International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	LABORATORY MONITORING OF METABOLIC SAFETY DURING CARIPRAZINE TREATMENT IN SCHIZOPHRENIA AND BIPOLAR DISORDER: A SYSTEMATIC REVIEW
DOI	https://doi.org/10.31435/ijitss.2(50).2026.5550
RECEIVED	16 March 2026
ACCEPTED	23 June 2026
PUBLISHED	30 June 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

LABORATORY MONITORING OF METABOLIC SAFETY DURING CARIPRAZINE TREATMENT IN SCHIZOPHRENIA AND BIPOLAR DISORDER: A SYSTEMATIC REVIEW

Klaudia Magdalena Kotlarska *(Corresponding Author, Email: kmmk2500@gmail.com)*
Laboratorium Analiz Medycznych LAMED, ul. Dobra 3, 08-400 Garwolin, Poland
ORCID ID: 0009-0005-6089-1161

Wojciech Kotlarski
Mazowiecki Szpital Wojewódzki im. św. Jana Pawła II w Siedlcach Sp. z o.o., ul. Poniatowskiego 26, 08-110 Siedlce, Poland
ORCID ID: 0009-0000-7043-3117

Tomasz Mainka
Mazowiecki Szpital Wojewódzki im. św. Jana Pawła II w Siedlcach Sp. z o.o., ul. Poniatowskiego 26, 08-110 Siedlce, Poland
ORCID ID: 0009-0005-7089-4008

Emil Sergejuk
Mazowiecki Szpital Wojewódzki im. św. Jana Pawła II w Siedlcach Sp. z o.o., ul. Poniatowskiego 26, 08-110 Siedlce, Poland
ORCID ID: 0009-0008-5906-4080

ABSTRACT

Introduction: Metabolic adverse effects are a major concern during long-term antipsychotic treatment because they contribute to cardiovascular complications, endocrine disturbances, and reduced treatment adherence. Cariprazine has been considered an antipsychotic with a relatively favorable metabolic profile, but the available evidence is dispersed across studies of schizophrenia and bipolar disorder. This systematic review evaluated the laboratory-monitored metabolic safety of cariprazine in these populations.
Methods: This systematic review was conducted in accordance with PRISMA 2020 guidelines. PubMed/Medline and EMBASE were searched for studies published between 2014 and 2026 using the phrase: (cariprazine OR VRAYLAR) AND (schizophrenia OR schizophreni OR "bipolar disorder" OR bipolar) AND (safety OR tolerability OR "adverse effects" OR metabol OR prolactin OR weight OR glucose OR lipid*). After screening and full-text assessment, 11 studies met the eligibility criteria and were included in the qualitative synthesis.
Results: Across the included studies, cariprazine was associated mainly with modest body weight increase and limited changes in glucose- and lipid-related laboratory markers. Short-term and long-term studies in schizophrenia did not indicate a consistent pattern of clinically significant metabolic deterioration. Similar findings were reported in bipolar mania and bipolar depression. Prolactin-related data suggested minimal endocrine burden.
Conclusions: Cariprazine appears to have a comparatively favorable laboratory-monitored metabolic safety profile in schizophrenia and bipolar disorder. However, regular monitoring of body weight and biochemical parameters remains necessary, and further long-term studies with standardized metabolic endpoints are needed.

KEYWORDS

Cariprazine; Metabolic Safety; Laboratory Monitoring; Schizophrenia; Bipolar Disorder

CITATION

Klaudia Magdalena Kotlarska, Wojciech Kotlarski, Tomasz Mainka, Emil Sergejuk. (2026) Laboratory Monitoring of Metabolic Safety During Cariprazine Treatment in Schizophrenia and Bipolar Disorder: a Systematic Review. *International Journal of Innovative Technologies in Social Science*. 2(50). doi: 10.31435/ijitss.2(50).2026.5550

Introduction

Schizophrenia and bipolar disorder are severe, chronic psychiatric illnesses that are associated with substantial functional impairment, reduced quality of life, and increased premature mortality. In both conditions, long-term pharmacological treatment remains a central component of care, but the benefits of symptom control must always be balanced against the risk of adverse effects. Among these, metabolic disturbances occupy a particularly important place because they contribute not only to treatment discontinuation and reduced adherence, but also to the excess burden of cardiovascular and endocrine disease observed in psychiatric populations. Patients with schizophrenia and bipolar disorder already have a higher baseline risk of obesity, insulin resistance, dyslipidemia, and diabetes compared with the general population, and exposure to antipsychotic medication may further amplify this vulnerability (1). For this reason, the metabolic safety of antipsychotic treatment is not a secondary issue, but a major determinant of long-term outcomes and an essential target of clinical monitoring (2).

Cariprazine is a newer atypical antipsychotic with partial agonist activity at dopamine D3 and D2 receptors and a preferential affinity for D3 receptors, a pharmacological profile that has attracted interest because of its potential relevance for efficacy across different symptom domains and for tolerability (3,4). Clinical trials have demonstrated the efficacy of cariprazine in the treatment of acute exacerbations of schizophrenia, as well as in bipolar I disorder, including manic, mixed, and depressive episodes (5). Over the last decade, cariprazine has gained an increasingly important place in the therapeutic landscape because it combines antipsychotic and mood-stabilizing utility with a generally favorable safety profile (6). At the same time, the growing use of cariprazine in everyday psychiatric practice has created a need for a more precise understanding of its safety characteristics, especially in relation to laboratory-monitored metabolic parameters that influence long-term morbidity and treatment acceptability (3).

The question of metabolic safety is especially relevant in antipsychotic treatment because not all agents share the same risk profile. Comparative evidence indicates that second-generation antipsychotics differ substantially in their effects on body weight, glucose metabolism, and lipid parameters, and these differences are clinically meaningful when selecting treatment for patients who already carry cardiometabolic risk factors (1). In this context, cariprazine has often been regarded as a compound with a relatively modest metabolic burden compared with agents more strongly associated with weight gain and metabolic dysregulation (7). Pooled analyses and long-term safety studies in schizophrenia have generally suggested limited changes in body weight and metabolic laboratory values during cariprazine therapy (8,9). Additional pooled evidence has reinforced the impression of relative metabolic stability across broader study populations (10). Dedicated analyses have further supported the view that its endocrine profile, including prolactin-related effects, may be relatively benign (11). Such findings are clinically important, yet they should not be interpreted as eliminating the need for systematic laboratory follow-up; rather, they highlight the need to define how stable and consistent this favorable profile is across diagnoses, study durations, and treatment settings (1,2).

