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Exploring the therapeutic potential of beta blockers in psychiatry: Review study

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ABSTRACT

Introduction: Beta-blockers, primarily used for cardiovascular conditions, have attracted attention for their potential benefits in psychiatry. Their ability to influence sympathetic nervous system activity makes them candidates for adjunctive treatment in various psychiatric disorders, including aggression, anxiety disorders, and withdrawal syndromes. **Methods:** A systematic review of scientific literature was conducted using PubMed. The search focused on placebo-controlled trials, cohort studies, open-label trials, and clinical trials that evaluated beta-blockers in psychiatric contexts. Relevant studies were analyzed for their efficacy in managing symptoms of aggression, anxiety disorders (PTSD and GAD), withdrawal syndromes, and opioid addiction. **Results:** Beta-blockers like propranolol effectively reduced aggression and agitation, thus improving patient safety and treatment outcomes. In anxiety disorders, particularly PTSD and GAD, these agents showed potential in alleviating symptoms by modulating sympathetic nervous system activation. The evidence for beta-blockers in alcohol withdrawal syndrome is mixed, with atenolol showing promise in reducing treatment failure rates and cravings, while propranolol's effects on specific symptoms remain inconclusive. In opioid withdrawal, propranolol may enhance patient retention in detoxification programs. **Discussion:** The findings support beta-blockers as valuable adjuncts in managing psychiatric conditions. Their effects on aggression and anxiety are promising, though the evidence for alcohol and opioid withdrawal is less consistent. Further research is necessary to better understand and optimize their use in clinical practice. **Conclusion:** Beta-blockers offer potential benefits across several psychiatric conditions, particularly in managing aggression and anxiety. While their role in withdrawal syndromes is promising, further studies are needed.

Keywords: Beta blockers, anxiety disorders, post-traumatic stress disorder, alcohol withdrawal syndrome, opioid withdrawal

1. INTRODUCTION

Beta-blockers, or beta-adrenergic receptor antagonists, have been under development since the 1950s. Since their inception, they have emerged as one of the most extensively utilized pharmacological agents in contemporary medicine. Their clinical importance is particularly prominent in cardiology, where they are indispensable for managing arterial hypertension, ischemic heart disease, and various cardiac arrhythmias, most notably atrial fibrillation. These agents function by inhibiting the beta-adrenergic receptors, leading to a reduction in heart rate, myocardial contractility, and, consequently, myocardial oxygen demand. Beyond their well-established therapeutic indications, beta-blockers have piqued the interest of clinicians and researchers for their potential off-label uses. For several years, considerable exploration has been made into the expanded clinical applications of these drugs.

Notably, psychiatry has been investigating the potential benefits of beta blockers for conditions outside their conventional scope. Understanding the pharmacology of beta blockers is crucial for exploring their potential applications in psychiatry and beyond, particularly their interaction with adrenergic receptors. Structurally resembling catecholamines, beta blockers primarily exert their effects through competitive antagonism of beta-adrenergic receptors. These receptors are categorized into B1, B2, and B3 types, each with distinct bodily distributions and functions. B1 receptors predominate in the heart and kidneys, while B2 receptors are found in various organs, including the lungs, liver, gastrointestinal tract, uterus, vascular smooth muscle, and skeletal muscle. B3 receptors are predominantly in adipose tissue (Arcangelo and Peterson, 2006; Frishman et al., 2005).

Beta-blockers can selectively block one or more receptor types, depending on their pharmacological properties. By antagonizing these receptors, beta blockers inhibit the effects of norepinephrine and other stress hormones, which play crucial roles in the sympathetic and central nervous systems (CNS). This blockade is central to their therapeutic action, reducing sympathetic activity and mitigating physiological responses to stress (Gorre and Vandekerckhove, 2010). In psychiatric conditions such as schizophrenia, anxiety disorders, bipolar disorder, and dementia, there is often dysregulation in norepinephrine turnover. Research suggests that increased norepinephrine activity may contribute to symptoms observed in these conditions (Ramos and Arnsten, 2007).

