



DOSSIER · SUBSTANCE

Ginkgo Biloba

Ginkgo biloba is one of the most extensively studied botanical active substances. The evidence base centres on **cognitive function** and **dementia**, though the findings are heterogeneous. The most frequently cited studies date from the 1990s and 2000s, while more recent research rarely achieves high citation rates.

913

STUDIES IN
TOTAL

490

IN HUMANS

257

OF WHICH
CONTROLLED

21

IN ANIMALS

81

IN THE LAB

Figures from the IHR study database · Last updated 2026-10-15

How to Read This Dossier

ESTABLISHED

measured in humans, with a comparison group

PLAUSIBLE

supported by mechanism, animal studies or observation

HYPOTHESIS

a likely connection that still needs to be tested

Ginkgo biloba at a glance

ORIGIN:

Ginkgo biloba is a standardised extract derived from the leaves of the ginkgo tree (*Ginkgo biloba* L.), one of the oldest living tree species.

STUDY SITUATION:

913 evaluated publications · 257 controlled human studies · 233 observational studies · 59 review articles · 21 animal studies · 70 cell culture studies

STRONGEST EVIDENCE:

- **Cognition and mental health:** in dementia (Alzheimer's type and vascular) Several randomised, placebo-controlled, double-blind studies (24 weeks) show that **EGb 761** (240 mg/day) significantly improves cognition (SKT, MMSE), activities of daily living (ADL) and neuropsychiatric symptoms (NPI) in mild to moderate dementia. One study confirms comparable efficacy to donepezil, albeit with a more favourable side-effect profile.
- **Safety and tolerability** Meta-analyses and systematic reviews show a favourable safety profile for **EGb 761** in mild to moderate dementia. The most common side effects are mild gastrointestinal complaints, headaches and dizziness. Discontinuation rates do not differ significantly from placebo.
- **Neuroprotection and oxidative stress (preclinical)** Preclinical studies indicate that **EGb 761** and its components (e.g., ginkgolide A) inhibit Aβ oligomerisation, reduce oxidative stress and exert neuroprotective effects in models such as *Caenorhabditis elegans*. These mechanisms may explain clinical effects in dementia but are not directly demonstrated in humans.
- **Inflammatory processes (preclinical)** Review articles describe anti-inflammatory effects of **EGb 761** through inhibition of the NF-κB signalling pathway, reduction of proinflammatory cytokines and modulation of microglia. These mechanisms may contribute to neuroprotective effects but are not clinically confirmed.

LIMITATIONS:

- **Cognition in mild cognitive impairment (MCI) and healthy older adults** Studies show inconsistent results: while some suggest improvements in combination with other therapies, robust long-term data are lacking. In cognitively healthy individuals, **EGb 761** shows no short-term benefits.
- **Standardisation and extract variability** Ginkgo extracts are not uniformly standardised. While **EGb 761** dominates in controlled studies, other preparations vary in composition and dosage, limiting comparability.
- **Bleeding complications and interactions** Case reports indicate a potential bleeding risk, particularly with concurrent anticoagulant use. However, the causal association is not conclusively established due to methodological limitations.

CLASSIFICATION:

Ginkgo biloba, particularly the standardised extract **EGb 761**, shows moderate efficacy in controlled studies for mild to moderate dementia, with improvements in cognition and activities of daily living, though evidence is inconsistent for MCI and healthy older adults.

1. What Ginkgo Biloba Is

Ginkgo biloba is an extract derived from the leaves of the **ginkgo tree** (*Ginkgo biloba* L.), one of the oldest living tree species. Traditionally used in Chinese medicine, the extract is now produced in a standardised form, with the **specialised extract EGb 761** dominating research. This contains defined proportions of **flavonoids**, **terpenelactones** (ginkgolides, bilobalide) and **proanthocyanidins**, which are considered the active constituents. It is typically administered orally as tablets, capsules or drops.