Available evidence on cariprazine safety comes from several types of sources, including short-term randomized controlled trials, pooled analyses of phase II and III studies, open-label extension trials, long-term safety evaluations, and broader reviews. In schizophrenia, studies examining acute exacerbation have repeatedly reported acceptable tolerability in the short term (12-14). Maintenance and extension studies have provided additional information on longer-term safety (8,9,15). Pooled datasets have also helped clarify adverse-event patterns across larger samples (10). Similarly, in bipolar disorder, trial data from mania and bipolar depression have supported the efficacy and tolerability of cariprazine (16-19). Most publications, however, have focused primarily on general safety outcomes rather than on a structured synthesis of laboratory-monitored metabolic endpoints. As a result, the available literature is informative but somewhat fragmented: weight change may be discussed in one analysis, prolactin in another, and broader safety endpoints in yet another. From the perspective of clinical practice, metabolic safety is best understood in an integrated

manner that includes anthropometric changes together with glucose- and lipid-related laboratory findings (1,7,11).

This issue is also relevant beyond the narrow scope of psychopharmacology. In routine care, inadequate metabolic monitoring may delay recognition of treatment-emergent dyslipidemia, impaired glucose tolerance, or clinically significant weight gain, thereby increasing the risk of preventable long-term complications (1,2). In patients with severe mental illness, this has broader implications for public health, healthcare utilization, and social functioning. A treatment option associated with comparatively limited metabolic disruption may improve not only individual tolerability but also continuity of care and long-term engagement with treatment (5). This point is especially important when safety is considered together with broader evidence on tolerability across diagnostic groups (7). Nevertheless, even medications considered metabolically favorable should be assessed through regular laboratory surveillance, because baseline patient characteristics, comorbid somatic illness, previous antipsychotic exposure, and concomitant treatment may all influence metabolic outcomes. Guideline-based management in bipolar disorder and contemporary reviews of cariprazine both support the importance of placing efficacy and tolerability within a broader framework of individualized and safety-conscious care (2,3,20).

Another important reason to examine this topic systematically is that the clinical position of cariprazine continues to evolve. Evidence from schizophrenia studies, bipolar trials, pooled analyses, and expert recommendations suggests that cariprazine is no longer viewed merely as a novel option, but increasingly as a relevant treatment across multiple phases and symptom profiles of major psychiatric disorders (5,6). Contemporary reviews have also highlighted its expanding place in therapeutic practice (3,4). As its use expands, clinicians need more than a general statement that the drug is “well tolerated”; they need a clearer synthesis of which metabolic parameters were assessed, how these parameters changed during treatment, and whether the available evidence supports the perception of a low metabolic burden (3,5). This is particularly important in the context of schizophrenia and bipolar disorder, where the cumulative cardiometabolic burden often results from the interaction between disease-related factors, lifestyle determinants, and long-term exposure to psychotropic medication. A focused review of laboratory monitoring therefore has both scientific and practical value (1,5).

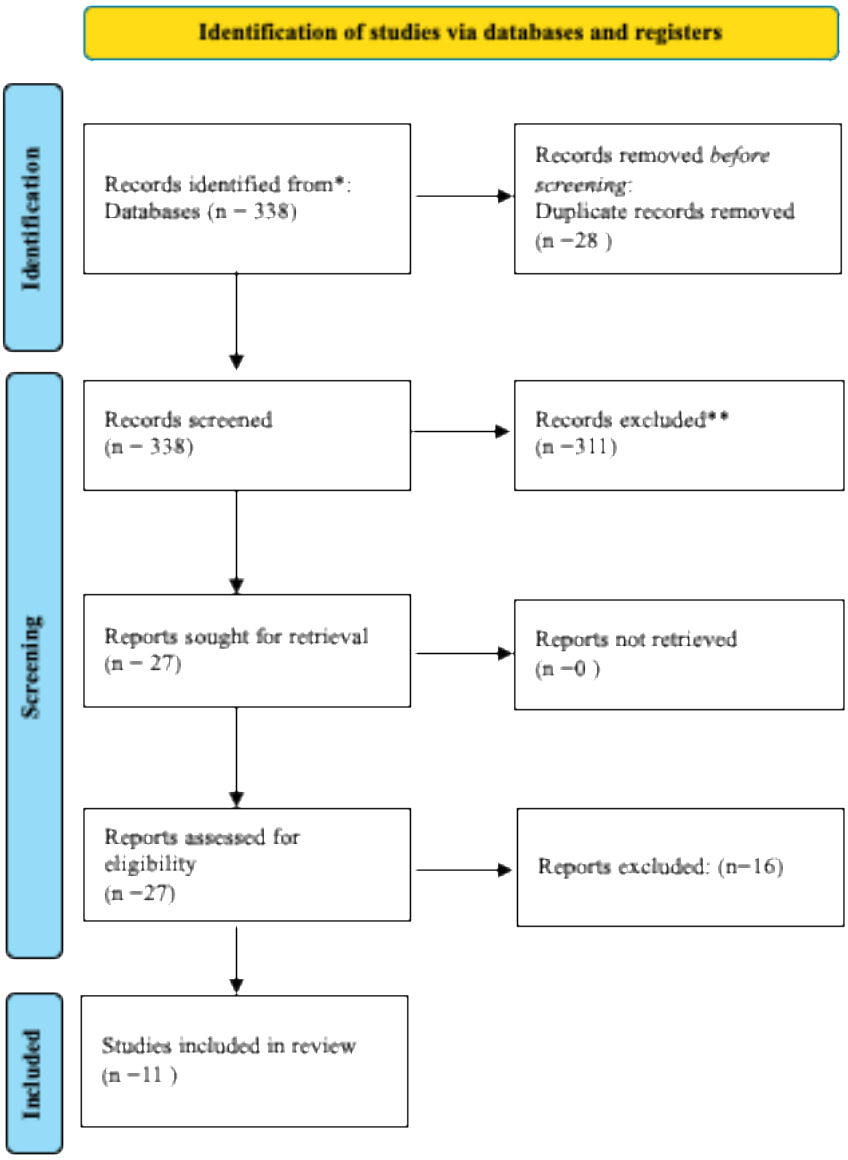
Therefore, the aim of this systematic review was to evaluate the available evidence on the laboratory-monitored metabolic safety of cariprazine treatment in patients with schizophrenia and bipolar disorder (1). Particular attention was given to changes in body weight and to laboratory parameters relevant to metabolic monitoring, including glucose- and lipid-related indices, as well as selected endocrine markers where appropriate. By integrating evidence from short-term and long-term studies, pooled analyses, and disorder-specific trials, this review seeks to provide a clinically useful synthesis of the metabolic profile of cariprazine and to clarify its place among antipsychotic treatments from the perspective of long-term safety and monitoring (5,7). The synthesis was informed by acute schizophrenia trials (12-14), longer-term schizophrenia studies (8,9,15), broader pooled analyses (10), prolactin-related evidence (11), and bipolar disorder trials covering mania and bipolar depression (17-19).

Materials and Methods:

This study was conducted as a systematic review in accordance with the PRISMA 2020 guidelines. The process of identification, screening, eligibility assessment, and final inclusion of studies was presented using the PRISMA 2020 flow diagram.

