



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	SAFETY OF LONG-TERM USE OF BENZODIAZEPINES IN GERIATRIC PATIENTS – A REVIEW OF CURRENT EVIDENCE AND RECOMMENDATIONS
----------------------	---

DOI	https://doi.org/10.31435/ijitss.3(51).2026.5859
------------	---

RECEIVED	14 July 2026
-----------------	--------------

ACCEPTED	15 September 2026
-----------------	-------------------

PUBLISHED	18 September 2026
------------------	-------------------

LICENSE	
----------------	--



The article is licensed under a **Creative Commons Attribution 4.0 International License**.

© The author(s) 2026.

This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

SAFETY OF LONG-TERM USE OF BENZODIAZEPINES IN GERIATRIC PATIENTS – A REVIEW OF CURRENT EVIDENCE AND RECOMMENDATIONS

Iga Rusin (Corresponding Author, Email: iga.rusin@wp.pl)

4th Military Clinical Hospital with Polyclinic SP ZOZ in Wrocław, Rudolfa Weigla 5, 50-985 Wrocław, Poland
ORCID ID: 0009-0002-4736-6571

Amadea Wrzeńska

4th Military Clinical Hospital with Polyclinic SP ZOZ in Wrocław, Rudolfa Weigla 5, 50-985 Wrocław, Poland
ORCID ID: 0009-0001-5394-3446

Mateusz Korowacki

University Clinical Hospital in Wrocław, Borowska 213, 50-556 Wrocław, Poland
ORCID ID: 0009-0003-8100-3026

Michał Przybylski

4th Military Clinical Hospital with Polyclinic SP ZOZ in Wrocław, Rudolfa Weigla 5, 50-985 Wrocław, Poland
ORCID ID: 0009-0007-8717-7392

Bartosz Świrad

4th Military Clinical Hospital with Polyclinic SP ZOZ in Wrocław, Rudolfa Weigla 5, 50-985 Wrocław, Poland
ORCID ID: 0009-0008-8158-7697

Krzysztof Mączka

University Clinical Hospital in Wrocław, Borowska 213, 50-556 Wrocław, Poland
ORCID ID: 0000-0002-9747-7926

Karol Kazimierzak

4th Military Clinical Hospital with Polyclinic SP ZOZ in Wrocław, Rudolfa Weigla 5, 50-985 Wrocław, Poland
ORCID ID: 0009-0007-3748-5477

Daniel Grobecki

University Clinical Hospital in Wrocław, Borowska 213, 50-556 Wrocław, Poland
ORCID ID: 0009-0005-1724-7784

Filip Kruczek

4th Military Clinical Hospital with Polyclinic SP ZOZ in Wrocław, Rudolfa Weigla 5, 50-985 Wrocław, Poland
ORCID ID: 0009-0002-3082-1435

Jakub Dyczko

4th Military Clinical Hospital with Polyclinic SP ZOZ in Wrocław, Rudolfa Weigla 5, 50-985 Wrocław, Poland
ORCID ID: 0009-0006-6401-3945

ABSTRACT

Background: Benzodiazepines remain frequently prescribed in older adults despite long-standing concerns regarding their safety. Age-related pharmacokinetic and pharmacodynamic changes, combined with increased sensitivity of the central nervous system, may amplify adverse effects. This narrative review aims to summarize current evidence on the risks associated with long-term benzodiazepine use in geriatric populations and to outline contemporary clinical recommendations for deprescribing.

Results: Long-term benzodiazepine use in older adults is consistently associated with an increased risk of adverse outcomes, particularly falls and fractures, impaired cognitive function, and delirium. Evidence regarding dementia risk remains inconclusive, with observational studies suggesting a possible association, though confounding by indication cannot be excluded. Concomitant use with opioids significantly increases the risk of respiratory depression and mortality. Major clinical guidelines, including Beers Criteria and STOPP/START criteria, strongly recommend avoiding benzodiazepines in older adults, except for specific short-term indications. Deprescribing strategies, including gradual dose tapering and substitution with non-pharmacological interventions such as cognitive behavioral therapy for insomnia (CBT-I), have demonstrated effectiveness in reducing long-term use.