For understanding the study situation, it is crucial to note that **ginkgo extracts are not uniformly standardised**. While **EGb 761** has been used in many controlled studies, other preparations vary in composition and dosage. The **bioavailability** of the constituents is limited, and interactions with cytochrome P450 enzymes may affect the efficacy of other medications. Furthermore, basic research often investigates isolated ginkgolides or flavonoids, the results of which cannot be directly extrapolated to complex extracts.

2. What the Studies Show in Detail

The foundational human studies and key reviews—by application area, including population, duration, preparation, outcome measures and results, as reported in the original publications. Numbers are included only where the study specifies them.

Distribution of controlled human studies by application area—the numbers are not additive, as a single study may be assigned to multiple areas:

APPLICATION AREA	CONTROLLED STUDIES
Safety and tolerability	142
Cognition and mental health	131
Metabolism	28
Fatigue and stress	25
Oxidative stress	23
Inflammatory processes	22

APPLICATION AREA	CONTROLLED STUDIES
Cardiovascular system	19
Musculoskeletal system	8

Cognition and Mental Health in Dementia (Alzheimer's Type and Vascular)

In a multicentre, randomised, double-blind, placebo-controlled study (24 weeks) involving 216 outpatients (age not explicitly stated, diagnosis: mild to moderate primary degenerative dementia of the Alzheimer's type or multi-infarct dementia according to DSM-III-R), the efficacy of the **Ginkgo biloba** specialised extract **Egb 761** was investigated. Patients received 240 mg **Egb 761** daily or placebo. Three primary endpoints were defined: Clinical Global Impressions (CGI Item 2) for psychopathological assessment, the Syndrom-Kurztest (SKT) for attention and memory, and the Nuremberg Age Inventory (NAB) for activities of daily living. A responder analysis (improvement in at least two of the three variables) showed significantly more therapeutic responders in the **Egb 761** group ($p < 0.005$, Fisher's Exact Test). The evaluation included 156 protocol-compliant patients; an intention-to-treat analysis with 205 patients confirmed the results ².

A further randomised, placebo-controlled, double-blind study (24 weeks) compared **Egb 761** (160 mg/day) with donepezil (5 mg/day) and placebo in patients aged 50–80 years with mild to moderate Alzheimer's dementia. Efficacy was measured using the Syndrom-Kurztest (SKT) and Mini-Mental State Examination (MMSE) for cognition, and the Clinical Global Impression (CGI) for overall clinical assessment. The results demonstrated comparable efficacy of **Egb 761** and donepezil in slowing cognitive decline, with both groups performing significantly better than placebo. The study confirmed the clinical efficacy of **Egb 761** in Alzheimer's dementia, though the abstract did not provide detailed effect sizes ⁴.

The standardised mean difference (SMD) for cognitive changes (measured using SKT or ADAS-Cog) was -0.58 (95% CI: -1.14 ; -0.01 , $p = 0.04$) in favour of **Egb 761** compared with placebo. For activities of daily living (ADL), no statistically significant difference was observed (SMD = -0.32 , 95% CI: -0.66 ; 0.03 , $p = 0.08$). Heterogeneity between the studies was high, limiting comparability ⁵.

A randomised, placebo-controlled, double-blind study (24 weeks) involving 410 patients (aged ≥ 50 years, with mild to moderate dementia and neuropsychiatric symptoms) investigated the efficacy of 240 mg **Egb 761** daily. Primary endpoints were changes in SKT (cognition) and the Neuropsychiatric Inventory (NPI, behavioural symptoms). The **Egb 761** group improved by 2.2 ± 3.5 points in the SKT, while the placebo group improved by only 0.3 ± 3.7 points ($p < 0.001$). In the NPI, an improvement of 4.6 ± 7.1 points was observed with **Egb 761** compared with 2.1 ± 6.5 points with placebo ($p < 0.001$). Secondary endpoints, such as ADCS-CGIC (clinical global impression) and ADL-IS (activities of daily living), confirmed the superiority of **Egb 761**. The study underscores the efficacy in dementia with neuropsychiatric symptoms, though without differentiation by dementia subtype ⁷.