The literature search was carried out in the PubMed/Medline and EMBASE databases. Publications issued between 2014 and 2026 were considered for inclusion. The selected time frame was based on the availability of studies concerning cariprazine, with the earliest relevant records identified from 2014. The search was performed using the following phrase: (cariprazine OR VRAYLAR) AND (schizophrenia OR schizophreni OR "bipolar disorder" OR bipolar) AND (safety OR tolerability OR "adverse effects" OR metabol OR prolactin OR weight OR glucose OR lipid*)**.

PRISMA 2020 flow diagram for updated systematic reviews which included searches of databases.



After completion of the database search, duplicate records were removed. The remaining articles were screened on the basis of title and abstract. Publications judged as potentially eligible underwent full-text assessment. Studies were included in the review if they evaluated cariprazine treatment in patients with schizophrenia or bipolar disorder and reported data on metabolic or prolactin-related outcomes, including body weight, glucose-related parameters, lipid profile, prolactin concentration, or other safety findings of laboratory relevance.

The final group of studies was included in the qualitative analysis and served as the basis for the synthesis of evidence regarding the metabolic safety of cariprazine in schizophrenia and bipolar disorder.

Results

The following articles were selected for inclusion in the analysis:

1. Durgam S, Starace A, Li D, Migliore R, Ruth A, Németh G, Laszlovszky I. *An evaluation of the safety and efficacy of cariprazine in patients with acute exacerbation of schizophrenia: a phase II, randomized clinical trial*. Schizophr Res. 2014 Feb;152(2-3):450-7. doi: 10.1016/j.schres.2013.11.041. Epub 2014 Jan 10. PMID: 24412468 (14).

2. Kane JM, Zukin S, Wang Y, Lu K, Ruth A, Nagy K, Laszlovszky I, Durgam S. *Efficacy and Safety of Cariprazine in Acute Exacerbation of Schizophrenia: Results From an International, Phase III Clinical Trial*. J Clin Psychopharmacol. 2015 Aug;35(4):367-73. doi: 10.1097/JCP.0000000000000346. PMID: 26075487 (12).

3. Earley W, Durgam S, Lu K, Laszlovszky I, Debelle M, Kane JM. *Safety and tolerability of cariprazine in patients with acute exacerbation of schizophrenia: a pooled analysis of four phase II/III randomized, double-blind, placebo-controlled studies*. Int Clin Psychopharmacol. 2017 Nov;32(6):319-328. doi: 10.1097/YIC.0000000000000187. PMID: 28692485; PMCID: PMC5625952 (13).

4. Durgam S, Greenberg WM, Li D, Lu K, Laszlovszky I, Nemeth G, Migliore R, Volk S. *Safety and tolerability of cariprazine in the long-term treatment of schizophrenia: results from a 48-week, single-arm, open-label extension study*. Psychopharmacology (Berl). 2017 Jan;234(2):199-209. doi: 10.1007/s00213-016-4450-3. Epub 2016 Nov 2. PMID: 27807604; PMCID: PMC5203812 (15).

5. Cutler AJ, Durgam S, Wang Y, Migliore R, Lu K, Laszlovszky I, Németh G. *Evaluation of the long-term safety and tolerability of cariprazine in patients with schizophrenia: results from a 1-year open-label study*. CNS Spectr. 2018 Feb;23(1):39-50. doi: 10.1017/S1092852917000220. Epub 2017 May 8. PMID: 28478771 (9).

6. Nasrallah HA, Earley W, Cutler AJ, Wang Y, Lu K, Laszlovszky I, Németh G, Durgam S. *The safety and tolerability of cariprazine in long-term treatment of schizophrenia: a post hoc pooled analysis*. BMC Psychiatry. 2017 Aug 24;17(1):305. doi: 10.1186/s12888-017-1459-z. PMID: 28836957; PMCID: PMC5571492 (8).

7. Barabássy Á, Sebe B, Acsai K, Laszlovszky I, Szatmári B, Earley WR, Németh G. *Safety and Tolerability of Cariprazine in Patients with Schizophrenia: A Pooled Analysis of Eight Phase II/III Studies*. Neuropsychiatr Dis Treat. 2021 Apr 7;17:957-970. doi: 10.2147/NDT.S301225. Erratum in: Neuropsychiatr Dis Treat. 2021 May 17;17:1481. doi: 10.2147/NDT.S316858. PMID: 33854317; PMCID: PMC8040316 (10).

8. Culpepper L, Vieta E, Kelly DL, Patel MD, Szatmari B, Hankinson A, Earley WR. *Minimal Effects of Cariprazine on Prolactin Levels in Bipolar Disorder and Schizophrenia*. Neuropsychiatr Dis Treat. 2022;18:995-1011. doi: 10.2147/NDT.S348143 (11).

9. Earley W, Durgam S, Lu K, Debelle M, Laszlovszky I, Vieta E, Yatham LN. *Tolerability of cariprazine in the treatment of acute bipolar I mania: A pooled post hoc analysis of 3 phase II/III studies*. J Affect Disord. 2017 Jun;215:205-212. doi: 10.1016/j.jad.2017.03.032. Epub 2017 Mar 9. PMID: 28343051 (17).

10. Earley W, Burgess MV, Rekeda L, Dickinson R, Szatmári B, Németh G, McIntyre RS, Sachs GS, Yatham LN. *Cariprazine Treatment of Bipolar Depression: A Randomized Double-Blind Placebo-Controlled Phase 3 Study*. Am J Psychiatry. 2019 Jun 1;176(6):439-448. doi: 10.1176/appi.ajp.2018.18070824. Epub 2019 Mar 8. PMID: 30845817 (18).

11. Earley WR, Burgess MV, Khan B, Rekeda L, Suppes T, Tohen M, Calabrese JR. *Efficacy and safety of cariprazine in bipolar I depression: A double-blind, placebo-controlled phase 3 study*. Bipolar Disord. 2020 Jun;22(4):372-384. doi: 10.1111/bdi.12852. Epub 2019 Nov 6. PMID: 31628698; PMCID: PMC7318333 (19).

Eleven studies were included in this stage of the review. The selected publications represented different study designs, including phase II and phase III randomized controlled trials, pooled analyses, and long-term open-label investigations. Together, they made it possible to assess laboratory monitoring of metabolic safety during cariprazine treatment in patients with schizophrenia and bipolar disorder. Across the included studies, the outcomes most relevant to the aim of the present review were body weight, glucose- and lipid-related laboratory parameters, and prolactin concentrations. Although the studies differed in design, duration, and primary endpoints, they consistently provided safety data that could be used to evaluate the metabolic profile of cariprazine. Taken as a whole, the evidence suggested a relatively stable and predictable pattern, with limited metabolic change during both acute and maintenance treatment. The most frequently reported finding was modest body weight increase, whereas clinically important abnormalities in metabolic laboratory markers were uncommon.

