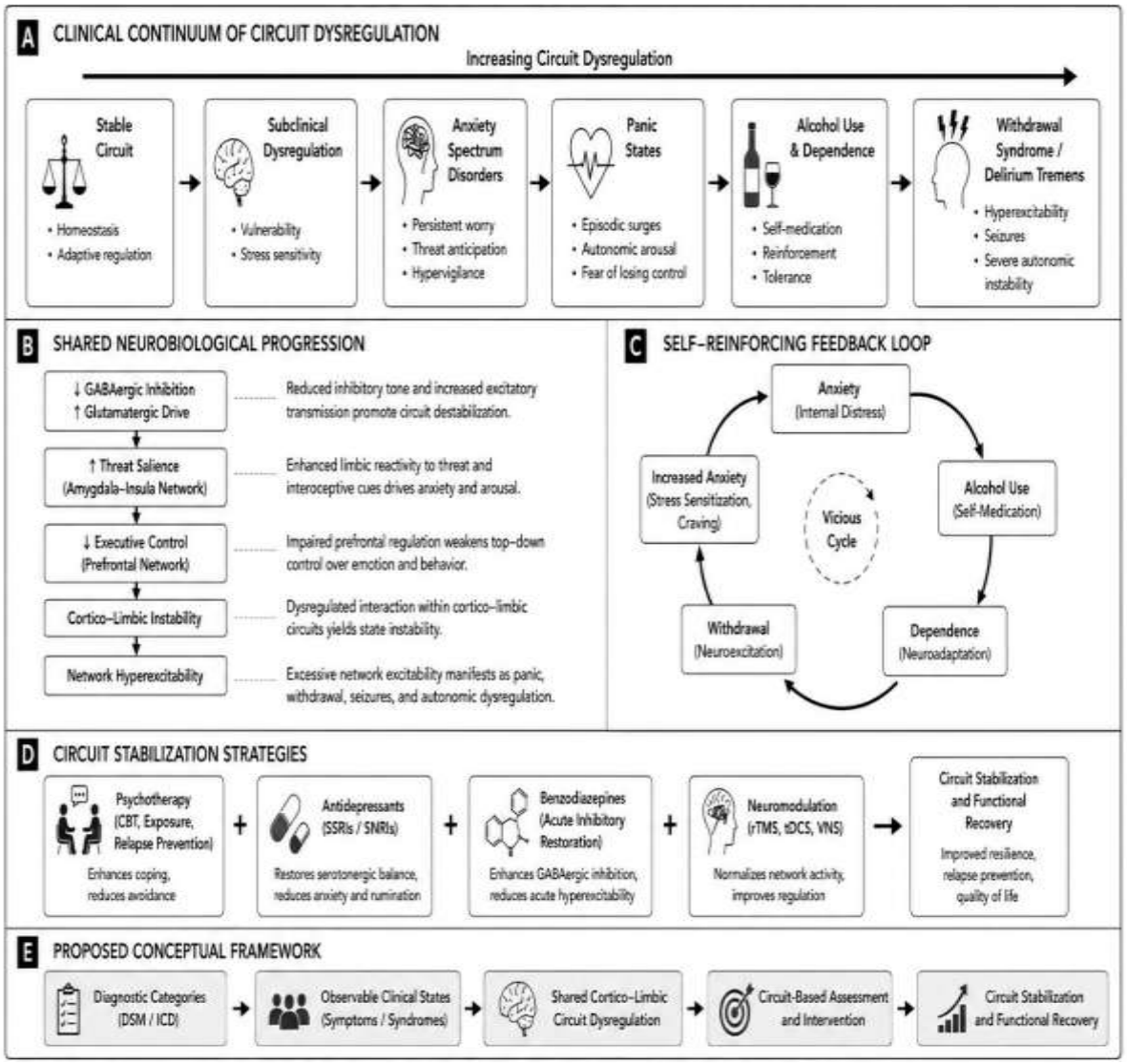

Figure 1: A shared cortico-limbic circuit dysregulation model linking anxiety disorders, alcohol use disorder and alcohol withdrawal states (Continuum of Circuit Dysregulation Model)

(A) Proposed clinical continuum depicting progression from stable circuit regulation through subclinical dysregulation, anxiety spectrum disorders, panic states, alcohol use/dependence, and ultimately alcohol withdrawal syndrome and delirium tremens, reflecting increasing circuit instability and hyperexcitability.
(B) Shared neurobiological progression characterized by reduced GABAergic inhibition, increased glutamatergic drive, enhanced threat salience, impaired executive control, cortico–limbic instability, and network hyperexcitability.
(C) Self-reinforcing feedback loop illustrating how anxiety may promote alcohol use as self-medication, while neuroadaptation, withdrawal, and stress sensitization subsequently exacerbate anxiety and relapse vulnerability.