The effects were dose-dependent and particularly pronounced in patients with neuropsychiatric symptoms. The results confirm the efficacy of **Egb 761** in mild to moderate dementia, though with limited generalisability due to the heterogeneity of the study populations ⁶.

HYPOTHESIS Several RCTs showed that **Egb 761** may produce a moderate but clinically relevant improvement in cognition and daily functioning in mild to moderate dementia (Alzheimer's type and vascular). The effects appear comparable to those of cholinesterase inhibitors such as donepezil, though with a more favourable side-effect profile. However, the evidence base is limited by high heterogeneity and a lack of long-term data. In particular, there is a lack of direct comparative studies with other anti-dementia drugs over longer periods, as well as investigations into efficacy in severe dementia.

Cognition and Mental Health in Mild Cognitive Impairment (MCI) and Healthy Older Adults

Participants were randomly assigned to the **Egb 761** or placebo group. Endpoints included changes in standardised tests such as the Selective Reminding Test (SRT), Wechsler Adult Intelligence Scale (WAIS-R), Trail Making Test (TMT), and Geriatric Depression Scale (GDS). The results showed no significant differences between **Egb 761** and placebo in cognitive performance or mood. The study suggests that **Egb 761** offers no short-term cognitive benefits in cognitively healthy older adults ¹³.

The meta-analysis showed that the combination of Ginkgo biloba with conventional therapy (e.g., cholinesterase inhibitors) resulted in significant improvements in the MMSE after 24 weeks compared with conventional therapy alone (mean difference: 1.90 , 95% CI: 1.41 ; 2.39 , $p < 0.00001$). A significant improvement was also observed for ADL (mean difference: -3.72 , 95% CI: -5.68 ; -1.76 , $p = 0.0002$). However, the quality of the studies was rated as moderate to poor, and the results do not allow conclusions about the efficacy of Ginkgo biloba alone when compared with placebo or conventional therapy ¹⁸.

HYPOTHESIS The evidence regarding the efficacy of **Egb 761** in mild cognitive impairment (MCI) is less consistent than in dementia. While some studies indicate improvements in cognition and daily functioning in MCI patients, particularly in combination with other therapies, robust long-term data are lacking. In cognitively healthy older adults, **Egb 761** appears to offer no short-term cognitive benefits. There is a lack of targeted studies on dementia prevention in MCI patients, as well as investigations into possible dose-dependent effects.

Neuroprotection and Oxidative Stress

In a preclinical study using transgenic *Caenorhabditis elegans* models, the effects of **Egb 761** and its individual components (e.g., ginkgolide A) on amyloid-beta ($A\beta$)-induced pathological behaviours were examined. **Egb 761** (dose not explicitly stated) and ginkgolide A reduced $A\beta$ -induced paralysis, improved chemotaxis behaviour, and decreased serotonin hypersensitivity. Additionally, **Egb 761** inhibited $A\beta$ oligomerisation and the deposition of $A\beta$ plaques. Interestingly, the study found that the antioxidant effect of **Egb 761** was not the primary mechanism underlying its neuroprotective effects, as ascorbic acid (vitamin C) reduced oxidative stress but had no comparable effect on $A\beta$ -induced paralysis. These findings are preclinical and do not investigate effects in humans ³⁷.

A review (2000) summarised preclinical studies on the neuroprotective mechanisms of **Egb 761**. **Egb 761** demonstrated antioxidant and free radical-scavenging properties, reversed age-related losses of neurotransmitter receptors (e.g., α_1 -adrenergic, 5-HT_{1A}, and muscarinic receptors) in preclinical models, and protected against ischaemic neuronal cell death. Furthermore, **Egb 761** improved the function of the hippocampal mossy fibre system and

increased the density of hippocampal high-affinity choline uptake sites in animal studies. These mechanisms may explain the observed cognitive improvements in dementia patients, though direct clinical evidence linking these preclinical effects to clinical efficacy is lacking ²⁹.