Conclusions: Current evidence indicates that long-term benzodiazepine use in geriatric patients is associated with significant and potentially preventable harms. Despite this, inappropriate prescribing remains common in clinical practice. Structured deprescribing strategies and increased adherence to clinical guidelines are essential to improving patient safety in older populations.

KEYWORDS

Benzodiazepines, Elderly, Geriatric Pharmacology, Falls, Delirium, Deprescribing, Cognitive Impairment

CITATION

Rusin, I., Wrzeńska, A., Korowacki, M., Przybylski, M., Świrad, B., Mączka, K., Kazimierzczak, K., Grobecki, D., Kruczek, F., & Dyczko, J. (2026). Safety of long-term use of benzodiazepines in geriatric patients – a review of current evidence and recommendations. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.5859](https://doi.org/10.31435/ijitss.3(51).2026.5859)

COPYRIGHT

© The author(s) 2026. This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

Introduction

Benzodiazepines are among the most commonly prescribed psychotropic medications worldwide, primarily used for the short-term management of anxiety, insomnia, and acute agitation. Despite clear recommendations limiting their duration of use, especially in older adults, long-term prescribing remains frequent in clinical practice [1,2]. This discrepancy between guidelines and real-world prescribing patterns is especially concerning in geriatric populations, who are more vulnerable to drug-related adverse outcomes due to age-associated physiological changes and multimorbidity.

Ageing is associated with reduced hepatic metabolism, decreased renal clearance, altered body composition with increased fat-to-lean mass ratio, and heightened central nervous system sensitivity to sedative-hypnotic agents [1,21]. These changes prolong drug half-life and increase the risk of accumulation and toxicity, even at standard therapeutic doses. As a result, older adults are particularly susceptible to the adverse effects of benzodiazepines, including sedation, impaired psychomotor function, and cognitive dysfunction.

Over the past two decades, accumulating evidence has linked benzodiazepine use in older adults with a range of clinically significant harms [7,10,15]. These include an increased risk of falls and fractures, primarily hip fractures, which are associated with substantial morbidity and mortality [8]. Additionally, concerns have been raised regarding potential associations with cognitive decline, delirium, and possibly dementia, although the latter remains controversial due to potential confounding factors in observational studies. The risk of respiratory depression and overdose is further amplified when benzodiazepines are used in combination with opioids or other central nervous system depressants [5].

In response to these concerns, multiple international guidelines, including the American Geriatrics Society Beers Criteria and the STOPP/START criteria, consistently recommend avoiding benzodiazepines in

older adults whenever possible. Despite these recommendations, deprescribing remains challenging in clinical practice due to physiological dependence, withdrawal symptoms, and continued prescribing for chronic insomnia and anxiety disorders [15,16].

Given the ongoing clinical relevance of this issue, a comprehensive synthesis of current evidence is warranted. This narrative review aims to evaluate the safety profile of long-term benzodiazepine use in geriatric patients, with particular emphasis on major adverse outcomes and current deprescribing recommendations.

Mechanism of Action and Pharmacokinetic Changes in the Elderly

Benzodiazepines exert their pharmacological effects through positive allosteric modulation of the gamma-aminobutyric acid type A (GABA_A) receptor complex in the central nervous system [20]. By binding to a specific benzodiazepine site located at the interface between the α and γ subunits of the receptor, they enhance the affinity of GABA for its receptor, increasing the frequency of chloride channel opening. This results in neuronal hyperpolarization and a generalized inhibitory effect on neuronal excitability, clinically manifesting as anxiolytic, sedative-hypnotic, muscle relaxant, and anticonvulsant properties.

While this mechanism is consistent across age groups, the clinical effects of benzodiazepines are significantly amplified in older adults due to age-related pharmacokinetic and pharmacodynamic changes. Pharmacokinetically, ageing is associated with reduced hepatic mass and decreased hepatic blood flow, which impair oxidative metabolism mediated primarily by cytochrome P450 enzymes [21]. This is relevant for long-acting benzodiazepines such as diazepam and chlordiazepoxide, which undergo hepatic phase I oxidation and generate active metabolites with prolonged half-lives.

