



Geriatrics · Educational evidence summary

Anticholinergic burden and cognition in aging

This geriatric evidence summary examines anticholinergic burden and cognition in aging.

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“Cognition” includes several different outcomes

Memory complaints, attention during an acute illness, performance on a cognitive test and a later dementia diagnosis are not interchangeable endpoints. A study that examines one of them does not establish the same relationship for all the others. The first step in reading anticholinergic evidence is to identify exactly which cognitive outcome was measured.

Exposure also has a time dimension. Current use, recent administration, repeated use over years and a remote prescription history describe different questions. A source-scale category attached to one medicine cannot summarize all of those histories without additional data and a defined method.

What evidence is considered here

This is a selected narrative review centered on the [prospective cumulative-exposure study by Gray and colleagues](#), the [inpatient diphenhydramine study](#), and the [randomized trial of anticholinergic burden reduction](#). The studies illustrate long-term association, an acute-care medication concern and an intervention question, respectively. No new quantitative synthesis or systematic search is claimed.

The [2023 AGS criteria](#) provide additional geriatric context. UnifiedACB’s source observations supply the medication-classification layer. These roles should remain separate: a criterion, a cohort finding and a source-scale value are not different forms of the same result.

Long-term exposure studies ask a historical question

Gray and colleagues examined cumulative strong-anticholinergic use in relation to incident dementia. The study is relevant because it considered exposure over time rather than merely whether a medicine appeared on one current list. Its reported association should retain that cumulative-use context.

It would be an overextension to turn that finding into a prediction that one dose causes dementia, or that every medication with a similar category has an identical long-term effect. The exposure



definition, drug set, follow-up and outcome ascertainment are part of the result. A current directory entry does not reproduce the study's longitudinal medication history.

Historical prescription data also have limits. Dispensing or prescribing does not prove every dose was taken, and medicines obtained outside the recorded system may be missed. These measurement questions can affect the apparent exposure and should be considered when interpreting a study or designing a new analysis.

Association does not remove alternative explanations

The conditions leading to treatment can be related to later cognitive outcomes. Symptoms may also precede a formal diagnosis and influence prescribing. These possibilities create confounding and timing problems that observational studies attempt to address but cannot erase simply by reporting an adjusted association.

This does not make the evidence unimportant. It changes the appropriate wording and the degree of certainty. "Associated with later dementia in this cohort" accurately preserves the design. "This medicine caused dementia" is a stronger claim requiring evidence beyond a source category or a single observational result.

The same caution applies when comparing medication classes. Differences in the people receiving treatment, the indication and exposure duration can influence comparisons. A numerical difference between groups is not automatically a pharmacological ranking of the medicines themselves.

Short-term testing has its own context

A cognitive test result reflects performance at the time of testing. Acute illness, fatigue, mood, sensory limitations and medication timing can influence what is observed. An exposure question should therefore be connected to the testing conditions rather than interpreted solely from a medication list.

The [hospitalized-patient study](#) illustrates why an acute-care context matters. An inpatient finding should not be treated as an estimate of every older adult's long-term cognitive trajectory. Conversely, a long-term dementia study does not diagnose the cause of a sudden change during hospitalization.

For a clinician reviewing a concern, a documented baseline and timeline can be more informative than a decontextualized total. The evidence table helps identify plausible exposures; the clinical assessment determines which cognitive question is actually being asked.

Does lowering burden improve cognition?

That is an intervention question. A study must show what was changed, what comparison was used, how cognition was measured and whether follow-up was adequate. A lower medication score is a process or exposure outcome. Cognitive improvement is a separate result that must be observed.

The [randomized frail-older-adult trial](#) is relevant to this distinction. Its findings should be read according to the tested intervention and endpoints, rather than converted into a general promise



about every medication change. Failure to demonstrate a particular benefit also does not establish that medication review has no value in every other context.

Clinical decisions additionally involve the benefit of treatment and the consequences of changing it. Cognitive concerns may be one part of that discussion, alongside the condition being treated and the person's priorities. A website classification cannot perform that balancing step by itself.

A subjective complaint and a standardized test result should also remain distinguishable in a summary. They may both be clinically important, but they are different observations. An article should state which was measured rather than replace either with the broader word "cognition" and imply that all cognitive outcomes were assessed.

How UnifiedACB supports the discussion

The [public source table](#) shows what individual scales report and where they differ. Ask can explain those classifications and the distinction between source evidence and supplemental geriatric content. It should not translate a category into a personal dementia percentage or imply that absent source data are reassuring.

Useful questions include which exposure period a cited study evaluated, whether the medication and route match the source, and what outcome the evidence actually measured. These questions preserve the scientific meaning of the evidence while making the clinical conversation more concrete.

The appropriate conclusion

Anticholinergic exposure deserves careful attention in cognitive assessment, particularly when the exposure history and clinical context are clearly documented. The evidence supports a focused review, not a deterministic prediction. Keep acute cognition, long-term dementia association and intervention benefit as separate claims, each tied to the study that examined it.

References

1. Fox C, et al. Effect of medications with anti-cholinergic properties on cognitive function, delirium, physical function and mortality: a systematic review. *Age Ageing*. 2014;43:604-615. [Source](#)
2. Vennard O, Stewart C, Tolia M, Soiza RL, Myint PK. Anticholinergic Medication Burden Scales: A Systematic Review. *J Am Geriatr Soc*. 2026;74(6):1771-1784. [Source](#)
3. American Geriatrics Society 2023 Updated AGS Beers Criteria for Potentially Inappropriate Medication Use in Older Adults. *J Am Geriatr Soc*. 2023;71:2052-2081. [Source](#)
4. American Geriatrics Society Beers Criteria Alternatives Panel. Alternative Treatments to Selected Medications in the 2023 AGS Beers Criteria. *J Am Geriatr Soc*. 2025;73:2657-2677. [Source](#)
5. Ruxton K, Woodman RJ, Mangoni AA. Drugs with anticholinergic effects and cognitive impairment, falls and all-cause mortality in older adults: a systematic review and meta-analysis. *Br J Clin Pharmacol*. 2015;80(2):209-220. doi:10.1111/bcp.12617. [Source](#)
6. Egberts A, et al. Anticholinergic Drug Burden and Delirium: A Systematic Review. *J Am Med Dir Assoc*. 2021;22:65-73.e4. [Source](#)



7. Gray SL, et al. Cumulative use of strong anticholinergics and incident dementia: a prospective cohort study. JAMA Intern Med. 2015;175:401–407. doi:10.1001/jamainternmed.2014.7663. [Source](#)
8. Agostini JV, Leo-Summers LS, Inouye SK. Cognitive and other adverse effects of diphenhydramine use in hospitalized older patients. Arch Intern Med. 2001;161:2091–2097. [Source](#)
9. Kersten H, et al. Cognitive effects of reducing anticholinergic drug burden in a frail elderly population: a randomized controlled trial. J Gerontol A Biol Sci Med Sci. 2013;68:271–278. doi:10.1093/gerona/gls176. [Source](#)
10. UnifiedACB. Preserved public source-observation dataset, v3.0 RC12 final; inspected for this editorial revision. [Source](#)

Explore the evidence

[Ask UnifiedACB](#) about a medication or source, browse the [Medication Library](#), or read the [methodology](#).

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