HYPOTHESIS Preclinical data suggest that EGb 761 and its components exert neuroprotective effects through multiple mechanisms, including inhibition of A β oligomerisation, antioxidant effects, and modulation of neurotransmitter systems. These mechanisms may underlie the observed clinical effects in dementia. However, clinical studies directly confirming these preclinical findings or quantifying the contribution of individual mechanisms to clinical efficacy are lacking.

Inflammatory Processes

A review (2000) described the anti-inflammatory and anti-oedematous properties of EGb 761 in preclinical models. EGb 761 reduced the production of pro-inflammatory cytokines and microglial activation, leading to a decrease in neuroinflammatory processes. Additionally, EGb 761 exhibited protective effects against free radicals and oxidative stress associated with inflammatory processes. These mechanisms may contribute to the efficacy of EGb 761 in neurodegenerative diseases such as Alzheimer's dementia, though clinical studies directly demonstrating these effects in patients are lacking ³⁸.

A recent review (2022) summarised the multimodal mechanisms of action of *Ginkgo biloba*, including anti-inflammatory effects. The flavonoids and terpenoids (e.g., ginkgolides) it contains inhibit the activation of the NF- κ B signalling pathway, reduce the expression of pro-inflammatory genes, and modulate immune cell activity. These effects may contribute to the neuroprotective action of EGb 761, though it remains unclear to what extent these mechanisms contribute to clinical efficacy in dementia or other neurological diseases ⁴⁰.

HYPOTHESIS The anti-inflammatory properties of EGb 761 may play a role in slowing neurodegenerative processes, particularly in dementia types with an inflammatory component (e.g., vascular dementia). However, clinical studies specifically investigating the anti-inflammatory effects of EGb 761 in dementia patients or quantifying the contribution of these mechanisms to clinical efficacy are lacking.

Safety and Side Effects

A systematic review (2005) analysed 15 case reports of bleeding complications associated with *Ginkgo biloba* intake. Most cases involved severe medical events, including eight intracranial haemorrhages. However, 13 of the 15 cases had additional risk factors for bleeding (e.g., concurrent use of anticoagulants). In only six cases was it reported that the bleeding did not recur after discontinuation of *Ginkgo biloba*. In three cases, bleeding time was increased during *Ginkgo biloba* intake. The authors concluded that a possible causal association between *Ginkgo biloba* and bleeding exists, though it cannot be definitively established due to the methodological limitations of the case reports ³⁹.

The most common side effects were gastrointestinal complaints, headaches, and dizziness, though these were generally mild and transient. Discontinuation rates due to side effects did not differ significantly between EGb 761 and placebo. The meta-analysis confirmed the favourable safety profile of EGb 761 in mild to moderate dementia ⁶.

HYPOTHESIS The available data suggest that EGb 761 has a favourable safety profile, particularly in comparison with other anti-dementia drugs. However, there is a potential risk of bleeding complications, especially with concurrent use of anticoagulants or antiplatelet agents. Systematic long-term studies on the safety of EGb 761 and targeted investigations into possible drug interactions are lacking.

Overall Evidence

The evidence on *Ginkgo biloba*, particularly the standardised extract EGb 761, shows consistent indications of moderate efficacy in mild to moderate dementia (Alzheimer's type and vascular). Several randomised controlled trials and meta-analyses demonstrate improvements in cognition, daily functioning, and neuropsychiatric symptoms, with effects appearing to be dose-dependent (typically 240 mg/day). The efficacy of EGb 761 is comparable to that of cholinesterase inhibitors such as donepezil, though with a more favourable side-effect profile. In mild cognitive impairment (MCI) and cognitively healthy older adults, the results are less conclusive, and robust long-term data on preventive effects are lacking.

Preclinical studies suggest multiple neuroprotective mechanisms of EGb 761, including antioxidant, anti-inflammatory, and anti-amyloidogenic effects. However, clinical studies directly linking these mechanisms to clinical efficacy are lacking. The safety of EGb 761 is well documented overall, though there is a potential risk of bleeding complications, particularly with concurrent use of anticoagulants. Systematic investigations into long-term effects, drug interactions, and targeted studies on efficacy in severe dementia or specific dementia subtypes are lacking.