In contrast, benzodiazepines primarily metabolized through phase II conjugation (e.g., lorazepam, oxazepam, temazepam) are less affected by hepatic aging processes; however, even these agents may demonstrate prolonged elimination due to reduced renal clearance and altered distribution. Age-related decline in glomerular filtration rate can contribute to delayed excretion of inactive metabolites, further increasing systemic exposure.

Changes in body composition also play a critical role [21]. Older adults typically exhibit an increased proportion of adipose tissue and decreased total body water. Because benzodiazepines are highly lipophilic, this shift leads to an increased volume of distribution and drug accumulation in fat compartments. Consequently, redistribution becomes prolonged, resulting in extended sedative effects even after single doses and significantly longer elimination half-lives with chronic use.

Pharmacodynamically, the central nervous system of elderly patients exhibits increased sensitivity to GABAergic agents [1]. This heightened sensitivity is thought to reflect age-related changes in receptor function, neuronal connectivity, and compensatory reductions in excitatory neurotransmission. Clinically, this translates into exaggerated sedative effects at lower serum concentrations, increasing the risk of oversedation, cognitive impairment, and psychomotor dysfunction.

Overall, the combination of altered drug metabolism, redistribution, elimination, and increased central nervous system sensitivity creates a pharmacological environment in which benzodiazepines have a narrower therapeutic window in older adults. These changes underpin the increased incidence of adverse outcomes observed in geriatric populations and form the basis for current recommendations to avoid or minimize their use in this group.

Clinical Risks of Long-Term Benzodiazepine Use in Older Adults

Cognitive Disorders and Dementia

One of the most extensively studied and debated potential risks of long-term benzodiazepine use in older adults is cognitive impairment, including possible associations with dementia [10,11,12]. Observational cohort studies have reported an association between chronic benzodiazepine exposure and an increased risk of cognitive decline and incident dementia. Proposed mechanisms include persistent GABAergic inhibition leading to reduced cognitive processing speed, impaired memory consolidation, and disruption of sleep architecture, especially slow-wave sleep, which is important for memory formation.

However, the evidence remains inconsistent [10]. Several large population-based studies and meta-analyses have demonstrated a modest association between benzodiazepine use and dementia, but this relationship is substantially weakened after adjusting for confounding factors such as anxiety, depression,

insomnia, and prodromal symptoms of dementia (reverse causality). In other words, benzodiazepines may be prescribed for early neuropsychiatric symptoms that are themselves early manifestations of neurodegenerative disease. This makes causal inference difficult. Consequently, while a potential association cannot be fully excluded, current evidence does not definitively establish benzodiazepines as an independent risk factor for dementia.

Falls and Fractures

Among all adverse outcomes, the strongest and most consistent evidence supports an association between benzodiazepine use and increased risk of falls and fractures in older adults [7,9]. Sedation, impaired balance, delayed reaction time, and psychomotor slowing significantly contribute to fall risk, exceptionally during nighttime awakening or in patients with baseline frailty.

Multiple large cohort studies and meta-analyses have demonstrated that both short-acting and long-acting benzodiazepines are associated with an increased risk of falls. This translates clinically into a higher incidence of fractures, especially hip fractures involving the femoral neck, which are of particular concern due to their association with loss of independence, prolonged hospitalization, and increased mortality in geriatric populations [8]. The risk appears dose-dependent and is highest during the initial weeks of therapy but remains elevated with chronic use.

Delirium

Benzodiazepines are recognized as important pharmacological contributors to delirium, especially in hospitalized older adults and emergency department settings [13]. Their sedative and amnestic effects, combined with central nervous system suppression, can precipitate or exacerbate acute confusional states.

The risk is especially pronounced in patients with pre-existing cognitive impairment, acute illness, dehydration, or polypharmacy. In intensive care and perioperative settings, benzodiazepines have been associated with longer duration of delirium and prolonged hospital stay [14]. As a result, many clinical guidelines now recommend minimizing benzodiazepine use in older hospitalized patients, reserving them primarily for alcohol or benzodiazepine withdrawal syndromes.

Dependence, Tolerance, and Withdrawal

Physiological dependence is a well-established consequence of prolonged benzodiazepine use, even at therapeutic doses [15]. Tolerance may develop over time, leading to dose escalation or reduced clinical efficacy, mainly for hypnotic effects in insomnia management. This contributes to a cycle of continued or increasing use despite diminishing benefit.