Beta-blockers are therefore investigated as adjunctive treatments in psychiatry, aiming to modulate sympathetic activity and potentially alleviate symptoms associated with these disorders. Their impact on the prefrontal cortex is particularly interesting. This part of the brain is essential for executive functions such as concentration, impulse control, behavioral adaptation, and working memory (Ramos and Arnsten, 2007). Studies indicate that norepinephrine influences these cognitive processes, suggesting a potential role for beta-blockers in enhancing cognitive function in psychiatric patients. This paper reviews current literature and studies exploring the therapeutic use of beta blockers in specific psychiatric disorders. By elucidating their pharmacological mechanisms and clinical applications, it aims to contribute to a better understanding of their potential benefits in psychiatric treatment.

2. METHOD

To collect data for this analysis, we reviewed the scientific literature on utilizing beta blockers in psychiatry. We systematically searched the authoritative, publicly available database PubMed. Our search strategy incorporated specific search terms, including "beta blockers", "psychiatry", "anxiety", "generalized anxiety disorder (GAD)", "alcohol withdrawal", "opioid withdrawal", "opioid addiction", "post-traumatic stress disorder (PTSD)", "agitation", and "aggression".

Articles identified through this search process underwent evaluation, with particular attention to methodologies employed in those studies. The inclusion criteria were randomized controlled trials, cohort studies, open-label trials, and clinical trials. The exclusion criteria were meta-analysis, cross-sectional studies, case reports, and reviews. After investigating the articles for these criteria, 21 studies that matched the purpose of this review were selected. This process is shown in the flow chart below (Figure 1).

