



DOSSIER · SUBSTANCE

Carnosine

Carnosine is a dipeptide of β -alanine and L-histidine. The literature is unusually distributed: The most cited work in the field is a mechanism review, and the most cited experimental works overall do not address carnosine but β -alanine and its effect on muscle carnosine content. In humans, observation and marker endpoints predominate over controlled longitudinal studies.

889

STUDIES IN
TOTAL

212

IN HUMANS

74

OF WHICH
CONTROLLED

151

IN ANIMALS

104

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

Carnosine at a Glance

ORIGIN:

Carnosine is a dipeptide composed of β -alanine and L-histidine; the enzyme carnosine synthase (ATPGD1) is responsible for its synthesis. It is present in measurable concentrations in skeletal muscle, alongside anserine, its methylated form. Three properties are discussed: the binding of reactive carbonyl compounds, an antioxidant activity, and the protection of proteins from glycation and cross-linking.

EVIDENCE BASE:

889 evaluated papers · 74 controlled human studies · 138 observational studies · 49 review articles · 151 animal studies · 85 cell-culture studies

STRONGEST EVIDENCE:

- **Metabolism:** A double-blind randomised pilot study in 15 overweight, non-diabetic individuals showed an attenuated rise in fasting insulin and insulin resistance under 2 g carnosine daily for 12 weeks ($P = 0.02$ and 0.04 , respectively).
- **Oxidative stress:** 90 children with diabetic nephropathy received 1 g carnosine daily for 12 weeks; HbA1c, urinary albumin excretion, $\alpha 1$ -microglobulin and malondialdehyde decreased, while total antioxidant capacity rose ($P < .001$).
- **Oncology:** 65 patients received FOLFOX-6 with or without 500 mg L-carnosine daily; in the carnosine group 40 % remained free of neuropathy versus 3.2 % without carnosine, NF- κ B fell by 27 % and TNF- α by 36.6 %.
- **Cognition:** 39 healthy older volunteers received 1.0 g of an anserine/carnosine formula (3:1) for 3 months; delayed verbal recall was preserved ($p = 0.0128$) and inflammatory cytokines declined. The single substance remains open.
- **Pharmacokinetics:** After a single dose of 60 mg carnosine per kilogram body weight in 25 healthy persons, only 8 showed a measurable rise in plasma carnosine; their urinary excretion was 2.6-fold higher.

LIMITATIONS:

- Observational designs predominate in humans; many controlled studies measure biochemical markers such as fasting glucose, triglycerides or advanced glycation end-products. A marker is not a disease course.
- Oral carnosine intake and measured tissue carnosine are not the same: carnosinase cleaves supplied carnosine in the blood, and the amount that reaches tissue unchanged depends on this breakdown.
- Several controlled studies test mixtures, for example anserine/carnosine in a 3:1 ratio or cinnamon, chromium and carnosine; no effect of the single substance carnosine can be inferred from a mixture.

CLASSIFICATION:

Carnosine is a muscle-derived dipeptide of β -alanine and L-histidine that has been investigated in humans predominantly via marker endpoints; central questions of supply and the single substance remain open.

1. What Carnosine Is

Carnosine is a **dipeptide** of β -alanine and L-histidine. The enzyme **carnosine synthase** (ATPGD1) is responsible for its synthesis from these two building blocks. It is present in measurable concentrations in skeletal muscle; alongside it exists **anserine**, the methylated form that has been examined together with carnosine in several studies. The literature mainly discusses three properties: the binding of reactive carbonyl compounds, an antioxidant activity, and the protection of proteins from glycation and cross-linking. An early basic-science contribution explicitly describes carnosine as a possible inhibitor of non-enzymatic glycation and protein cross-linking. These three properties run through the entire literature, from cell culture to controlled studies, and explain why carnosine is investigated equally in metabolism, cognition, muscle and the cardiovascular system.

One point is decisive for the evaluation of all studies: **supplied and measured carnosine are not the same**. Orally ingested carnosine is cleaved in the blood by **carnosinase**; how much of it reaches tissue unchanged depends on this breakdown. A second route has been investigated far more intensively in research: the supply of **β -alanine**, the rate-limiting building block, raises the carnosine concentration in muscle. It is measured predominantly in muscle tissue, in the present studies in the vastus lateralis. The variety of forms is considerable: L-carnosine, orodispersible levo-carnosine, anserine-carnosine mixtures and combination preparations with zinc, chromium or plant extracts. Anyone reading a study must therefore first clarify which substance was supplied, which marker was measured and whether a single substance or a mixture was tested.