(D) Circuit stabilization strategies, including psychotherapy, antidepressants, benzodiazepines, and neuromodulation, conceptualized as interventions targeting restoration of network stability and functional recovery.
(E) Proposed conceptual framework suggesting that conventional diagnostic categories may represent observable clinical states emerging from varying degrees of shared cortico–limbic circuit dysregulation, supporting a circuit-based approach to psychiatric assessment and treatment.

6. Clinical Implications

6.1 Rethinking Diagnosis: From Categories to Circuit States

The continuum model proposed has implications for psychiatric conditions conceptualisation and diagnosis. The classical diagnostic model, e.g., DSM-5-TR and ICD-11, is a model of classification and naming that does not assume the dynamic (or overlapping) nature of anxiety and alcohol-related disorders [8-10]. While some of these frameworks can be relevant in clinical communication and administration, several limitations can be seen in patients with developing and comorbid symptoms.
A circuit-based framework moves away from a static diagnostic classification to a framework of dynamic states of neural dysregulation. From this perspective, a series of clinical problems (such as generalised anxiety, panic, alcohol dependence and withdrawal syndromes) may be viewed as related clinical expressions within a shared cortico-limbic framework. This view is in line with dimensional models of psychopathology and transdiagnostic models like RDoC [38] which rely on underlying neurobiological systems rather than symptom-based categorisations.
It is significant to emphasize that this redefinition doesn't cancel out any of the present diagnostic categories but describes their phenotype with clinically useful terms. Clinicians might be able to have higher levels of diagnostic coherence and better clinical reasoning in their diagnoses based on the combination of categorical diagnosis and dimensional, circuit-based knowledge.

6.2 Treatment as Circuit Stabilisation

This may provide some helpful insight for rethinking existing treatment plans. Interventions should not be considered as "acting upon" specific symptoms but can be considered as returning a balance to dysregulated circuits.
Good examples of this principle are pharmacological therapies. The mechanism of action of benzodiazepines is that they increase GABAergic inhibition, thus reversing hyperexcitable conditions experienced in both anxiety and alcohol withdrawal [25,27]. are shown to be effective in the prevention of acute anxiety and the reduction of withdrawal complications, and this is a fact that accentuates their applicability in maintaining the balance of inhibitory-excitatory.
Likewise, selective serotonin reuptake inhibitors (SSRIs), which are often used in anxiety disorders, are believed to affect cortico-limbic circuits in the long term, improving emotional control and decreasing the limbic hyperreactivity [39]. Their main mechanism is the serotonergic pathways, but downstream effects of the pathway lead to normalisation of network dynamics and enhanced prefrontal control.
According to this model, the use of non-pharmacological interventions, particularly psychotherapeutic interventions like cognitive-behavioural therapy (CBT), plays an important role in the treatment of traumatised patients. These interventions enhance thinking and emotion regulation and reduce the maladaptive coping responses. Psychotherapy is applied to favourably control the limbic activity. These approaches may be implemented in combination with pharmacological treatment to constitute the multimodal treatment approach to target the dysfunction of the underlying circuit rather than the symptoms.

6.3 The Importance of Timing: Early Intervention and Prevention

One of the key implications of the continuum model is the recognition that the timing of intervention may substantially influence clinical outcomes. The initial phases of dysregulation of circuits, usually in the form of mild or subclinical anxiety, provide an important window of opportunity. At this stage, neural systems may retain sufficient plasticity to allow effective stabilisation and prevention of further progression, which further stabilise and prevents the development.
Lack of coping with early dysregulation can result in the use of maladaptive coping strategies such as alcohol intake that initially offers symptomatic relief but eventually entraps the underlying neurobiological imbalance according to neuroadaptive mechanisms [40]. The further a condition develops into dependence and withdrawal, the more complicated and resource-consuming interventions are, and the more morbidity is related to them.
An active, mechanism-based strategy focuses on early detection and treatment and lifelong monitoring along the course of an illness. This can lower the risk of progressing and relapsing, as well as enhance functional outcomes in the long run.