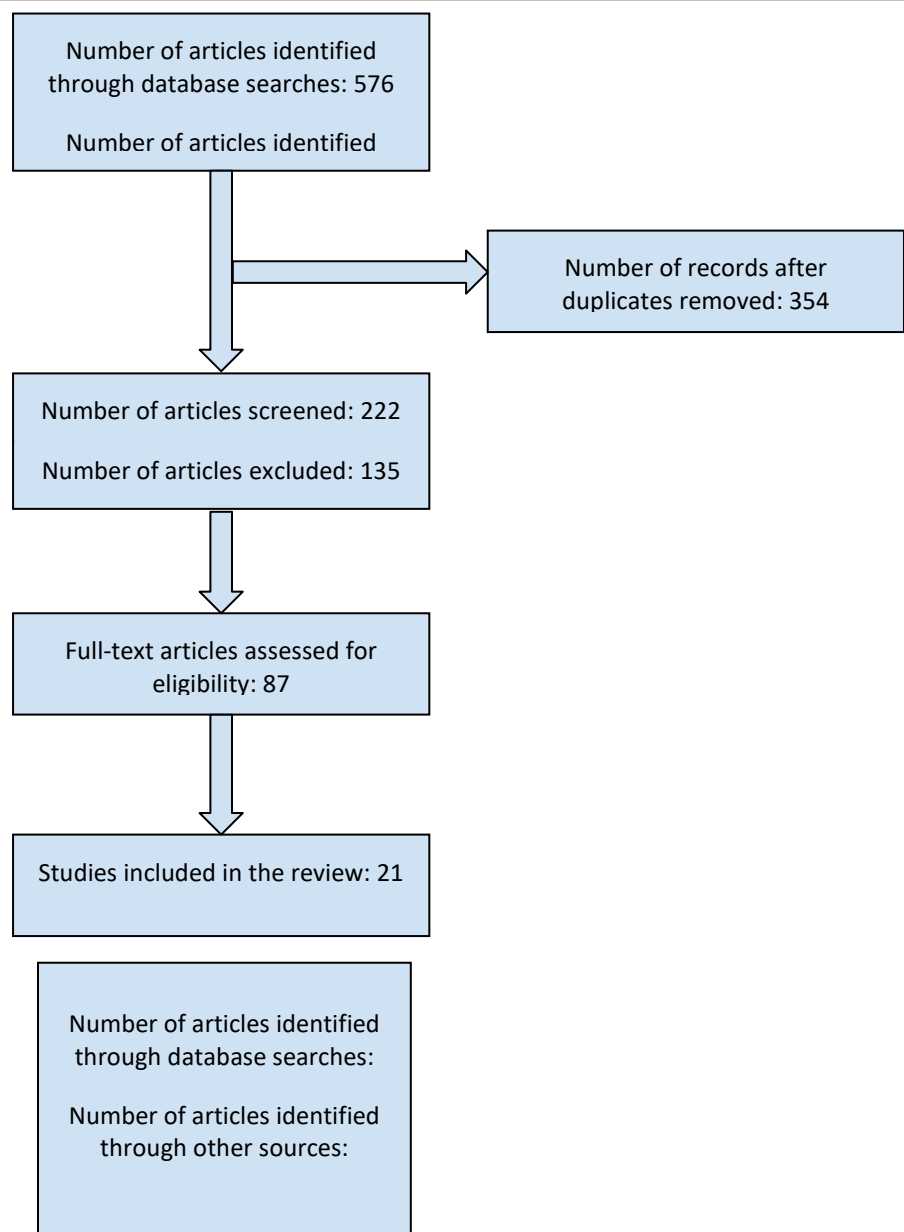


Figure 1 Flow chart

3. RESULTS

Beta-blockers usage in the treatment of aggression and agitation

Agitation and aggression are pervasive issues in psychiatric patients, particularly in the controlled environment of inpatient units. These behaviors endanger staff, fellow patients, and the patients themselves and complicate treatment and recovery efforts. Therefore, there is a critical need to explore novel, less invasive interventions that can effectively mitigate these challenges. By prioritizing the development of such approaches, we aim to enhance the safety and overall well-being of psychiatric patients under care. A review of research papers retrieved from the PubMed database supports the rationale for using beta blockers in managing aggression and agitation in psychiatric patients. A pivotal study conducted in 1989 investigated the effects of propranolol in 20 psychiatric patients through a placebo-controlled trial (Silver et al., 1999).

Participants, aged over 18 years, had a documented history of recurrent aggressive behavior spanning at least six months and were diagnosed with chronic mental illness accompanied by psychosis, organic mental illness, or mental disability. The study administered

propranolol starting at 80 mg/day, with gradual increases every 2 to 4 days (depending on the clinical response), up to a maximum tolerated dose of 1440 mg/day or 20 mg/kg/day. Patients remained on this regimen for a minimum of 2 months. Results indicated that approximately one-third of the participants experienced a greater than 50% reduction in the frequency and severity of aggressive episodes and overall agitation. Another comparative study assessed the efficacy of carbamazepine and propranolol in managing aggressive attacks among psychiatric patients, demonstrating positive outcomes with both medications (Mattes, 1990).

Furthermore, investigations utilizing nadolol and pindolol as adjunctive therapies for schizophrenia patients have indicated significant reductions in aggressive behavior (Caspi et al., 2001; Ratey et al., 1992). Moreover, in a population-based longitudinal cohort study that examined the effects of beta-blockers on psychiatric and behavioral outcomes in over 1.4 million individuals in Sweden, it was found that beta-blocker use was associated with reduced rates of violent crime and psychiatric hospitalizations (Molero et al., 2023). All the mentioned findings underscore the potential role of beta blockers as adjunctive treatments in psychiatric care, particularly in mitigating aggression and enhancing overall patient management. The abovementioned studies are compared in the table below (Table 1).

Table 1 Studies on beta-blocker usage in the treatment of aggression and agitation

Study	Beta-blocker	Number of participants	Duration	Patients characteristics	Primary outcome
Silver et al., 1999	Propranolol	20	Two months	Psychiatric patients with chronic mental illness and psychosis	Propranolol led to a significant reduction in the frequency and severity of aggressive episodes in about 33% of participants. The improvement was measured using standardized scales, showing a greater than 50% reduction in aggression and overall agitation.
Mattes, 1990	Propranolol	29	Average 23 days	Psychiatric patients with aggressive attacks	Propranolol significantly decreased the frequency of aggressive attacks in patients. The study used clinical observations and ratings to confirm a notable reduction in aggressive behavior.
Caspi et al., 2001	Pindolol	30	12 weeks	Schizophrenic patients with a history of aggression	A statistically significant reduction in both the number and severity of aggressive incidents.

					No significant changes in Positive and Negative Syndrome Scale (PANSS) scores were observed.
Ratey et al., 1992	Nadolol	41	17 weeks	Chronic psychiatric inpatients with frequent aggressive outbursts	Nadolol produced a significant decrease in the frequency of aggressive outbursts and reductions in hostility, negative symptoms, and hyperarousal/tension
Molero et al., 2023	Various beta-blockers	1,400,766	Eight years	Population-based Cohort in Sweden	Beta-blocker use was associated with reduced violent crime charges

Beta-blocker usage in the treatment of anxiety disorders

Anxiety disorders encompass a broad scope of conditions; most important are generalized anxiety disorder (GAD), panic disorder, post-traumatic stress disorder (PTSD), social phobias, and specific phobias. In a notable 2010 study, beta-adrenergic receptor blockade demonstrated a reduction in activity within the basolateral amygdala, a brain region crucial for regulating fear responses to environmental stimuli, among healthy individuals exposed to images featuring faces of varying emotional intensity (Hurlemann et al., 2010). This neurophysiological finding underpins the potential therapeutic role of beta blockers in managing anxiety and fear disorders.