Methodology of the evidence base

The key studies on *Ginkgo biloba* are predominantly randomised, placebo-controlled, double-blind trials lasting 12 to 52 weeks, with most studies spanning 24 weeks. Typical endpoints include cognitive tests (e.g., SKT, MMSE, ADAS-Cog), activities of daily living (ADL scales), neuropsychiatric symptoms (NPI), and global clinical assessments (CGI, ADCS-CGIC). Study populations generally include patients with mild to moderate dementia (Alzheimer's type or vascular) or mild cognitive impairment (MCI), while severe dementia or specific subgroups (e.g., dementia with Lewy bodies) are rarely investigated. Systematic weaknesses in the evidence base include high heterogeneity of populations, lack of long-term data (> 1 year), insufficient differentiation by dementia subtype, and the absence of direct comparative studies with other anti-dementia drugs over longer periods.

Dementia studies: Why results vary so widely

The
MOST CITED CONTROLLED STUDIES

on **Ginkgo biloba** in dementia date from the 1990s and 2000s and show **partially positive effects** on cognitive performance. However, the results are **inconsistent**: while some studies report improvements in standardised tests, others find no significant differences compared to placebo. A key issue is the **heterogeneity of study populations**. Many studies include patients with **mild to moderate dementia**, but diagnostic criteria vary. Additionally, different **cognitive scales** are used, making comparisons difficult.

Another critical point is the

SELECTION OF EXTRACTS

. Most positive results come from studies using **EGb 761**, a standardised extract not always identically present in other preparations. More recent studies with different extracts or dosages show **clear effects less frequently**, which may indicate an **extract-specific effect**. Furthermore, long-term data on **clinically relevant endpoints** such as care dependency or quality of life are lacking. The **published evidence** may be skewed by **publication bias**, as negative results are less frequently published.

3. How Robust the Research Is

Research on **Ginkgo biloba** includes a substantial number of human studies, with a notable portion being controlled trials. The focus lies on **safety** and **cognitive functions**, followed by **metabolism**, **fatigue**, and **antioxidant effects**. Most controlled studies examine **dementia** or **mild cognitive impairment**, with older studies being cited more frequently than newer ones. Basic research and observational studies complement the evidence but do not provide causal proof of clinical effects.

The methodological quality of the studies is **inconsistent**. While some randomised, placebo-controlled trials using EGb 761 have high citation rates, many older studies lack detailed information on **blinding**, **drop-out rates**, or **long-term outcomes**. Meta-analyses yield contradictory results due to the inclusion of different extracts, dosages, and patient populations. Moreover, **surrogate parameters** such as cognitive test scores are not always equivalent to clinically relevant improvements. **Safety data** are robust, but interactions with anticoagulants are documented.

4. Common Claims — and What the Data Show

STATEMENT	EVIDENCE
Ginkgo biloba in healthy older adults	Controlled studies show no consistent effects on cognitive performance in cognitively healthy individuals. Some research reports slight improvements in certain
Prevention of dementia	Observational studies suggest an association between ginkgo intake and a reduced risk of dementia, but controlled studies are lacking. A causal effect is not es
Effects on depression or anxiety disorders	The data situation is still emerging . Some smaller studies examine ginkgo as an adjunctive therapy, but the results are not sufficiently robust to allow for an asse
Antioxidant effects in everyday life	Laboratory studies demonstrate antioxidant properties, yet it remains unclear whether these are clinically relevant . Controlled human studies provide no conclus

5. Profile: Molecule and Charge

Ginkgo biloba is not a single molecule but a **mixture** — there is no molecular formula or fixed molecular mass. What can be described are functional groups, charge behaviour, and redox properties; these are the focus of the next section.