The phase II randomized clinical trial by Durgam et al. assessed cariprazine in patients with acute exacerbation of schizophrenia and provided early information on the short-term safety profile of the drug (14). In this study, laboratory parameters and body weight were monitored as part of the broader safety assessment. The reported findings did not indicate clinically meaningful metabolic destabilization during treatment. No major adverse pattern involving glucose- or lipid-related measures was described, and metabolic abnormalities were not emphasized as a central concern. This trial is relevant because it represents one of the earliest controlled studies in which the safety profile of cariprazine was characterized in acute schizophrenia. From the perspective of the present review, its importance lies in showing that even at this early stage of clinical evaluation, cariprazine did not appear to be associated with a clearly unfavorable metabolic signal. In practical

terms, this created the basis for later studies to examine whether this apparently limited metabolic burden would remain stable in larger and longer trials.

A similar short-term pattern was observed in the international phase III study by Kane et al. (12). This trial again evaluated cariprazine in patients with acute exacerbation of schizophrenia and confirmed that major metabolic disturbances were not a prominent feature of treatment. Body weight was monitored during the study, and laboratory assessments were included in the safety evaluation, but the overall results did not suggest substantial metabolic worsening. The importance of this publication lies in the fact that phase III trials generally provide a more robust and clinically applicable evaluation of safety than earlier developmental studies. In this context, the study by Kane et al. strengthened the emerging impression that cariprazine was not linked to a pronounced short-term metabolic burden. When viewed together, the phase II and phase III trials present a coherent picture: acute treatment with cariprazine in schizophrenia was not associated with marked deterioration in the laboratory parameters relevant to metabolic monitoring.

A broader perspective on short-term treatment in schizophrenia was provided by Earley et al. in the pooled analysis of four phase II/III randomized, double-blind, placebo-controlled studies (13). Because this analysis combined data from several trials, it allowed a more reliable overview of metabolic safety than any individual study alone. The authors reported only modest mean increases in body weight and did not identify a clinically meaningful adverse pattern in metabolic laboratory markers. Glucose- and lipid-related measures remained generally stable across the pooled population, and no major signal of metabolic deterioration was detected. This publication is particularly important in the context of a systematic review because pooled analyses help distinguish a consistent drug-related pattern from isolated results observed in smaller studies. In the case of cariprazine, the pooled short-term findings supported the view that acute treatment was associated with relatively limited metabolic impact. The consistency between this analysis and the earlier individual trials further strengthens the impression that the short-term metabolic profile of cariprazine in schizophrenia is relatively favorable.

Long-term metabolic safety in schizophrenia was described by Durgam et al. in a 48-week, single-arm, open-label extension study (15). This study is particularly valuable because adverse metabolic effects of antipsychotic treatment often emerge or become more noticeable over longer periods of exposure. In this publication, the authors reported that no major new laboratory concerns related to metabolic safety were identified during extended treatment. Although some increase in body weight occurred, the overall pattern remained limited and did not suggest progressive metabolic deterioration. The available data therefore indicated that the safety profile seen in short-term studies remained generally stable during longer follow-up. This observation is clinically relevant because long-term treatment is the setting in which metabolic burden becomes especially important for adherence, monitoring strategy, and overall management. Even though the open-label design requires cautious interpretation, the study contributes important information by showing that prolonged exposure to cariprazine did not reveal a clearly worsening metabolic pattern.

Comparable findings were reported by Cutler et al. in a 1-year open-label study of cariprazine in schizophrenia (9). The authors again found that long-term treatment was not associated with pronounced worsening of laboratory measures relevant to metabolic monitoring. Some weight gain was observed during maintenance treatment, but the available data did not suggest marked disruption of glucose or lipid balance. This study is an important complement to the 48-week extension trial, because it provides another long-duration dataset pointing in the same direction. The fact that two separate open-label studies yielded similar conclusions adds credibility to the overall interpretation of long-term safety. Within the scope of the present review, the results reported by Cutler et al. support the view that metabolic changes during extended treatment with cariprazine are generally limited rather than progressive or clinically alarming.

The long-term pooled post hoc analysis conducted by Nasrallah et al. offered an additional perspective on sustained metabolic safety during continued cariprazine exposure (8). By integrating data from longer studies, the authors were able to examine whether cumulative treatment was associated with progressive metabolic worsening. Their findings did not reveal such a pattern. Mean changes in body weight and metabolic laboratory parameters remained limited, and the overall results were consistent with previously reported long-term data. This pooled analysis is especially useful because it provides a broader overview than any single long-term trial and makes it possible to evaluate whether the safety trends observed in individual studies remain visible when datasets are combined. For the purposes of the present review, the findings by Nasrallah et al. reinforce the conclusion that cariprazine does not appear to carry a clear risk of steadily increasing metabolic burden over time.

The broadest overview among the schizophrenia studies was presented by Barabássy et al., who performed a pooled analysis of eight phase II/III studies (10). This publication is one of the strongest sources included in the present review because it summarizes metabolic and laboratory safety across multiple trials and treatment settings. The authors reported relatively small average changes in body weight and did not identify a clinically important pattern of metabolic laboratory deterioration. Because the analysis incorporated data from a large population, it provides a particularly valuable assessment of consistency across studies. In the context of metabolic monitoring, this is important because it suggests that the favorable safety profile of cariprazine is not limited to one study, one dose range, or one duration of exposure. Instead, the available evidence from this pooled dataset indicates that limited metabolic change is a recurring finding across the broader schizophrenia program. This study therefore serves as one of the central pillars supporting the overall conclusions of the present review.

An additional aspect of laboratory safety was examined by Culpepper et al., who specifically evaluated prolactin changes in patients with schizophrenia and bipolar disorder treated with cariprazine (11). Although prolactin is not always grouped together with glucose and lipid parameters, it remains an important laboratory marker in the safety monitoring of antipsychotic therapy and may carry clinically relevant endocrine consequences. The authors reported minimal effects of cariprazine on prolactin concentrations across the analyzed populations. This finding is relevant because it broadens the concept of laboratory monitoring beyond strictly metabolic variables and suggests that cariprazine is associated not only with limited weight and biochemical changes, but also with a relatively low endocrine burden. In the context of the present review, this study complements the metabolic findings from the other included publications and supports the view that the safety profile of cariprazine remains favorable across different domains of laboratory surveillance.

The evidence base was extended further by studies conducted in bipolar disorder. In the pooled post hoc analysis by Earley et al. on acute bipolar I mania, cariprazine was generally well tolerated and did not show a strong signal of metabolic laboratory deterioration during treatment (17). The authors did not describe marked worsening of the monitored biochemical parameters, and the overall safety profile remained compatible with limited metabolic burden. This publication is important because it indicates that the relatively favorable profile observed in schizophrenia may also be present in patients treated for manic episodes. Data on metabolic safety in bipolar disorder are typically less extensive than in schizophrenia, so pooled analyses such as this one are especially valuable. For the present review, the study by Earley et al. suggests that cariprazine does not exhibit a substantially different or more concerning metabolic pattern in bipolar mania than that observed in schizophrenia.