Given the diverse trajectories and underlying mechanisms of anxiety disorders, this paper focuses on two specific conditions, PTSD and GAD, examining their distinct clinical presentations, and the potential use of beta blockers in these contexts will be explored to elucidate their role in augmenting current treatment strategies. Post-traumatic stress disorder (PTSD) is a condition developing in response to experiencing or being a witness to a traumatic event of extreme stress, which exceeds an individual's capacity to cope and integrate effectively. Symptoms commonly include heightened mental tension, anxiety, nightmares, and flashbacks related to the traumatic event. Individuals with PTSD often experience feelings of helplessness and emotional exhaustion. These symptoms are typically triggered by reminders or cues associated with the traumatic experience.

In 2003, a study was published comparing the effects of propranolol on the incidence of PTSD in patients exposed to traumatic events (Vaiva et al., 2003). Participants were recruited from a local emergency department within 2 to 20 hours after experiencing a severe traumatic event, for example, a traffic accident or being assaulted. Eleven patients agreed to take propranolol, while eight opted for participation without medication, forming two groups. The propranolol group received 120 mg daily for seven days, while the control group received neither propranolol nor placebo. Although the groups were not randomized, the investigators ensured that the trauma's demographic characteristics, severity, and nature did not differ significantly between them.

Two months post-event, both groups were screened for PTSD. Among those taking propranolol, PTSD was diagnosed in 1 out of 11 patients, whereas in the non-medicated group, the diagnosis was made in 3 out of 8 patients. Additionally, another study involving emergency department patients corroborated the positive effects of propranolol in mitigating stress and anxiety responses triggered by text-image stimuli associated with traumatic memories (Hoge et al., 2011). Similar outcomes were observed in additional small-scale studies (Pitman et al., 2002; Brunet et al., 2008). Moreover, in a 2015 study, propranolol proved to help improve the cognitive function of patients with chronic PTSD (Mahabir et al., 2016).

However, not all literature unequivocally supports the use of beta blockers in treating traumatic stress syndrome. A study comparing the effects of propranolol and gabapentin on PTSD symptoms in post-accident patients found no significant difference compared to placebo (Stein et al., 2007). Also, in a study from 2017, acute propranolol treatment did not show any effect on PTSD

prevalence in patients who suffered burn-related trauma (Rosenberg et al., 2018). These findings underscore the need for further research to understand better the efficacy and limitations of beta blockers in managing PTSD symptoms following traumatic events. The abovementioned studies are compared in the table below (Table 2).

Table 2 Studies on Beta-blocker usage in the treatment of post-traumatic stress disorder

Study	Beta-blocker	Number of participants	Duration	Patients characteristics	Primary outcome
Vaiva et al., 2003	Propranolol	19	Seven days	Patients exposed to severe traumatic events within 20 hours	PTSD was diagnosed in 1/11 patients in the propranolol group vs. 3/8 in the control group. Suggests propranolol may reduce PTSD incidence.
Hoge et al., 2011	Propranolol	41	19 days	Emergency department patients with acute psychological trauma	There was no significant difference in PTSD symptoms or physiological reactivity between the propranolol and placebo groups. High adherence subgroups showed lower reactivity with propranolol.
Pitman et al., 2002	Propranolol	41	Ten days	Patients who experienced acute psychological trauma within 6 hours	The Propranolol group had lower PTSD severity scores and fewer physiological responses to traumatic imagery compared to placebo. Results suggest the potential for acute post-trauma propranolol to prevent PTSD.
Brunet et al., 2008	Propranolol	19	One day	Patients with chronic PTSD	Propranolol was given after

					memory retrieval reduced physiological responses during mental imagery of traumatic events. Results suggest that propranolol can be effective even when administered after memory reactivation.
Mahabir et al., 2016	Propranolol	41	Two weeks	Patients with chronic PTSD	Propranolol improved cognitive functioning, particularly processing speed, in PTSD patients
Stein et al., 2007	Propranolol (compared with gabapentin)	48	14 days	Surgical trauma patients	There was no significant difference in depressive or PTSD symptoms between propranolol, gabapentin, and placebo. Suggests limited efficacy of these treatments in preventing PTSD following acute traumatic injury
Rosenberg et al., (2018)	Propranolol	202	Seven years	Children who suffered severe burns	There is no significant difference in PTSD prevalence between propranolol and control groups.