6. Charge, Electrons, and Radicals

Ginkgo biloba extracts exhibit electrochemical properties linked to radicals and redox processes. The antioxidant activity is attributed to flavonoid and ginkgolide components, which reduce ROS levels in tissues ²⁹. In controlled human studies, a decrease in ROS as a biomarker was observed ²⁷. Additionally, the prevention of membrane damage by free radicals is described, suggesting stabilisation of cellular structures ²⁶. The antioxidant effect is documented independently of direct cation exchange or specific electron transfer.

Reductions in oxidative stress due to Ginkgo biloba extracts have been measured in humans in several randomised, placebo-controlled studies. The involvement of free radicals in the pathogenesis of tardive dyskinesia (TD) was assumed^{24 25}, and the extracts showed significant radical-scavenging effects^{25 24}. Serum markers such as glutathione (GSH) and ROS were used as indicators of redox status, with improvements noted under treatment²⁷. Antioxidant capacity is discussed as a central mechanism of the observed effects²⁸. Whether this effect is due to direct electrochemical charge transfer or cation exchange was not specifically investigated in the studies. Further measurements of electron transfer processes or charge changes in humans are not available.

7. Interactions with Other Substances

Substances with which this compound has been **jointly** studied or discussed in human trials and reviews — whether in combination, comparison, or via shared mechanisms. The number indicates studies in which both appear.

SUBSTANCE	SHARED STUDIES	OF WHICH CONTROLLED IN HUMANS
Chronic inflammation	71	21
Stress, cortisol, and inflammation	70	18
Adaptogens	41	11
Turmeric and curcumin	20	2
Quercetin	13	0
Resveratrol	10	2
Anthocyanins (berries)	8	0
Therapeutic fasting and intermittent fasting	8	4
Acupuncture	6	5
Vitamin B12	6	2

Data from controlled human studies are available on the interactions of *Ginkgo biloba* with medicinal products. Administration showed no clinically relevant effects on the activity of the CYP enzymes CYP2C19 and CYP2D6 (90% confidence intervals: 0.681–1.122 and 0.667–1.281, respectively)³⁰. It was concluded that *Ginkgo biloba* has no relevant potential for metabolic drug interactions via these enzymes³⁰. Combination treatments with acetylsalicylic acid (325 mg/day) over 4 weeks revealed no statistically or clinically detectable changes in coagulation parameters^{32 35}. Even in elderly patients with peripheral arterial occlusive disease or cardiovascular risk factors, no increased safety risks were observed with this combination³⁵.

In the acute treatment of ischaemic stroke, *Ginkgo biloba* was investigated in combination with rt-PA (recombinant tissue plasminogen activator). No significant increase in haemorrhagic transformations was observed (odds ratio 0.885; 95% CI 0.450–1.741)³⁶. The combination with aspirin following acute ischaemic stroke also did not lead to an increased rate of vascular events³¹. No unexpected interactions were reported with concurrent administration of donepezil or steroids, though no superior efficacy of the combination therapy was demonstrated either^{33 34}.

8. Overdose, Tolerability, and Limitations

No specific dosage data on overdose, toxicity, or upper limits of *Ginkgo biloba* extracts are available from the existing studies. In controlled human trials, a daily dose of 240 mg was deemed safe. The discontinuation rate due to adverse effects was significantly lower in *Ginkgo* groups than in placebo groups (odds ratio 1.72)¹⁶. Contraindications were not explicitly stated in the studies.

Tolerability was described as good, with similar rates of adverse effects in verum and placebo groups^{12 15}. A meta-analysis confirmed the safety of this dosage, with no evidence of dose-dependent toxicity^{16 16}. Laboratory, animal, or tissue studies on overdose or acute toxicity are not included in the available evidence. Further investigations into higher doses or long-term effects would be necessary to define upper limits or specific risks.

9. The Most Robust Studies

Ranked by evidential strength, not by prominence. The full evaluation includes significantly more studies; only the most reliable are listed here.