Metabolic and laboratory safety in bipolar depression was also addressed in the randomized, double-blind, placebo-controlled phase III trial by Earley et al. (18). Although efficacy was the primary aim of the study, the authors included body weight and laboratory parameters in the safety evaluation. The published findings did not indicate pronounced adverse metabolic effects during treatment, and no clear signal of clinically important worsening in routine metabolic monitoring markers emerged. This study is relevant because depressive episodes in bipolar disorder often require sustained pharmacological management, and tolerability may directly affect adherence and long-term outcomes. In this context, the absence of a substantial metabolic signal is clinically meaningful. For the purposes of the present review, the study supports the view that cariprazine in bipolar depression is associated with limited short-term metabolic burden and a generally stable laboratory safety profile.

Comparable findings were reported in another phase III study of bipolar I depression (19). In that trial, cariprazine was again described as generally well tolerated, and the safety results did not indicate marked disruption of metabolic laboratory measures. The importance of this study lies in the fact that it confirms the observations of the previous bipolar depression trial in an independent dataset. Replication across separate studies is particularly valuable in a systematic review because it strengthens confidence in the stability of the observed pattern. In this case, the consistency between the two phase III bipolar depression studies suggests that the relatively limited metabolic impact of cariprazine is reproducible and not dependent on one specific trial. Taken together, these studies indicate that the favorable laboratory safety profile of cariprazine extends across depressive as well as manic presentations of bipolar disorder.

Overall, the included studies indicate a relatively consistent pattern of metabolic safety across schizophrenia and bipolar disorder. The most frequently observed change was modest body weight increase, whereas clinically meaningful abnormalities in glucose- and lipid-related laboratory measures were uncommon. The evidence relating to prolactin also suggests a limited endocrine burden. Importantly, both short-term and long-term studies, including pooled datasets, did not indicate progressive metabolic

deterioration over time. The consistency of findings across individual trials, pooled analyses, and longer observational periods is one of the most notable features of the available evidence. No included publication demonstrated a clear and repeated pattern of major metabolic destabilization attributable to cariprazine. Instead, the findings remained largely convergent across diagnoses and study designs. Taken together, these data support the conclusion that cariprazine has a relatively favorable profile in the context of laboratory monitoring of metabolic safety, although regular follow-up of body weight and biochemical markers should continue to be regarded as part of standard clinical practice.

Discussion

The findings of this systematic review suggest that cariprazine is associated with a relatively favorable laboratory-monitored metabolic safety profile in both schizophrenia and bipolar disorder. Across the included studies, the most consistent observation was that treatment with cariprazine was linked primarily to modest body weight increase, while clinically meaningful abnormalities in glucose- and lipid-related laboratory parameters were uncommon. In addition, the available evidence on prolactin indicates a limited endocrine burden. Taken together, these results support the view that cariprazine belongs to the group of antipsychotic agents with comparatively low metabolic liability, although this should not be interpreted as an argument against regular metabolic surveillance. Rather, the findings suggest that cariprazine may offer a useful balance between efficacy and tolerability in populations already exposed to substantial cardiometabolic risk (1,7).

This interpretation is clinically important because metabolic safety remains one of the most relevant determinants of long-term treatment success in psychiatry. Consensus recommendations on antipsychotic treatment have for many years emphasized that weight gain, disturbances in glucose homeostasis, and dyslipidemia are not merely secondary tolerability issues, but major contributors to morbidity, premature mortality, and treatment discontinuation (21). Likewise, guideline evaluations in schizophrenia have repeatedly stressed that cardiometabolic monitoring should be treated as an integral component of psychiatric care rather than an optional add-on (22). However, the practical implementation of such recommendations has often been incomplete, and systematic reviews of screening practices have shown that guideline-concordant metabolic monitoring remains suboptimal in routine care (23). Against this background, the present results are especially relevant, because they indicate that even when a treatment appears metabolically sparing, structured laboratory follow-up remains justified and necessary.

The studies included in the present review also need to be interpreted against the broader epidemiological background of severe mental illness. Independent meta-analytic evidence indicates that metabolic syndrome and related abnormalities are already highly prevalent in schizophrenia, even before considering the influence of specific pharmacological regimens (24). A similar pattern has been demonstrated in bipolar disorder, where the burden of metabolic syndrome and cardiometabolic risk factors is also markedly elevated (25). Moreover, when schizophrenia, bipolar disorder, and major depressive disorder are examined together, pooled evidence suggests that metabolic syndrome is not confined to one diagnostic group, but constitutes a broader somatic comorbidity of severe mental illness (26). This context is critical for interpreting the results of the present review. The relatively modest metabolic changes observed during cariprazine treatment should not be taken to mean that metabolic risk is absent in these patients. Rather, it suggests that cariprazine may not substantially add to an already existing vulnerability in the same way as antipsychotics with higher metabolic burden.

One of the most important findings of the present review is the consistency of observations across study designs. In schizophrenia, both individual randomized trials and pooled analyses pointed in the same direction. Short-term phase II and phase III studies did not indicate a marked signal of metabolic deterioration, and pooled analyses of acute treatment similarly suggested only modest changes in body weight and stable laboratory parameters (12–14). This consistency is methodologically important. When single studies, pooled acute analyses, and broader phase II/III syntheses all suggest a similar safety pattern, confidence in the robustness of the overall interpretation increases. In the case of cariprazine, the convergence of short-term evidence supports the conclusion that metabolic worsening is not a dominant feature of treatment during acute exacerbation of schizophrenia.

Long-term findings in schizophrenia were also generally coherent. Open-label extension studies and pooled post hoc long-term analyses did not reveal a convincing pattern of progressive metabolic deterioration during extended exposure (8–10,15). This point deserves emphasis because some antipsychotic-related metabolic complications become more apparent with time. Weight gain may accumulate gradually, and laboratory abnormalities may emerge only after months of treatment. For that reason, the absence of a clear

long-term signal in the available cariprazine studies is clinically reassuring. At the same time, it should be interpreted with caution. Open-label designs are inherently more vulnerable to bias than randomized blinded trials, and long-term extension studies may preferentially retain individuals who tolerate treatment well. Even so, the agreement between several long-duration datasets suggests that the relatively limited metabolic burden seen in short-term trials is not obviously reversed during longer treatment.

The pooled schizophrenia dataset by Barabássy et al. deserves special attention in this context because it offers one of the broadest summaries of metabolic and laboratory safety currently available for cariprazine (10). From the perspective of evidence synthesis, this kind of pooled analysis is particularly useful because it reduces the influence of isolated findings and provides a more stable estimate of the overall safety pattern. Its findings are also compatible with the current understanding that antipsychotics differ substantially in their metabolic effects and that not all second-generation agents should be discussed as a homogeneous class (1). In practical terms, this strengthens the argument that cariprazine may be especially relevant for patients in whom cardiometabolic risk is already a significant therapeutic consideration.