Withdrawal symptoms can be significant and include rebound anxiety, insomnia, irritability, tremor, perceptual disturbances, and, in severe cases, seizures. In older adults, discontinuation is often particularly challenging due to long duration of use, comorbid psychiatric conditions, and fear of symptom recurrence. These factors contribute to low deprescribing rates in clinical practice despite strong guideline recommendations .

Mortality

Several observational studies have reported an association between benzodiazepine use and increased all-cause mortality in older adults [18,19]. Proposed mechanisms include increased risk of falls, respiratory depression, and worsening of comorbid conditions such as chronic obstructive pulmonary disease.

However, the relationship between benzodiazepines and mortality remains controversial [18]. Confounding by indication is a major limitation, as patients prescribed benzodiazepines often have higher baseline morbidity, including anxiety disorders, insomnia, and frailty, which themselves increase mortality risk. Additionally, concomitant medication use and polypharmacy complicate causal interpretation.

Despite these limitations, the consistency of observational signals across multiple studies warrants clinical caution. Most guidelines therefore recommend minimizing benzodiazepine use in older adults, most importantly in those with multimorbidity or concomitant central nervous system depressant therapy.

Drug Interactions

Polypharmacy is highly prevalent in geriatric populations, making drug–drug interactions a clinically significant concern in patients receiving benzodiazepines. Age-related pharmacokinetic changes, combined with reduced physiological reserve, further amplify the clinical consequences of these interactions. Benzodiazepines are specifically problematic when combined with other central nervous system (CNS) depressants, drugs affecting hepatic metabolism, or medications that independently increase fall risk or cognitive impairment.

Central nervous system depressants

The most clinically important interactions occur with other CNS depressants, including opioids, alcohol, sedating antidepressants, antipsychotics, and first-generation antihistamines [5]. Concurrent use of benzodiazepines and opioids significantly increases the risk of respiratory depression, sedation, overdose, and mortality. This combination has been identified in multiple regulatory warnings, including those issued by the FDA and EMA, due to the additive or synergistic depressant effects on brainstem respiratory centers.

Alcohol potentiates the sedative and psychomotor impairing effects of benzodiazepines through additive GABAergic activity. Even moderate alcohol intake can markedly increase the risk of confusion, falls, and accidental injury in older adults. Similarly, tricyclic antidepressants, sedating selective serotonin reuptake inhibitors (e.g., paroxetine), and antipsychotics may contribute to excessive sedation and cognitive impairment when used concurrently.

Pharmacokinetic interactions via cytochrome P450 enzymes

Certain benzodiazepines, particularly those undergoing oxidative metabolism (e.g., diazepam, alprazolam, midazolam), are metabolized by hepatic cytochrome P450 isoenzymes, especially CYP3A4 and CYP2C19 [22]. Concomitant administration of CYP inhibitors can lead to increased serum concentrations and prolonged sedation.

Strong CYP3A4 inhibitors, such as macrolide antibiotics (e.g., clarithromycin), azole antifungals (e.g., ketoconazole), and certain calcium channel blockers (e.g., verapamil), may significantly reduce benzodiazepine clearance. Conversely, CYP inducers such as carbamazepine or rifampicin may reduce therapeutic efficacy, potentially leading to dose escalation and subsequent withdrawal risk when the inducer is discontinued.

Importantly, benzodiazepines primarily metabolized via phase II conjugation (e.g., lorazepam, oxazepam, temazepam) are less susceptible to CYP-mediated interactions, which is why they are generally preferred in elderly patients with polypharmacy or hepatic impairment.

Additive risk of falls and cognitive impairment

Beyond direct pharmacological interactions, benzodiazepines exhibit clinically relevant pharmacodynamic synergy with other medications that impair balance, cognition, or blood pressure regulation. Antihypertensive agents, mainly alpha-blockers and diuretics, may contribute to orthostatic hypotension, further increasing fall risk when combined with benzodiazepine-induced sedation and impaired coordination.

