Glucosamine/Chondroitin Tied to Faster AD Progression

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For patients with early Alzheimer's disease (AD) or those at high risk, the use of over-the-counter joint supplements such as glucosamine and chondroitin has been a common practice, often without clear evidence of cognitive benefit. New data suggests that far from being benign, these widely used supplements may be associated with an accelerated rate of AD progression, presenting a critical consideration for clinicians advising patients on supplement use.

KEY TAKEAWAYS

- The Pivot: Common joint supplements, previously considered inert regarding cognition, are now implicated in AD progression.
- The Data: Patients using glucosamine/chondroitin showed a significantly faster decline in cognitive scores compared to non-users.
- The Action: Clinicians should counsel patients with AD or at risk of AD about the potential adverse cognitive effects of glucosamine and chondroitin.

The widespread availability and perceived safety of dietary supplements, including those for joint health, often lead to their uncritical adoption by patients. Glucosamine and chondroitin, frequently combined, are among the most popular non-prescription interventions for osteoarthritis symptoms. Their mechanism of action is thought to involve supporting cartilage structure and reducing inflammation. However, their systemic effects, particularly on neurological health, have received less scrutiny. Given the increasing prevalence of Alzheimer's disease and the imperative to identify modifiable risk factors or exacerbating agents, understanding the full profile of commonly used supplements is essential. The global burden of Alzheimer's disease continues to rise, posing significant challenges to healthcare systems and individual well-being. Identifying factors that influence the trajectory of cognitive decline, even subtle ones, holds considerable clinical importance. Patients often seek complementary therapies for various conditions, and the potential for interactions or unintended consequences with existing neurological conditions warrants thorough investigation.

What the study did

A retrospective cohort study analysed data from a large neuroimaging and clinical database, including patients with a diagnosis of mild cognitive impairment (MCI) or early-stage Alzheimer's disease. The study identified participants who reported regular use of glucosamine and/or chondroitin supplements for at least 12 months prior to baseline assessment. Cognitive function was assessed using standardised scales, including the Mini-Mental State Examination (MMSE) and the Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog), at baseline and annually over a follow-up

period of up to 5 years. Brain imaging data, specifically hippocampal volume and amyloid beta plaque load, were also collected at regular intervals for a subset of participants. The primary outcome was the rate of decline in ADAS-Cog scores, with secondary outcomes including MMSE decline and changes in neuroimaging biomarkers. Confounding factors such as age, sex, education level, APOE ε4 status, and baseline cognitive scores were adjusted for in the statistical models. The study's selection criteria ensured that participants had established cognitive impairment, allowing for the observation of progression rather than incidence. Data collection involved trained clinicians and neuropsychologists, ensuring consistency in cognitive assessments. Neuroimaging protocols adhered to established standards for volumetric analysis and amyloid PET quantification, providing objective measures of brain pathology. The longitudinal design was crucial for assessing rates of change over time, which is more informative than single-point comparisons in progressive neurodegenerative diseases.

The analysis included N=1,542 participants, of whom N=312 reported consistent use of glucosamine and/or chondroitin. The mean age of the cohort was 72.3 years (SD 8.1), and 58% were female. At baseline, there were no statistically significant differences in mean ADAS-Cog or MMSE scores between supplement users and non-users after adjusting for covariates (P>.05). However, over the follow-up period, participants using glucosamine/chondroitin demonstrated a significantly faster rate of decline in ADAS-Cog scores compared to non-users. The annualised increase in ADAS-Cog score (indicating worsening cognition) was +2.1 points (95% CI 1.8-2.4) for supplement users versus +1.4 points (95% CI 1.2-1.6) for non-users (P50% acceleration in cognitive decline. Similarly, the annualised decline in MMSE scores was more pronounced in the supplement user group, with a mean decrease of -1.8 points (95% CI -2.0 to -1.6) compared to -1.1 points (95% CI -1.3 to -0.9) in non-users (PNeuroimaging data from a subset of N=450 participants showed that glucosamine/chondroitin users exhibited a more rapid rate of hippocampal atrophy, with an annualised volume reduction of -2.5% (95% CI -2.9% to -2.1%) compared to -1.8% (95% CI -2.1% to -1.5%) in non-users (P=.003). No significant differences were observed in the rate of amyloid beta plaque accumulation between the two groups. The study's limitations include its observational design, which precludes definitive conclusions regarding causality. Residual confounding, despite extensive adjustments, remains a possibility. Furthermore, the self-reported nature of supplement use may introduce recall bias, and dosage consistency could not be precisely verified. Future prospective, randomised controlled trials are warranted to confirm these findings and elucidate potential underlying biological mechanisms. The lack of information on specific formulations, purity, and co-ingestion of other supplements represents an additional limitation. While the study adjusted for several key demographic and genetic factors, other lifestyle variables, such as diet, physical activity, and comorbidities not captured in the database, could potentially influence both supplement use and cognitive decline. The generalizability of these findings may also be limited to populations with similar demographic and clinical characteristics to those included in the database.

CLINICAL IMPLICATIONS

This analysis presents a sobering counterpoint to the prevailing assumption that joint supplements are harmless, particularly in a vulnerable population. For clinicians managing patients with early AD or MCI, the data suggests a need to actively inquire about glucosamine and chondroitin use. While the evidence is observational, the magnitude of the accelerated cognitive decline warrants a cautious approach. Advising patients to discontinue these supplements, especially if their primary indication is mild arthralgia, seems a

prudent step given the potential for harm and the lack of established cognitive benefit.

The broader implications extend to the supplement industry and regulatory bodies. The 'natural' label often confers an unwarranted sense of safety, leading to widespread consumption without rigorous scrutiny of long-term effects, especially in complex disease states like AD. This study underscores the necessity for more stringent pre-market evaluation and post-market surveillance of dietary supplements, particularly when used by individuals with chronic conditions or those taking multiple prescription therapies. The current regulatory framework, which largely exempts supplements from the rigorous testing required for prescription medicines, leaves a significant gap in patient protection.

Patients, often seeking any perceived advantage in the face of a devastating diagnosis, may be inadvertently exacerbating their condition. This highlights the critical role of primary care physicians and specialists in providing evidence-based guidance. The conversation should shift from merely asking about prescription medications to a comprehensive review of all ingested substances, including over-the-counter supplements. Educating patients on the potential risks, rather than just the purported benefits, of such products is now an essential component of AD management.

EDITORIAL & AI STANDARDS

AI tools are used to structure and summarise evidence from peer-reviewed primary sources. Articles that carry a signed Editor's take have been read in full by William Lopes. The others are not individually read by an editor, so check the cited source before relying on a figure.

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