Generalized anxiety syndrome (GAD- general anxiety disorder) is a disorder for which the most characteristic symptoms are chronic states of mental tension, worry, and slow-flowing anxiety. Publications exploring the use of beta-adrenergic receptor antagonists (beta blockers) in the treatment of generalized anxiety disorder (GAD) are notably sparse. One significant study investigated the effects of betaxolol in a cohort of 31 patients, including 13 inpatients and 18 outpatients diagnosed with GAD or similar anxiety disorders (Swartz, 1998). Patients received betaxolol at 10 to 80 mg doses, adjusted based on individual clinical responses. The

study found that betaxolol effectively resolved or significantly reduced anxiety symptoms to minimal levels in 100% of inpatients and 85% of outpatients.

Similarly, encouraging outcomes were reported in another study that examined the administration of propranolol and atenolol to patients initially diagnosed with generalized anxiety syndrome by a general practitioner while awaiting psychiatric appointments (Peet and Ali, 1986). Moreover, a 2023 study involving 74 patients with autism spectrum disorder showed promising results for the usage of propranolol as a means to lower anxiety levels in this patient demographic (Beverdors et al., 2024). These results underscore the potential therapeutic benefits of beta blockers in managing anxiety symptoms across diverse clinical settings. Despite these promising results, the current literature on beta blockers in GAD remains limited within commonly used databases. The abovementioned studies are compared in the table below (Table 3).

Table 3 Studies on Beta-blocker usage in the treatment of generalized anxiety syndrome

Study	Beta-blocker	Number of participants	Duration	Patients characteristics	Primary outcome
Swartz, 1998	Betaxolol	31	One year	Patients with GAD or adjustment disorder with anxiety (13 outpatients, 18 inpatients)	Betaxolol rapidly reduced anxiety, with 85% of outpatients and 100% of inpatients improving to no more than marginally ill. Panic attacks in patients with concurrent panic disorder ceased within two days. Significant improvements were noted, even in longstanding cases and those with obsessive-compulsive personality disorder.
Peet and Ali, 1986	Propranolol and Atenolol	49	Three weeks	Patients diagnosed with GAD by a general practitioner while awaiting psychiatric appointments.	Significant improvement in symptoms in propranolol and atenolol groups compared to placebo, suggesting effectiveness in managing anxiety symptoms in

					patients with GAD.
Beversdorf et al., 2024	Propranolol	74	12 weeks	Patients with autism spectrum disorder	Propranolol showed significant improvement in anxiety but no significant effects on social interaction or language.

Beta-blockers usage in the treatment of alcohol withdrawal syndrome

Alcohol dependence is classified as a medical disorder in the DSM-IV, characterized by an individual meeting three of seven criteria over 12 months (American Psychiatric Association, 1994). These criteria include increased alcohol tolerance, symptoms of alcohol withdrawal syndrome, consuming alcohol in greater amounts and for more extended periods than intended, inability to cut down on usage despite a desire to do so, spending excessive time obtaining and using alcohol, reducing time dedicated to other life activities due to alcohol, and continued alcohol use despite awareness of its negative health consequences. This condition significantly impacts both bodily and mental health, with profound societal repercussions.

Enhancing our understanding of pharmacotherapy alongside psychotherapy is crucial for effectively treating alcohol-related disorders and supporting patient recovery. During alcohol addiction, one common complication is alcohol withdrawal syndrome, which encompasses a range of symptoms appearing after the abrupt cessation or significant reduction in alcohol consumption following prolonged use (Rybakowski et al., 2010). These symptoms include headaches, muscle twitching, increased sweating, nausea, tachycardia, hypertension, anxiety, irritability, hallucinations, altered consciousness, and seizures (Rybakowski et al., 2010). These symptoms are attributed to heightened sympathetic nervous system activity mediated by catecholamines.