Similarly, anticholinergic medications can exacerbate benzodiazepine-associated cognitive dysfunction, contributing to delirium, memory impairment, and functional decline. In older adults, the cumulative anticholinergic burden is an important but often underrecognized factor that compounds benzodiazepine toxicity.

Clinical implications

The presence of multiple interacting medications significantly narrows the therapeutic index of benzodiazepines in older adults. These interactions not only increase the risk of acute adverse events such as falls, delirium, and respiratory depression, but also contribute to long-term functional decline and loss of independence. As a result, clinical guidelines strongly recommend avoiding benzodiazepines in combination with opioids and other sedatives whenever possible, and favoring agents with more predictable metabolism in patients requiring anxiolytic or hypnotic therapy.

Careful medication review, deprescribing strategies, and interdisciplinary management are essential components of reducing interaction-related harm in geriatric patients receiving benzodiazepines.

Clinical Guidelines

Beers Criteria (American Geriatrics Society)

The American Geriatrics Society (AGS) Beers Criteria represent one of the most widely recognized frameworks for identifying potentially inappropriate medications in older adults [1]. Benzodiazepines are consistently listed as medications to avoid in geriatric patients, regardless of duration of action, due to increased sensitivity and reduced metabolism in this population.

The Beers Criteria highlight several key risks, including cognitive impairment, delirium, falls, fractures, and motor vehicle accidents. Importantly, the guideline emphasizes that older adults exhibit increased pharmacodynamic sensitivity to benzodiazepines, making even standard doses potentially harmful. Exceptions are limited to specific indications such as seizure disorders, severe generalized anxiety disorder unresponsive to other therapies, and periprocedural sedation. However, even in these cases, use is recommended to be short-term and closely monitored.

STOPP/START Criteria

The STOPP/START criteria (Screening Tool of Older Persons' Prescriptions / Screening Tool to Alert to Right Treatment) provide a European-based framework for optimizing pharmacotherapy in older adults [2]. Within STOPP criteria, benzodiazepines are identified as potentially inappropriate when used for longer than four weeks, especially in the treatment of insomnia, agitation, or anxiety.

The STOPP guidelines specifically associate long-term benzodiazepine use with increased risk of falls, cognitive decline, and dependence. In contrast, the START criteria emphasize when pharmacological treatment may be underutilized; however, benzodiazepines are generally not included as recommended therapies for chronic conditions in older adults, reflecting their unfavorable risk–benefit profile.

Overall, STOPP/START reinforces the principle that benzodiazepines should be avoided in long-term therapy and replaced with safer pharmacological or non-pharmacological alternatives whenever possible.

NICE Guidelines (United Kingdom)

The National Institute for Health and Care Excellence (NICE) provides clear recommendations regarding benzodiazepine use, particularly in the management of insomnia and anxiety disorders [3,4]. NICE guidelines strongly advise against routine long-term use of benzodiazepines due to risks of tolerance, dependence, and adverse cognitive and psychomotor effects.

For insomnia, NICE recommends non-pharmacological interventions such as cognitive behavioral therapy for insomnia (CBT-I) as first-line treatment. Benzodiazepines, if used at all, should be prescribed at the lowest effective dose for the shortest possible duration, typically not exceeding 2–4 weeks.

In anxiety disorders, NICE similarly prioritizes psychological therapies and antidepressants over benzodiazepines, which are reserved for short-term crisis management only. This approach reflects growing concern regarding dependency and functional impairment in older adults.

EMA and FDA Safety Warnings

Regulatory agencies such as the European Medicines Agency (EMA) and the U.S. Food and Drug Administration (FDA) have issued strong safety warnings regarding benzodiazepine use, particularly in combination with other central nervous system depressants [5,6].

Both agencies emphasize the increased risk of profound sedation, respiratory depression, coma, and death when benzodiazepines are used concurrently with opioids or alcohol. As a result, boxed warnings have been introduced in many countries to highlight these risks.

Additionally, regulatory communications stress the dangers of long-term use, including dependence, withdrawal syndromes, cognitive impairment, and increased risk of falls in older adults. These warnings have contributed to updated prescribing restrictions and increased emphasis on deprescribing strategies in clinical practice.