The bipolar disorder data included in this review broadly supported the same conclusion, although the evidence base was somewhat narrower than in schizophrenia. The pooled analysis in acute bipolar mania and the phase III trials in bipolar depression did not demonstrate a strong pattern of metabolic laboratory deterioration during treatment (17–19). This is an important observation because bipolar disorder is itself associated with increased metabolic risk, and long-term pharmacotherapy in this population is often complicated by weight gain, insulin resistance, and dyslipidemia from both illness-related and treatment-related factors (2,25,26). The fact that the currently available cariprazine studies in bipolar disorder do not suggest marked metabolic worsening is therefore encouraging. At the same time, the bipolar evidence remains less extensive than the schizophrenia data, particularly with regard to long-term laboratory outcomes. This means that the conclusion for bipolar disorder, although favorable, should still be considered somewhat less secure than the conclusion for schizophrenia.

Another relevant aspect of the present review is the inclusion of prolactin findings. Although prolactin is not a metabolic marker in the narrowest sense, it is an important laboratory parameter in the broader assessment of antipsychotic safety, particularly because endocrine adverse effects may significantly influence quality of life, adherence, and long-term treatment acceptability. The study by Culpepper et al. suggested that cariprazine exerts only minimal effects on prolactin concentrations (11). This finding aligns with the broader impression that cariprazine may have a comparatively benign endocrine profile. In clinical practice, this matters because prolactin-related adverse effects may become particularly burdensome in long-term therapy, even when they do not immediately threaten physical health in the same way as diabetes or severe dyslipidemia. Thus, from the perspective of a laboratory-monitoring framework, the prolactin data complement the metabolic findings and reinforce the overall impression of favorable tolerability.

The present review also has direct implications for monitoring practice. Existing consensus statements and reviews on antipsychotic-related cardiometabolic care consistently emphasize that monitoring should include baseline and follow-up assessment of weight, waist circumference when feasible, blood pressure, fasting glucose or HbA1c, and lipid parameters (21,22,27). More recent reviews continue to stress that cardiometabolic screening should remain systematic and longitudinal, especially in schizophrenia, where physical health care is often fragmented and insufficiently integrated into psychiatric treatment (28). The current results do not challenge these recommendations. On the contrary, they help refine them. The data do not support abandoning laboratory monitoring in patients treated with cariprazine; rather, they suggest that such monitoring may often confirm relative stability rather than detect frequent large abnormalities. This is still clinically useful, because treatment decisions are not made on average effects alone. Individual patients may differ substantially in baseline vulnerability, previous exposure to antipsychotics, family history, lifestyle, and comorbid medical illness.

The distinction between a relatively favorable metabolic profile and the absence of metabolic risk is particularly important. A medication may be less metabolically disruptive than other agents and still require structured surveillance. This principle is widely recognized in both psychiatric and medical recommendations, but in practice it is often undermined by low monitoring rates and incomplete follow-up (23,27). Implementation-focused studies have therefore attempted to improve screening and management of metabolic effects in antipsychotic-treated populations (29). From this perspective, the present review supports a two-part clinical message: first, cariprazine appears to be a reasonable option when metabolic safety is a therapeutic priority; second, its use should still be embedded in a routine framework of anthropometric and laboratory monitoring. These two statements are complementary, not contradictory.

There are several limitations in the evidence base included in this review, and these should be acknowledged clearly. First, many of the studies were not designed primarily to investigate metabolic outcomes. In several trials, metabolic data were reported as part of broader safety assessments rather than as prespecified primary endpoints. As a result, the level of detail was variable, and not all studies provided equally granular information on laboratory changes. Second, the included studies differed in design, treatment duration, populations, and analytical approach. Acute randomized trials, open-label extensions, and pooled post hoc analyses each contribute differently to the evidence base, but they are not methodologically equivalent. Third, the bipolar disorder evidence was less extensive than the schizophrenia evidence, especially with regard to longer-term follow-up. Fourth, the review relied largely on trial-based data, which may not fully reflect real-world populations characterized by multimorbidity, polypharmacy, and imperfect adherence.

A further interpretive limitation concerns the difficulty of separating illness-related metabolic vulnerability from medication-related effects. Meta-analytic evidence indicates that at least some metabolic abnormalities are already present in early or untreated stages of schizophrenia, although they become more prominent in chronic and medicated populations (30). This is highly relevant when assessing cariprazine, because the baseline metabolic state of psychiatric patients is rarely neutral. Consequently, a clinically meaningful question is not only whether cariprazine changes metabolic parameters, but whether it worsens them beyond the substantial background risk carried by the disorder itself and by previous treatment exposure. The currently available evidence suggests that such additional worsening is relatively limited, but this conclusion necessarily remains probabilistic rather than absolute.

The review itself also has limitations. It is based on a narrative synthesis rather than a formal quantitative meta-analysis. This means that although clear patterns were identified, the review cannot provide pooled effect estimates for weight change or laboratory outcomes. Moreover, the included studies varied in the exact metabolic measures reported and in the way safety data were summarized, which made strict quantitative comparison difficult. Another limitation is that some sources initially identified during the broader search process were available only as conference abstracts and were therefore excluded from the final analysis. This improved methodological rigor but may also have reduced the breadth of exploratory data considered. Finally, because the review focused specifically on laboratory-monitored metabolic safety, other important tolerability domains, such as akathisia or sedation, were not discussed in equal depth.

Despite these limitations, the present review has several strengths. It brings together evidence from schizophrenia and bipolar disorder within a single clinically focused framework. Instead of treating weight change, laboratory markers, and endocrine effects as isolated findings, it considers them together under the concept of laboratory monitoring of metabolic safety. This is important because treatment decisions in psychiatry are made in an integrated clinical context. For many patients, especially those with pre-existing obesity, diabetes risk, lipid abnormalities, or poor prior tolerability to other antipsychotics, a drug's metabolic profile may be as decisive as its symptomatic efficacy. By focusing specifically on monitored metabolic outcomes, this review provides information that is directly relevant to routine prescribing and follow-up.

The implications for the field are therefore both practical and conceptual. Practically, the findings suggest that cariprazine may be considered when metabolic tolerability is a key treatment priority in schizophrenia or bipolar disorder. Conceptually, the review illustrates the importance of moving beyond broad statements that a drug is "safe" or "well tolerated" and instead asking which specific laboratory-monitored parameters were assessed and how they changed over time. This approach is likely to become increasingly important as psychiatric care continues to emphasize personalized treatment, shared decision-making, and long-term physical health outcomes alongside symptom control.

In conclusion, the available evidence indicates that cariprazine is associated with a relatively favorable profile in the context of laboratory-monitored metabolic safety during treatment of schizophrenia and bipolar disorder. Across the included studies, the most consistent findings were modest body weight increase, generally stable glucose- and lipid-related parameters, and minimal prolactin elevation. The results are therefore compatible with the view that cariprazine has comparatively limited metabolic and endocrine burden. Nevertheless, this favorable profile does not eliminate the need for regular metabolic monitoring. Instead, it highlights the importance of maintaining structured laboratory surveillance while recognizing that cariprazine may represent a useful therapeutic option for patients in whom long-term metabolic safety is a major clinical concern.