Controlled Human Studies

- ¹ The risk-benefit profile of commonly used herbal therapies: Ginkgo, St. John's Wort, Ginseng, Echinacea, Saw Palmetto, and Kava (2002)
- ² Proof of efficacy of the ginkgo biloba special extract EGb 761 in outpatients suffering from mild to moderate primary degenerative dementia of the Alzheimer type (1996)
- ³ Ginkgo biloba for cerebral insufficiency (1992)

- ⁴ Ginkgo biloba and donepezil: a comparison in the treatment of Alzheimer's dementia in a randomised placebo-controlled double-blind study (2006)
- ⁵ Effects of Ginkgo biloba in dementia: systematic review and meta-analysis (2010)
- ⁶ Efficacy and adverse effects of ginkgo biloba for cognitive impairment and dementia: a systematic review and meta-analysis (2015)
- ⁷ Ginkgo biloba extract EGb 761® in dementia with neuropsychiatric features: a randomised, placebo-controlled trial to confirm the efficacy and safety of (2012)
- ⁸ Efficacy and tolerability of Ginkgo biloba extract EGb 761® in dementia: a systematic review and meta-analysis of randomised placebo-controlled trials (2014)
- ⁹ The efficacy of ginkgo for elderly people with dementia and age-associated memory impairment: new results of a randomised clinical trial (2000)
- ¹⁰ A 26-week analysis of a double-blind, placebo-controlled trial of the ginkgo biloba extract EGb 761 in dementia (2000)
- ¹¹ Treatment of dementia and mild cognitive impairment with or without cerebrovascular disease: Expert consensus on the use of Ginkgo biloba extract, EGb 761 (2019)
- ¹² Efficacy and safety of a once-daily formulation of Ginkgo biloba extract EGb 761 in dementia with neuropsychiatric features: a randomised controlled trial (2011)
- ¹³ A double-blind, placebo-controlled, randomised trial of Ginkgo biloba extract EGb 761 in a sample of cognitively intact older adults: neuropsychological effects (2002)
- ¹⁴ Ginkgo biloba special extract in dementia with neuropsychiatric features: a randomised, placebo-controlled, double-blind clinical trial (2007)
- ¹⁵ Efficacy and tolerability of a once-daily formulation of Ginkgo biloba extract EGb 761® in Alzheimer's disease and vascular dementia: results from a randomised controlled trial (2012)
- ¹⁶ Meta-analysis of the efficacy and safety of Ginkgo biloba extract for the treatment of dementia (2015)
- ¹⁷ An updated review of randomised clinical trials testing the improvement of cognitive function of Ginkgo biloba extract in healthy individuals and those with cognitive impairment (2019)
- ¹⁸ Ginkgo biloba for mild cognitive impairment and Alzheimer's disease: a systematic review and meta-analysis of randomised controlled trials (2016)
- ¹⁹ Efficacy and safety of Ginkgo biloba extract EGb 761 in mild cognitive impairment with neuropsychiatric symptoms: a randomised, placebo-controlled, double-blind trial (2014)
- ²⁰ Ginkgo biloba extract EGb 761®, donepezil, or both combined in the treatment of Alzheimer's disease with neuropsychiatric features: a randomised, double-blind trial (2009)
- ²¹ Ginkgo biloba for cognitive impairment and dementia (2002)
- ²² Ginkgo biloba special extract EGb 761 in generalised anxiety disorder and adjustment disorder with anxious mood: a randomised, double-blind, placebo-controlled trial (2007)
- ²³ Treatment effects of Ginkgo biloba extract EGb 761® on the spectrum of behavioural and psychological symptoms of dementia: meta-analysis of randomised controlled trials (2018)
- ²⁴ Extract of Ginkgo biloba treatment for tardive dyskinesia in schizophrenia: a randomised, double-blind, placebo-controlled trial (2011)

10. Summary

The body of evidence on **Ginkgo biloba**, particularly the standardised extract **EGb 761**, consistently indicates moderate efficacy in mild to moderate dementia (Alzheimer's type and vascular). Randomised controlled trials show improvements in cognitive test scores (e.g., SKT, MMSE), activities of daily living (ADL), and neuropsychiatric symptoms (NPI) at a typical daily dose of 240 mg. The safety of EGb 761 is well documented, with mild gastrointestinal complaints and headaches being the most common adverse effects. Preclinical data suggest multiple neuroprotective mechanisms, including antioxidant, anti-inflammatory, and anti-amyloidogenic effects, though these have not been directly confirmed in clinical studies.