6.4 Integrated and Longitudinal Care Approaches

The pattern of integrated and longitudinal models of care can also be supported by the circuit-based model. The dynamic character of circuit dysregulation means that patients need continuous assessment and treatment plans that can respond to the fluctuations in a clinical condition.

Rather than regarding anxiety and alcohol use disorder as two separate disease processes, an integrative therapy considers their interaction and looks at them as one unit of treatment. It might include pharmacotherapy, psychotherapy, and psychosocial treatment, which are used in combination according to their place on the continuum [41].
Continuous monitoring becomes very valuable and can be considered in the context of this re-emerging model, that of circuit instability. Prompt intervention in response to the signs may help maintain stability and prevent any further development of the situation.

6.5 Implications for Resource-Limited Settings

The circuit-oriented model is of practical value in resource-limited settings, specifically in the Indian states. Because no attention is paid to diagnostic categorization of the patient, simplifying the complex differential diagnosis process and focusing on the mechanism of the condition helps clinicians use a much narrower treatment plan.
It promotes scalable response, early treatment, and efficient use of available resources. It is also supportive of the actual clinical practice in life where patients tend to present more often with overlapping symptoms that may not necessarily fall within any specific category [42].

7. Measurement vs Mechanism

Clinical scales that are standardised have now become essential to psychiatric practice, as are required to evaluate the seriousness of the symptoms and apply the same in deciding how to treat the patient. Various scales such as Clinical Institute Withdrawal Assessment for Alcohol, revised (CIWA-Ar) and Hamilton Anxiety Rating Scale (HAM-A) are typical in the evaluation of alcohol withdrawal disorder and anxiety disorder respectively [12,13]. Such tools give formal and repeated measures of clinical condition and have been shown to be useful in tracking treatment response and severity levels.
The disadvantage inherent in these tools, however, is that the tools are symptom-based in nature. It can be used in describing the clinical phenomena which are seen and reported, but not the neurobiological processes underlying them. It is one of the instances of CIWA-Ar that quantifies such characteristics as tremor, autonomic instability, and agitation, clinical signs of central nervous system hyperexcitability, but does not provide information about the circuitry changes that lead to the manifestations. Likewise, HAM-A evaluates the psychological and somatic aspects of anxiety without identifying various neural mechanisms that can contribute to the development of similar symptom profiles [13].
This non-connectivity is connected to an even greater conceptual gap in psychiatry: disorder is categorically defined, and symptoms are dimensionally scaled, but the neurobiological architecture underlying such symptoms is hardly ever quantified in the conventional clinical practice. This would mean that measures of assessment that are currently available would not be able to deal with the heterogeneity of pathophysiological processes underlying it, and their reliance on mechanism-based interventions or precision-oriented interventions would be limited.
The issue of why there is a gap between measurement and mechanism needs to be filled in is mentioned in the circuit-based paradigm of this study. The new discoveries in neuroscience and digital health technology offer possible avenues to such integration. The pattern of disrupted communication on cortico-limbic networks can be ascertained using neuroimaging techniques, such as functional connectivity, as objective signs of impairment in circuits [43]. Similarly, electrophysiological techniques such as electroencephalography (EEG) and event-related potentials can be used to capture dynamic processes in neural activity in real-time and inform on excitatory and inhibitory equilibrium and network stability.
Simultaneously, with the advent of digital phenotyping, continuous and ecologically valid measurement of behavioural and physiological status can be done based on data provided by smartphones and wearable devices. Indirect measures of stability of underlying circuits may involve things such as sleep patterns, activity levels, heart rate variability, and behavioural rhythms. These plans may be particularly beneficial in instances that are characterised by changeable symptomatology, as in this situation, real-time observation and the timely detection of aggravation or a relapse can be carried out.
As much as these advances have been made, there are still several challenges. Neurobiological scales are also time-consuming and cannot be standardized and are not at present in standard clinical practice. Moreover, the neural data is complex and even more needs to be translated into clinically actionable insights; further methodological development and validation would be needed. Hence, although integration of both biological and digital markers has significant potential, caution should be taken in applying the practice regarding their feasibility and clinical relevance.
Interestingly, also, a possible way forward here would be to create hybrid methods of assessment which involve a blend of traditional clinical measures with simplistic biological/behavioural indications of circuit activity. These models would provide a more specific image of the condition of a patient but be feasible to use in practice.
In short, it can be observed that the scales of clinical assessment can be put to good use in the sense that they can, in fact, be able to quantify the manifestation of psychopathology, but it cannot be used to determine the neural processes that may be able to determine psychopathology. Closing this measurement-mechanism divide is an important move to progressing a circuit-based psychiatry. A combination of subjective measures of symptoms