Conclusions

The findings of this systematic review suggest that cariprazine is associated with a relatively favorable profile in the context of laboratory-monitored metabolic safety in patients with schizophrenia and bipolar disorder. Across the included studies, the most consistent observations were modest body weight increase, generally stable glucose- and lipid-related laboratory parameters, and minimal prolactin elevation. Although the overall evidence remains somewhat heterogeneous in terms of study design, duration, and reporting of metabolic outcomes, the available data consistently indicate that cariprazine is not associated with a pronounced pattern of metabolic deterioration during either short-term or long-term treatment.

These findings are clinically relevant because metabolic adverse effects remain one of the major challenges of antipsychotic therapy and a key factor influencing long-term treatment acceptability, adherence, and physical health outcomes. The results of the present review support the view that cariprazine may represent a useful therapeutic option in patients for whom cardiometabolic safety is an important consideration. At the same time, the relatively favorable metabolic profile of cariprazine should not be interpreted as eliminating the need for regular monitoring. Even treatments considered metabolically sparing require structured follow-up of body weight and laboratory parameters, particularly in populations already burdened by high baseline cardiometabolic risk.

Further research is needed to better define the long-term metabolic effects of cariprazine in real-world clinical settings, especially in bipolar disorder, where the evidence base remains less extensive than in schizophrenia. Future studies should place greater emphasis on prespecified metabolic endpoints, standardized reporting of glucose- and lipid-related parameters, and longer follow-up periods that allow more reliable assessment of cumulative cardiometabolic burden. It would also be valuable to investigate the influence of baseline metabolic status, previous antipsychotic exposure, comorbid medical conditions, and concomitant pharmacotherapy on metabolic outcomes during cariprazine treatment.

Overall, cariprazine appears to have a comparatively low metabolic and endocrine burden among antipsychotic treatment options, but it should not be regarded as exempt from laboratory surveillance. A clinically responsible approach to its use should combine the benefits of a favorable tolerability profile with consistent metabolic monitoring and individualized long-term care. Such an approach may help optimize both psychiatric stability and physical health outcomes in patients with schizophrenia and bipolar disorder.

REFERENCES

1. Pillinger, T., McCutcheon, R. A., Vano, L., Mizuno, Y., Arumuham, A., Hindley, G., Beck, K., Natesan, S., Efthimiou, O., Cipriani, A., & Howes, O. D. (2020). Comparative effects of 18 antipsychotics on metabolic function in patients with schizophrenia, predictors of metabolic dysregulation, and association with psychopathology: A systematic review and network meta-analysis. *The Lancet Psychiatry, 7*(1), 64–77. https://doi.org/10.1016/S2215-0366(19)30416-X
2. Yatham, L. N., Kennedy, S. H., Parikh, S. V., Schaffer, A., Bond, D. J., Frey, B. N., Sharma, V., Goldstein, B. I., Rej, S., Beaulieu, S., Alda, M., MacQueen, G., Milev, R. V., Ravindran, A., O'Donovan, C., McIntosh, D., Lam, R. W., Vazquez, G., Kapczinski, F., . . . Berk, M. (2018). Canadian Network for Mood and Anxiety Treatments (CANMAT) and International Society for Bipolar Disorders (ISBD) 2018 guidelines for the management of patients with bipolar disorder. *Bipolar Disorders, 20*(2), 97–170. https://doi.org/10.1111/bdi.12609
3. Selvan, P., Devkare, P., Shetty, A., Dharmadhikari, S., Khandhedia, C., Mane, A., Mehta, S., & Andrade, C. (2024). A review on the pharmacology of cariprazine and its role in the treatment of negative symptoms of schizophrenia. *Frontiers in Psychiatry, 15*, Article 1385925. https://doi.org/10.3389/fpsyt.2024.1385925
4. Misiak, B., Bieńkowski, P., & Samochowiec, J. (2018). Cariprazine—A novel antipsychotic drug and its place in the treatment of schizophrenia. *Psychiatria Polska, 52*(6), 971–981. https://doi.org/10.12740/PP/OnlineFirst/80710
5. Do, A., Keramatian, K., Schaffer, A., & Yatham, L. (2021). Cariprazine in the treatment of bipolar disorder: Within and beyond clinical trials. *Frontiers in Psychiatry, 12*, Article 769897. https://doi.org/10.3389/fpsyt.2021.769897
6. Vieta, E., McIntyre, R. S., Yu, J., Aronin, L. C., Kramer, K., & Nguyen, H. B. (2024). Full-spectrum efficacy of cariprazine across manic and depressive symptoms of bipolar I disorder in patients experiencing mood episodes: Post hoc analysis of pooled randomized controlled trial data. *Journal of Affective Disorders, 366*, 136–145. https://doi.org/10.1016/j.jad.2024.08.119
7. Lao, K. S., He, Y., Wong, I. C., Besag, F. M., & Chan, E. W. (2016). Tolerability and safety profile of cariprazine in treating psychotic disorders, bipolar disorder, and major depressive disorder: A systematic review with meta-analysis of randomized controlled trials. *CNS Drugs, 30*(11), 1043–1054. https://doi.org/10.1007/s40263-016-0382-z