HYPOTHESIS The consistent findings from multiple RCTs and meta-analyses suggest that **EGb 761** may produce a clinically relevant, albeit moderate, improvement in cognition and daily functioning in mild to moderate dementia. Given its favourable side-effect profile, **EGb 761** could serve as an alternative or adjunct to cholinesterase inhibitors, especially in patients with intolerances or contraindications. **Hypothesis:** The neuroprotective mechanisms demonstrated in preclinical studies (e.g., inhibition of Aβ oligomerisation, anti-inflammatory effects) may underlie the clinical effects. However, these mechanisms remain unconfirmed in clinical studies, and their contribution to efficacy is not quantified. **Hypothesis:** The evidence is less conclusive for mild cognitive impairment (MCI) and cognitively intact older adults. While some studies suggest improvements when combined with other therapies, robust long-term data on preventive effects are absent. It remains unclear whether **EGb 761** can slow the progression from MCI to dementia.

Future studies should specifically investigate the long-term efficacy and safety of **EGb 761** in dementia, particularly in comparison with other anti-dementia drugs and over periods exceeding 24 weeks. Direct comparative trials with cholinesterase inhibitors or NMDA receptor antagonists could clarify the positioning of **EGb 761** in dementia therapy. Additionally, targeted research is needed on its efficacy in severe dementia and its preventive potential in MCI. Preclinical findings on neuroprotective mechanisms should be validated through clinical studies to quantify the contribution of individual mechanisms (e.g., anti-inflammatory effects, antioxidant activity) to clinical efficacy. Systematic long-term studies on safety, particularly regarding bleeding complications and interactions with anticoagulants, are necessary to further establish the risk profile of **EGb 761**.

11. Unresolved Questions

- **Long-term effects:** Are there clinically relevant benefits or risks after years of use that are not captured in short-term studies?

- **Extract specificity:** Are the effects of **EGB 761** transferable to other Ginkgo preparations, or does it possess a **unique efficacy profile**?
- **Mechanism:** Which **molecular targets** are responsible for the observed effects, and how do they interact with neurodegenerative processes?

12. Further Paths

Topics, pathways, and networks in which this substance appears—for those who wish to explore beyond the substance itself.

- Interaction network

Topics, mechanisms and chains in which this substance appears — for those who want to look beyond the substance.

Frequently Asked Questions

Can Ginkgo biloba improve cognitive performance in healthy individuals?

The evidence is **inconsistent**. Some controlled studies show **slightly positive effects** in specific cognitive tests, while others find **no difference** from placebo. The effects are **not consistent** and are often not clinically relevant. Meta-analyses conclude that **no clear benefit** for cognitively healthy individuals has been established.

How safe is the use of Ginkgo biloba, particularly when taken alongside other medications?

Ginkgo biloba is considered **well tolerated** within the studied dosage ranges, but **documented interactions** exist. Of particular relevance is the **inhibition of cytochrome P450 enzymes**, which can affect the metabolism of certain medications (e.g., anticoagulants). Additionally, Ginkgo may **increase bleeding tendency**, which should be considered when taken concurrently with blood thinners.

Why do dementia studies on Ginkgo biloba yield such varied results?

The heterogeneity of the results can be attributed to several factors: **different extracts** (e.g., EGB 761 vs. other preparations), **varying dosages**, **inconsistent diagnostic criteria**, and **diverse cognitive testing methods**. Furthermore, many studies are methodologically weak, limiting their validity. Recent meta-analyses indicate that the effects are **smaller** than those reported in older studies.

Sources

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