and objective measures of circuit functionality has the potential to enable a more accurate diagnosis, a more specific treatment and ultimately an improved clinical outcome.

8. Indian Context

Anxiety disorders and alcohol use disorders represent a growing public health concern in India, and such trends are having a lot of impact on population wellbeing and health care systems as well as socioeconomic status. The national epidemiological statistics indicate that alcohol is among the most consumed psychoactive drugs in the country, and it is likely to co-occur with psychiatric disorders, most commonly anxiety disorders [44]. This two-fold burden is contributing to morbidity, reduced productivity, and a huge burden on healthcare facilities.

The lack of access to mental healthcare services and their uneven distribution among Indians is a huge challenge. The special psychiatric services are often confined to urban areas, and rural and semi-urban locations have a large population that does not receive adequate services [5]. There are other reasons, such as social stigma, unawareness and constraints, that lead to delayed behavior problems in seeking help. This leads to patients presenting in more acute stages of disease, like alcohol dependence and withdrawal, and rarely in less acute and more responsive stages of anxiety or mild dysregulation.

In clinical practice, practical constraints of treatment frequently result in emphasising short-term symptom management over long-term care plans, causing discontinuous care and management. The continuum framework provides a potential circuit-based conceptualization of anxiety, alcohol consumption, and withdrawal. This is a complex conceptualization of the patient treatment process in terms of neurobiology. The model is quite consistent with the clinical presentation that is close to the real world, where symptoms can overlap and change over time, without adhering to strict diagnostic terms. Moreover, the approach is resource-efficient and makes use of clinical tests that are already available without the necessity of complex technology, enabling clinicians to make consistent treatment choices by targeting underlying processes in which there is a balance between inhibitory and excitatory systems.

In addition, the emphasis on early detection and treatment is mentioned with the help of this framework, which is particularly significant in India where late presentation is not a rare occurrence. Eventually, the progression of the disorder to higher levels (dependence and withdrawal) can be avoided through the assistance of early diagnosis of dysregulation of the circulation symptoms (which are always present or felt) [45].

A circuit-based model could also play a role in the creation of more integrated mental-health programs, a public health approach. Such programs can be used to address anxiety, and substance use could be addressed with the benefit of enhancing screening, lessening service fragmentation and continuity of care. Besides, the approach may be applicable in training primary care providers by simplifying complicated psychiatric concepts into a simplified and mechanism-based knowledge.

However, it should be mentioned that some implementation challenges might arise. The challenges of insufficient neurobiological model awareness, dependence on inconsistent clinical education, and the constraints offered by an entire system could be obstacles to extensive implementation. In this respect, the attempts to implement such a framework in practice should be supported by some education, capacity building and adaptation to the local healthcare systems [46].

To conclude, implementing integrated mental health methods within the Indian healthcare framework is both a challenge and an opportunity to advance towards more cohesive approaches in mental health care. A circuit-based continuum model provides a scalable, practical and clinically relevant model consistent with the real-life situation of a patient and resource problems. This approach can increase treatment outcomes and decrease the burden of psychiatric illness in India by increasing conceptual clarity and encouraging early and mechanism-informed intervention.

9. Future Directions

The proposed circuit-based continuum model offers a conceptual basis in which to re-conceptualise the association between anxiety disorders and alcohol use withdrawal states. Nevertheless, systematic progress in research, measurement and intervention design is needed to translate this framework into clinical practice.

An urgent need for future studies is a longitudinal research design, following individuals on a continuum of proposed states of anxiety to alcohol usage, addiction, and impact. Most of the available evidence is not longitudinal, and it is therefore not able to indicate time relationships and causality. Future research combining clinical, behavioural, and neurobiological data would be a worthwhile contribution to the current understanding of the dynamics of circuit dysregulation over time and possibly provide initial indicators of progression.