Recent studies have explored the potential of propranolol in managing alcohol withdrawal symptoms by reducing urinary catecholamine levels excreted during this syndrome (Sellers et al., 1976). However, the clinical impact of reducing catecholamine levels on overall symptomatology remains a subject of ongoing investigation. The efficacy of the usage of beta-blockers, such as propranolol, in alleviating alcohol withdrawal symptoms raises essential questions about their role in clinical practice. Further research is warranted to clarify the precise mechanisms by which beta-blockers modulate withdrawal symptoms and to assess their potential as adjunctive treatments in enhancing outcomes for individuals undergoing alcohol withdrawal.

This ongoing exploration holds promise for optimizing therapeutic strategies and improving care for patients grappling with alcohol-related disorders. In a pivotal 1989 study, researchers investigated the efficacy of atenolol as an adjunct to standard pharmacotherapy in managing alcohol withdrawal syndrome. The study randomly allocated 88 patients to receive atenolol and 92 patients to receive a placebo, assessing them over two weeks for symptom severity and treatment outcomes (Horwitz et al., 1989). Treatment failure, defined as leaving the study, breaking abstinence, or experiencing withdrawal symptoms lasting more than five days, occurred in 37% of the atenolol group compared to 52% in the placebo group. Additionally, the atenolol group reported a significantly lower proportion of patients experiencing severe alcohol cravings (7% vs. 20%).

However, another study comparing diazepam and propranolol showed that while propranolol effectively reduced symptoms of sympathetic activation during alcohol withdrawal, it did not significantly impact the incidence of seizures or disturbances of consciousness (Worner, 1994). Similarly, two additional studies found in the PubMed database did not support the notion that beta blockers influence muscle tremors, mood disturbances, or cognitive function in patients following abrupt alcohol withdrawal (Mendelson et al., 1974; Teravainen and Larsen, 1976).

These findings highlight the mixed evidence regarding the efficacy of beta blockers in managing various aspects of alcohol withdrawal syndrome. While atenolol shows promise in reducing treatment failure rates and cravings, the effects of propranolol on specific withdrawal symptoms remain inconclusive. Further research is essential to elucidate the mechanisms and optimize the role of beta blockers in comprehensive treatment strategies for alcohol-related disorders. The abovementioned studies are compared in the table below (Table 4).

Table 4 Studies on beta-blocker usage in the treatment of alcohol withdrawal syndrome

Study	Beta-blocker	Number of participants	Duration	Patients characteristics	Primary outcome
Horwitz et al., 1989	Atenolol	180	Two weeks	Patients with alcohol withdrawal syndrome	Atenolol reduced treatment failure rates (37% vs. 52% for placebo) and significantly lowered severe alcohol cravings (7% vs. 20% for placebo). Suggests efficacy in managing alcohol withdrawal symptoms as an adjunct to standard pharmacotherapy.
Worner, 1994	Propranolol (compared with diazepam)	37	Seven days	Male alcoholics admitted electively for detoxification	Propranolol effectively managed withdrawal symptoms but was associated with one seizure and increased withdrawal, requiring parenteral paraldehyde. Diazepam managed symptoms without seizures or hallucinations.
Mendelson et al., 1974	Propranolol	64	Three days	Adult male chronic alcoholic addicts	Propranolol pretreatment failed to block or attenuate cognitive, perceptual, motor, and affective changes induced by acute alcohol

					intoxication. The double-blind study under controlled conditions found no significant influence on withdrawal symptoms.
Teravainen and Larsen, 1976	Propranolol	28	Three days	Patients with alcohol withdrawal syndrome	Propranolol was tested on positional tremors using electrical recording of tremor amplitude and frequency. There was no significant difference between propranolol and placebo, indicating limited efficacy in reducing tremor during acute alcohol withdrawal.

Beta-blocker usage in the treatment of opioid withdrawal and addiction

Opioid use disorder is a psychiatric disorder marked by a strong desire for opioids, persistent use despite experiencing physical and psychological decline, increased tolerance over time, and withdrawal symptoms upon stopping opioids. Withdrawal symptoms from opioids can include nausea, muscle aches, diarrhea, difficulty sleeping, agitation, and depression (American Psychiatric Association, 1994). A 1974 study assessed the efficacy of propranolol in facilitating the completion of a 9-day methadone-assisted detoxification program for opiate-dependent patients (Jacob et al., 1975). The study included 30 detoxifications involving 28 patients (22 males and six females, aged 18-28 years).