Clinical Implications

Across all major guidelines, there is a strong and consistent consensus: benzodiazepines should be avoided in older adults whenever possible and, if necessary, used only at the lowest effective dose for the shortest duration. The convergence of recommendations from AGS, STOPP/START, NICE, and regulatory agencies such as EMA and FDA underscores the well-established risk profile of these medications in geriatric populations.

Therapeutic Alternatives

Given the well-documented risks associated with long-term benzodiazepine use in older adults, current clinical practice increasingly emphasizes safer pharmacological and non-pharmacological alternatives. The choice of therapy depends on the underlying indication—most commonly insomnia, anxiety disorders, or neuropathic symptoms—and should be individualized according to comorbidities and functional status.

Cognitive Behavioral Therapy for Insomnia (CBT-I)

Cognitive Behavioral Therapy for Insomnia (CBT-I) is considered the first-line treatment for chronic insomnia in both general and geriatric populations [23,24]. It addresses maladaptive behaviors and cognitive patterns that perpetuate sleep disturbance, including sleep-related anxiety, poor sleep hygiene, and conditioned arousal.

CBT-I typically includes sleep restriction, stimulus control, cognitive restructuring, and relaxation techniques. Evidence consistently demonstrates that CBT-I is as effective as pharmacotherapy in the short term and superior in maintaining long-term sleep improvement. Importantly, it avoids the risks of sedation, cognitive impairment, tolerance, and dependence associated with benzodiazepines, making it suitable for older adults.

Selective Serotonin Reuptake Inhibitors (SSRIs) and Serotonin-Noradrenaline Reuptake Inhibitors (SNRIs)

SSRIs and SNRIs are first-line pharmacological treatments for generalized anxiety disorder and depressive disorders, conditions frequently associated with benzodiazepine prescribing [3]. Unlike benzodiazepines, these agents do not cause dependence and are safer for long-term use.

Commonly used SSRIs include sertraline and escitalopram, while SNRIs such as venlafaxine and duloxetine may be preferred in patients with comorbid pain syndromes. However, in older adults, these medications must be introduced cautiously due to potential adverse effects such as hyponatremia, gastrointestinal symptoms, and initial activation anxiety. Despite these limitations, their risk profile is generally more favorable than that of benzodiazepines in chronic use.

Melatonin and Melatonin Receptor Agonists

Melatonin is increasingly used as a safer alternative for insomnia in older adults, due to age-related reductions in endogenous melatonin secretion [26]. Exogenous melatonin can improve sleep onset latency and circadian rhythm regulation with a relatively benign side effect profile.

Prolonged-release formulations may offer modest benefits in sleep maintenance insomnia. Importantly, melatonin does not cause respiratory depression, cognitive impairment, or dependence, making it a preferred option in geriatric patients where benzodiazepines are contraindicated or should be avoided.

Pregabalin

Pregabalin is sometimes used as an alternative for generalized anxiety disorder or neuropathic pain, conditions that may otherwise lead to benzodiazepine prescribing [25]. It modulates calcium channels and reduces excitatory neurotransmitter release, producing anxiolytic and analgesic effects.

While pregabalin does not carry the same dependence risk as benzodiazepines, it is not without limitations. Adverse effects such as dizziness, somnolence, peripheral edema, and increased risk of falls must be carefully considered, particularly in frail older adults. Additionally, there is emerging evidence of misuse potential, necessitating cautious prescribing and monitoring.

Deprescribing Protocols

Deprescribing benzodiazepines is a critical component of safe pharmacotherapy in geriatric patients [15]. Abrupt discontinuation is not recommended due to the risk of withdrawal symptoms, including rebound insomnia, anxiety, agitation, and, in severe cases, seizures.

Effective deprescribing protocols typically involve gradual dose reduction (tapering), often decreasing the dose by 10–25% every 1–2 weeks, depending on patient tolerance and duration of use [15]. In some cases, switching to a longer-acting benzodiazepine may facilitate tapering. Adjunctive support with CBT-I, patient education, and close clinical follow-up significantly improves success rates.

Multidisciplinary involvement, including primary care physicians, pharmacists, and mental health professionals, has been shown to enhance adherence to deprescribing plans and reduce relapse rates [16]. Importantly, patient engagement and reassurance are essential, as fear of symptom recurrence is a major barrier to discontinuation.