The other significant direction is the development of circuit-informed assessment tools. The clinical tools that are in use today are mainly used to measure the severity of symptoms without measuring the underlying neural processes. More future directions can be based on combining old rating scales with objective indicators of neuroimaging-based measures of connectivity, electrophysiological (e.g., EEG indices of excitatory-inhibitory balance) and behavioural or cognitive indicators of regulatory control. These hybrid models might allow for better characterize patient subtypes and make a mechanism-guided stratification.

Simultaneously, clinical trials around circuit-based hypotheses are required. Instead of classifying groups of patients as having either of two diagnostic categories or the other, future research can classify subjects according to patterns of circuit dysfunction most common in the patient-inhibitory deficit vs. limbic hyperreactivity. Subsequently, custom-crafted interventions could be offered to treat these mechanisms, e.g., pharmacological

control of GABAergic or glutamatergic circuits, psychotherapeutic activities to involve increased cognitive control, and novel neuromodulation techniques, in which the prefrontal circuits are involved.
Digital technology is another aspect that holds promise. Continuous monitoring of behavioural and physiological parameters with data obtained with smartphones and wearable devices is a possibility with the aid of digital phenotyping. These strategies could support a timely identification of circuit instability, forecast relapse, and application of just-in-time measures, especially in those areas where clinical follow-up is not possible regularly.
These opportunities notwithstanding, there are several challenges that must be overcome. Biomarker standardization, circuit-based measures validation, and accessibility are still a pressing method of concern in a variety of healthcare environments. Besides, the complex neurobiological data also need an interdisciplinary approach involving psychiatry, neuroscience, and data science to translate all these data into a format that can be put into clinical practice.
Finally, to develop a circuit-based psychiatric model, it is necessary to combine longitudinal studies, mechanisms-based evaluation, and intervention-specific strategies. This framework may contribute to the development of more precise and mechanism-informed psychiatric models, producing better results and having a more consistent awareness of psychiatric illness.

10. CONCLUSION

The traditional classification of anxiety disorders and alcohol withdrawal has long functioned as a diagnostic and therapeutic framework for psychiatric conditions. Recent knowledge gleaned from clinical experiences, affective neuroscience, and addiction studies, however, indicates that these diseases may be more closely intertwined than is currently recognized. Based on this review, anxiety disorders, alcohol use, alcohol dependence, and withdrawal may be progressive steps of a common dysfunction of a cortico-limbic circuit involving decreased prefrontal control, increased limbic activation, and an imbalance in GABA and glutamate.
Alcohol-related illnesses and anxiety are not considered separate disease categories, but as clinical states on a continuum of rising levels of circuit instability. Anxiety can be a sign of early dysregulation, but alcohol use, dependence and withdrawal are increasingly severe forms of the same underlying dysfunction in the nervous system. This circuit theory also offers an explanation for the high prevalence of comorbidity, symptom overlap, and clinical course of alcohol-related disorders and anxiety disorders.
Interestingly, contemporary clinical practices, an effective attempt at symptom management, are more likely to be carried out without explicit agreement to mechanisms. Biological facts, incomplete at the circuit-level, underlying these disorders, are classified in diagnostic systems and measured in clinical scales, both of which are incomplete. The treatment plans may thus be derailed and reactive [24-25].
A circuit-based framework provides an integrative view which can help to cross this conceptual divide. It allows a more integrated view of psychiatric illness by putting an emphasis on the common neurobiological processes and dynamic functional states. The strategy can help in earlier identification of circuit dysregulation (for intervention) and may justify a single model of care for the pharmacological, psychological and emerging neuromodulator therapies.
It should be noted, however, that the framework did not displace the existing diagnostic systems; rather, it complemented them. While it is empirically based, the use of category classification could be further improved if it is also incorporated with circuit-based understanding and, if necessary, with further improvement of clinical reasoning and the precision of treatment.
To sum up, the model of dysregulation of circuits is more reliable, biologically sound, and clinically applicable to explain alcohol withdrawal and anxiety withdrawal. This perspective has led to a greater understanding of the relationship between psychiatry and neuroscience, as well as to more comprehensive, efficient and personalized avenues for mental health care. The future generation will require even closer integration of clinical insights, neurobiological studies, and ingenious equipment to gain understanding of the neurological basis of mental illnesses and to treat them to stabilisation.

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