8. Nasrallah, H. A., Earley, W., Cutler, A. J., Wang, Y., Lu, K., Laszlovszky, I., Németh, G., & Durgam, S. (2017). The safety and tolerability of cariprazine in long-term treatment of schizophrenia: A post hoc pooled analysis. *BMC Psychiatry, 17*, Article 305. https://doi.org/10.1186/s12888-017-1459-z
9. Cutler, A. J., Durgam, S., Wang, Y., Migliore, R., Lu, K., Laszlovszky, I., & Németh, G. (2018). Evaluation of the long-term safety and tolerability of cariprazine in patients with schizophrenia: Results from a 1-year open-label study. *CNS Spectrums, 23*(1), 39–50. https://doi.org/10.1017/S1092852917000220
10. Barabássy, Á., Sebe, B., Acsai, K., Laszlovszky, I., Szatmári, B., Earley, W. R., & Németh, G. (2021). Safety and tolerability of cariprazine in patients with schizophrenia: A pooled analysis of eight phase II/III studies. *Neuropsychiatric Disease and Treatment, 17*, 957–970. https://doi.org/10.2147/NDT.S301225
11. Culpepper, L., Vieta, E., Kelly, D. L., Patel, M. D., Szatmari, B., Hankinson, A., & Earley, W. R. (2022). Minimal effects of cariprazine on prolactin levels in bipolar disorder and schizophrenia. *Neuropsychiatric Disease and Treatment, 18*, 995–1011. https://doi.org/10.2147/NDT.S348143
12. Kane, J. M., Zukin, S., Wang, Y., Lu, K., Ruth, A., Nagy, K., Laszlovszky, I., & Durgam, S. (2015). Efficacy and safety of cariprazine in acute exacerbation of schizophrenia: Results from an international, phase III clinical trial. *Journal of Clinical Psychopharmacology, 35*(4), 367–373. https://doi.org/10.1097/JCP.0000000000000346
13. Earley, W., Durgam, S., Lu, K., Laszlovszky, I., Debelle, M., & Kane, J. M. (2017). Safety and tolerability of cariprazine in patients with acute exacerbation of schizophrenia: A pooled analysis of four phase II/III randomized, double-blind, placebo-controlled studies. *International Clinical Psychopharmacology, 32*(6), 319–328. https://doi.org/10.1097/YIC.0000000000000187
14. Durgam, S., Starace, A., Li, D., Migliore, R., Ruth, A., Németh, G., & Laszlovszky, I. (2014). An evaluation of the safety and efficacy of cariprazine in patients with acute exacerbation of schizophrenia: A phase II, randomized clinical trial. *Schizophrenia Research, 152*(2–3), 450–457. https://doi.org/10.1016/j.schres.2013.11.041
15. Durgam, S., Greenberg, W. M., Li, D., Lu, K., Laszlovszky, I., Németh, G., Migliore, R., & Volk, S. (2017). Safety and tolerability of cariprazine in the long-term treatment of schizophrenia: Results from a 48-week, single-arm, open-label extension study. *Psychopharmacology, 234*(2), 199–209. https://doi.org/10.1007/s00213-016-4450-3
16. Durgam, S., Earley, W., Lipschitz, A., Guo, H., Laszlovszky, I., Németh, G., Vieta, E., Calabrese, J. R., & Yatham, L. N. (2016). An 8-week randomized, double-blind, placebo-controlled evaluation of the safety and efficacy of cariprazine in patients with bipolar I depression. *The American Journal of Psychiatry, 173*(3), 271–281. https://doi.org/10.1176/appi.ajp.2015.15020164
17. Earley, W., Durgam, S., Lu, K., Laszlovszky, I., Debelle, M., Vieta, E., & Yatham, L. N. (2017). Tolerability of cariprazine in the treatment of acute bipolar I mania: A pooled post hoc analysis of 3 phase II/III studies. *Journal of Affective Disorders, 215*, 205–212. https://doi.org/10.1016/j.jad.2017.03.032
18. Earley, W., Burgess, M. V., Rekeda, L., Dickinson, R., Szatmári, B., Németh, G., McIntyre, R. S., Sachs, G. S., & Yatham, L. N. (2019). Cariprazine treatment of bipolar depression: A randomized double-blind placebo-controlled phase 3 study. *The American Journal of Psychiatry, 176*(6), 439–448. https://doi.org/10.1176/appi.ajp.2018.18070824
19. Earley, W. R., Burgess, M. V., Khan, B., Rekeda, L., Suppes, T., Tohen, M., & Calabrese, J. R. (2020). Efficacy and safety of cariprazine in bipolar I depression: A double-blind, placebo-controlled phase 3 study. *Bipolar Disorders, 22*(4), 372–384. https://doi.org/10.1111/bdi.12852
20. Keramatian, K., Chithra, N. K., & Yatham, L. N. (2023). The CANMAT and ISBD guidelines for the treatment of bipolar disorder: Summary and a 2023 update of evidence. *Focus, 21*(4), 344–353. https://doi.org/10.1176/appi.focus.20230009
21. American Diabetes Association, American Psychiatric Association, American Association of Clinical Endocrinologists, & North American Association for the Study of Obesity. (2004). Consensus development conference on antipsychotic drugs and obesity and diabetes. *The Journal of Clinical Psychiatry, 65*(2), 267–272. https://doi.org/10.4088/JCP.v65n0219
22. De Hert, M., Vancampfort, D., Correll, C. U., Mercken, V., Peuskens, J., Sweers, K., van Winkel, R., & Mitchell, A. J. (2011). Guidelines for screening and monitoring of cardiometabolic risk in schizophrenia: Systematic evaluation. *The British Journal of Psychiatry, 199*(2), 99–105. https://doi.org/10.1192/bjp.bp.110.084665
23. Mitchell, A. J., Delaffon, V., Vancampfort, D., Correll, C. U., & De Hert, M. (2012). Guideline concordant monitoring of metabolic risk in people treated with antipsychotic medication: Systematic review and meta-analysis of screening practices. *Psychological Medicine, 42*(1), 125–147. https://doi.org/10.1017/S003329171100105X
24. Mitchell, A. J., Vancampfort, D., Sweers, K., van Winkel, R., Yu, W., & De Hert, M. (2013). Prevalence of metabolic syndrome and metabolic abnormalities in schizophrenia and related disorders: A systematic review and meta-analysis. *Schizophrenia Bulletin, 39*(2), 306–318. https://doi.org/10.1093/schbul/sbr148
25. Vancampfort, D., Vansteelandt, K., Correll, C. U., Mitchell, A. J., De Herdt, A., Sienaert, P., Probst, M., & De Hert, M. (2013). Metabolic syndrome and metabolic abnormalities in bipolar disorder: A meta-analysis of prevalence rates and moderators. *The American Journal of Psychiatry, 170*(3), 265–274. https://doi.org/10.1176/appi.ajp.2012.12050620

26. Vancampfort, D., Stubbs, B., Mitchell, A. J., De Hert, M., Wampers, M., Ward, P. B., Rosenbaum, S., & Correll, C. U. (2015). Risk of metabolic syndrome and its components in people with schizophrenia and related psychotic disorders, bipolar disorder, and major depressive disorder: A systematic review and meta-analysis. *World Psychiatry, 14*(3), 339–347. https://doi.org/10.1002/wps.20252
27. Marder, S. R., Essock, S. M., Miller, A. L., Buchanan, R. W., Casey, D. E., Davis, J. M., Kane, J. M., Lieberman, J. A., Schooler, N. R., Covell, N., Stroup, T. S., Weissman, E. M., Wirshing, D. A., Hall, C. S., Pogach, L., Pi-Sunyer, X., Bigger, J. T., Jr., Friedman, A., Kleinberg, D., . . . Shon, S. (2004). Physical health monitoring of patients with schizophrenia. *The American Journal of Psychiatry, 161*(8), 1334–1349. https://doi.org/10.1176/appi.ajp.161.8.1334
28. Castle, D., & Li, A. (2023). Physical health monitoring for people with schizophrenia. *Australian Prescriber, 46*(4), 75–79. https://doi.org/10.18773/austprescr.2023.024
29. Owen, R. R., Drummond, K. L., Viverito, K. M., Marchant, K., Pope, S. K., Smith, J. L., Landes, R. D., Norman, S. R., Schoenbaum, M., & Kirchner, J. E. (2013). Monitoring and managing metabolic effects of antipsychotics: A cluster randomized trial of an intervention combining evidence-based quality improvement and external facilitation. *Implementation Science, 8*, Article 120. https://doi.org/10.1186/1748-5908-8-120
30. Mitchell, A. J., Vancampfort, D., De Herdt, A., Yu, W., & De Hert, M. (2013). Is the prevalence of metabolic syndrome and metabolic abnormalities increased in early schizophrenia? A comparative meta-analysis of first-episode, untreated, and treated patients. *Schizophrenia Bulletin, 39*(2), 295–305. https://doi.org/10.1093/schbul/sbs082