Clinical Perspective

Overall, non-pharmacological interventions such as CBT-I remain the cornerstone of treatment, while pharmacological alternatives like SSRIs, SNRIs, melatonin, and selected agents such as pregabalin may be used selectively. Deprescribing strategies are essential to reduce long-term harm and should be systematically integrated into routine geriatric care.

Conclusions

Current evidence consistently indicates that long-term benzodiazepine use in geriatric patients lacks a favorable safety profile [7,10,15]. Although these agents may provide short-term symptomatic relief in anxiety and insomnia, their prolonged use is associated with a range of clinically significant adverse outcomes, including cognitive impairment, increased risk of falls and fractures, delirium, and clinically relevant drug interactions. Importantly, the potential association with dementia and increased mortality, while still partly controversial due to limitations of observational data, further strengthens concerns regarding long-term safety.

A key issue highlighted in this review is the absence of demonstrated long-term therapeutic benefit that outweighs these risks in older adults. Age-related pharmacokinetic and pharmacodynamic changes significantly narrow the therapeutic index of benzodiazepines, making prolonged use hazardous in this population. As such, chronic prescribing cannot be considered a safe or evidence-based strategy in geriatric care.

There is a clear and growing need for systematic deprescribing of benzodiazepines in older adults. Evidence-based tapering protocols, combined with appropriate non-pharmacological interventions such as cognitive behavioral therapy, as well as safer pharmacological alternatives when necessary, should be prioritized in clinical practice. Deprescribing should be viewed not as treatment withdrawal, but as an active, structured, and patient-centered therapeutic intervention aimed at reducing harm and improving functional outcomes.

Finally, education of both healthcare professionals and patients plays a central role in addressing inappropriate long-term use. Clinicians require greater awareness of guideline recommendations and the cumulative risks associated with chronic benzodiazepine therapy, while patients should be informed about potential harms and supported throughout the discontinuation process. Improving prescribing practices through education, guideline adherence, and multidisciplinary collaboration is essential to reducing the burden of benzodiazepine-related harm in the aging population.

Authors contributions:

Conceptualization: Daniel Grobecki, Krystian Mączka, Karol Kazimierczak

Methodology: Bartosz Świrad, Mateusz Korowacki, Filip Kruczek

Software: Krystian Mączka, Jakub Dyczko, Filip Kruczek, Michał Przybylski

Check: Bartosz Świrad, Mateusz Korowacki, Amadea Wrzesińska

Formal analysis: Daniel Grobecki, Jakub Dyczko, Karol Kazimierczak

Investigation: Krystian Mączka, Mateusz Korowacki, Karol Kazimierczak

Resources: Bartosz Świrad, Jakub Dyczko, Amadea Wrzesińska, Michał Przybylski

Data curation: Daniel Grobecki, Iga Rusin, Michał Przybylski

Writing- rough preparation: Mateusz Korowacki, Iga Rusin, Karol Kazimierczak,

Writing- review and editing: Krystian Mączka, Filip Kruczek, Amadea Wrzesińska

Visualization: Bartosz Świrad, Jakub Dyczko, Amadea Wrzesińska

Supervision: Daniel Grobecki, Iga Rusin, Filip Kruczek

Project Administration: Krystian Mączka, Iga Rusin, Michał Przybylski

Receiving funding: Not applicable - no specific funding.

All authors have read and agreed with the published version of the manuscript.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors **Institutional Review Board Statement:** Not applicable.

Institutional Review Board Statement: This study did not require Institutional Review Board approval because it involved no identifiable human data and was based solely on publicly available sources.

Data Availability Statement: Data sharing is not applicable in this article as no new data were created or analyzed in this article.

Conflicts of Interest: The authors declare no conflict of interest.

During the preparation of this work, the authors used Gemini AI for the purpose of improving the stylistic quality of the text and standardizing citations. After using this tool/service, the authors reviewed and edited the content as needed and takes full responsibility for the substantive content of the publication.