Patients received methadone according to a standardized schedule and were randomly assigned to receive propranolol doses of 20 mg, 10 mg, or 0 mg twice daily from day 3 to day 8. Of the 30 detoxifications, only three patients completed the entire 9-day period, all from the 40 mg propranolol group. Five patients remained until day 8; none were from the placebo group, two were from the 20 mg propranolol group, and three were from the 40 mg propranolol group. Notably, one patient from the 40 mg propranolol group completed detoxification twice. Clinical application of propranolol in similar settings could involve its incorporation into standard detoxification protocols for opiate dependence. Administering propranolol, as identified in this study, may potentially improve patient retention and overall treatment adherence.

Moreover, a 2011 study examined how propranolol affects drug-related memories in abstinent heroin addicts (Zhao et al., 2011). Seventy participants learned a word list with heroin-related and neutral words. Propranolol, given before memory retrieval, impaired the reconsolidation of heroin-related memories but not neutral ones. This study suggests propranolol could help disrupt persistent drug-related memories in recovering heroin addicts, potentially aiding in treatment. Unfortunately, no further papers can be found in the used databases. The abovementioned studies are summarized in the table below (Table 5).

Table 5 Study on beta-blocker usage in the treatment of opioid and addiction

Study	Beta-blocker	Number of participants	Duration	Patients characteristics	Primary outcome
Jacob et al., 1975	Propranolol	28	Nine days	Opiate-dependent patients undergoing a 9-day methadone-assisted detoxification program	Propranolol facilitated the completion of the detoxification program. Three patients from the 40 mg propranolol group completed the nine-day period, while five patients remained until day 8 (two from the 20 mg group and three from the 40 mg group). No patients from the placebo group remained. Propranolol may enhance patient retention and adherence to detoxification protocols.
Zhao et al., 2011	Propranolol	70	Three days	Abstinent heroin addicts	Propranolol impaired the reconsolidation of drug-related positive and negative memories but not neutral ones. Propranolol may help disrupt persistent drug-related memories in recovering heroin addicts.

4. DISCUSSION

Based on the detailed exploration of beta blockers' potential applications in psychiatric contexts, including the management of aggression and agitation, anxiety disorders such as PTSD and GAD, as well as alcohol and opioid withdrawal syndromes, this review consolidates significant findings and implications. The reviewed studies consistently suggest a promising role for beta-blockers in

augmenting traditional therapies across various psychiatric conditions. Specifically, in managing aggression and agitation, beta blockers like propranolol have demonstrated efficacy in reducing the frequency and severity of aggressive episodes, thus enhancing patient safety and therapeutic outcomes in psychiatric settings. Moreover, their use in anxiety disorders, particularly PTSD and GAD, underscores their potential to modulate fear responses and alleviate anxiety symptoms by targeting sympathetic nervous system activation and catecholamine-mediated stress responses.

These findings support that beta-blockers could be valuable adjuncts in comprehensive treatment strategies for these challenging conditions. In the context of alcohol withdrawal syndrome, while evidence remains mixed regarding their impact on specific symptoms such as seizures and cognitive disturbances, beta blockers like atenolol have shown promise in reducing overall treatment failure rates and cravings, potentially enhancing the management of withdrawal symptoms alongside standard pharmacotherapies. Similarly, in opioid withdrawal, early studies suggest that propranolol may facilitate patient retention in detoxification programs. However, further research is needed to establish its efficacy and optimize dosing strategies within clinical protocols.

5. CONCLUSION

This review highlights beta-blockers' potential benefits in psychiatric disorders. They have shown promise in managing aggression and agitation, with propranolol reducing aggressive episodes. In anxiety disorders like PTSD and GAD, beta-blockers help alleviate symptoms by targeting sympathetic nervous system activation. Evidence for their use in alcohol and opioid withdrawal is mixed but suggests potential benefits, such as reducing treatment failure rates and aiding patient retention in detox programs. Further research is needed to refine their application and dosing in these contexts.

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Authors' Contribution

Adam Jaskulski: Conceptualization, writing- rough preparation, editing

Dominika Kabała: Writing- rough preparation, editing

Michał Bielecki: Writing- review and editing

Agata Zapałowska: Methodology, supervision, investigation

Tymon Zatorski: Formal analysis, investigation

Milena Szczepańska: Conceptualization, supervision

Marcin Głód: Visualization, supervision

Project administration: Adam Jaskulski

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Conflict of interest

The authors declare that there is no conflict of interests.

Data and materials availability

All data sets collected during this study are available upon reasonable request from the corresponding author.

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