REFERENCES

1. American Geriatrics Society Beers Criteria® Update Expert Panel. (2023). American Geriatrics Society 2023 updated AGS Beers Criteria®. *J Am Geriatr Soc*, 71(7), 2052–2081.
2. O'Mahony, D., O'Sullivan, D., Byrne, S., et al. (2023). STOPP/START criteria version 3. *Age Ageing*, 52(1), afac649.
3. National Institute for Health and Care Excellence (NICE). (2020). *Generalised anxiety disorder and panic disorder (CG113)*. 2020 update.
4. National Institute for Health and Care Excellence (NICE). (n.d.). *Insomnia management (CG66)*. Updated guidance.
5. U.S. Food and Drug Administration (FDA). (2016). *Boxed warning: Opioid and benzodiazepine combination*.
6. European Medicines Agency (EMA). (n.d.). *Benzodiazepine safety and dependence warnings*. PRAC reports.
7. Woolcott, J. C., Richardson, K. J., Wiens, M. O., et al. (2009). Meta-analysis of medications and falls. *Arch Intern Med*, 169, 1952–1960.
8. Donnelly, K., Bracchi, R., Hewitt, J., et al. (2017). Benzodiazepines and hip fracture risk. *PLoS One*, 12, e0174730.
9. Leipzig, R. M., Cumming, R. G., & Tinetti, M. E. (1999). Drugs and falls in older people: Systematic review. *J Am Geriatr Soc*, 47, 30–39.
10. Gray, S. L., Dublin, S., Yu, O., et al. (2016). Benzodiazepine use and dementia risk. *BMJ*, 352, i90.
11. Zhong, G., Wang, Y., Zhang, Y., & Zhao, Y. (2015). Benzodiazepines and dementia: Meta-analysis. *PLoS One*, 10, e0127836.
12. Billioti de Gage, S., Moride, Y., Ducruet, T., et al. (2014). Benzodiazepine use and Alzheimer's disease. *BMJ*, 349, g5205.
13. Inouye, S. K., Westendorp, R. G. J., & Saczynski, J. S. (2014). Delirium in elderly people. *N Engl J Med*, 370, 1369–1377.
14. Pandharipande, P. P., Girard, T. D., Jackson, J. C., et al. (2013). Long-term cognitive impairment after ICU delirium. *N Engl J Med*, 369, 1306–1316.
15. Pottie, K., Thompson, W., Davies, S., et al. (2018). Deprescribing benzodiazepines guideline. *Can Fam Physician*, 64, 339–351.
16. Tannenbaum, C., Martin, P., Tamblyn, R., et al. (2014). Reduction of inappropriate benzodiazepine use. *JAMA Intern Med*, 174(6), 890–898.
17. Vicens, C., Bejarano, F., Sempere, E., et al. (2014). Withdrawal from long-term benzodiazepine use. *Br J Gen Pract*, 64, e563–e571.
18. Weich, S., Pearce, H. L., Croft, P., et al. (2014). Benzodiazepine use and mortality. *BMJ*, 348, g1996.
19. Kripke, D. F., Langer, R. D., & Kline, L. E. (2012). Hypnotics and mortality risk. *BMJ Open*, 2, e000850.
20. Shader, R. I., & Greenblatt, D. J. (1993). Use of benzodiazepines in anxiety disorders. *N Engl J Med*, 328, 1398–1405.
21. Greenblatt, D. J., Harmatz, J. S., & Shader, R. I. (1991). Pharmacokinetics of benzodiazepines in elderly. *Clin Pharmacokinet*, 21, 167–177.
22. Olkkola, K. T., & Ahonen, J. (2008). Midazolam and CYP3A interactions. *Clin Pharmacokinet*, 47, 749–766.
23. Morin, C. M., & Benca, R. (2012). Chronic insomnia. *Lancet*, 379, 1129–1141.
24. Qaseem, A., Kansagara, D., Forciea, M. A., et al. (2016). Management of chronic insomnia. *Ann Intern Med*, 165, 125–133.
25. Wafford, K. A., & Ebert, B. (2008). Gabapentin and pregabalin pharmacology. *CNS Drug Rev*, 14, 61–75.
26. Wade, A. G., Ford, I., Crawford, G., et al. (2007). Efficacy of prolonged-release melatonin in elderly. *Curr Med Res Opin*, 23, 2597–2605.