2. What the Studies Show in Detail

The key human studies and the most important reviews — by therapeutic area, with population, duration, preparation, measured variable and result as reported in the original papers. Numbers appear only where the work states them.

Distribution of **controlled human studies** by therapeutic area — the numbers are not additive, as one study may be assigned to several areas:

THERAPEUTIC AREA	CONTROLLED STUDIES
Metabolism	33
Cognition and psyche	25
Safety and tolerability	24

THERAPEUTIC AREA	CONTROLLED STUDIES
Fatigue and stress	22
Oxidative stress	21
Inflammatory processes	18
Heart and circulation	11
Musculoskeletal system	10

Metabolism

In a double-blind randomised pilot study, 15 non-diabetic persons with overweight or obesity (age 43 ± 8 years; body-mass index 31 ± 4 kg/m²) received 2 g carnosine daily, 15 persons received placebo, each for 12 weeks. Insulin sensitivity and secretion, glucose tolerance in the oral glucose-tolerance test, blood pressure, plasma lipid profile, skeletal muscle by ¹H-MRS and urinary carnosine excretion were measured. Urinary carnosine concentration rose under supplementation ($P < 0.05$). In the persons treated with carnosine the rise in fasting insulin and insulin resistance was attenuated; this remained significant after adjustment for age, sex and change in body weight ($P = 0.02$ and $P = 0.04$). In persons with impaired glucose tolerance, two-hour glucose and insulin were lower under carnosine than under placebo ($P < 0.05$). The authors themselves describe the work as a pilot study with correspondingly small group size ¹⁵.

Left-ventricular ejection fraction was determined echocardiographically; cardiopulmonary exercise testing, the 6-minute walk test and quality of life (visual analogue scale and EQ-5D questionnaire) were recorded at baseline and after 6 months. Under L-carnosine the walking distance in the 6-minute walk test ($P = 0.014$) and quality of life on the visual analogue scale ($P = 0.039$) improved between baseline and follow-up. The authors note that no prior study in heart-failure patients existed ¹⁹.

A study in 25 healthy individuals examined whether low carnosinase activity favours the blood carnosine level after carnosine administration. After a single dose of 60 mg carnosine per kilogram body weight, blood and urine were collected and carnosine as well as β -alanine were determined by HPLC; additionally, the CNDP1 genotype, basal plasma carnosinase activity and protein content were recorded. In 8 of the 25 individuals (responders), a measurable increase in plasma carnosine was detectable up to 1 hour after administration. Individuals without a measurable increase (non-responders) showed an approximately 2-fold higher carnosinase protein content and an approximately 1.5-fold higher activity in plasma. Urinary excretion of carnosine was 2.6-fold higher in responders than in non-responders ⁵³.

In laboratory experiments carnosine reacted with methylglyoxal and thereby inhibited methylglyoxal-mediated protein modification, as shown electrophoretically. When proteins were exposed to an AGE formed from lysine and methylglyoxal, carnosine was also able to inhibit this modification; the authors see this as a second point of attack and discuss the potential as a non-toxic modulator of diabetic complications ⁵¹. These works are preclinical. In a further double-blind, placebo-controlled study, 19 physically active college men (control group $n = 10$, beta-alanine group $n = 9$) performed sprint tests on a non-motorised treadmill against a resistance of 15 % of body weight; supplementation consisted of 4 g in the first week and 6 g beta-alanine daily or a placebo of rice flour in the following 4 weeks. No significant group differences were found for peak and mean horizontal performance or for performance decline (% fatigue) ($p > 0.05$) ⁴⁵.

Inflammatory Processes

In a randomised, double-blind, placebo-controlled study, 54 patients with type 2 diabetes received either two capsules of L-carnosine of 500 mg each ($n = 27$) or placebo ($n = 27$) for 12 weeks. Blood samples and information on food intake were collected at baseline and after 12 weeks. Under L-carnosine, fat mass decreased and fat-free mass increased compared with placebo (1.5 % and 1.7 % respectively) ($P < 0.05$).

39 healthy older volunteers (60–78 years) took part in a double-blind randomised controlled study and received 1.0 g of an anserine/carnosine formula in a 3:1 ratio or placebo daily for 3 months. Delayed verbal recall, measured with the Wechsler Memory Scale-Logical Memory, remained significantly preserved in the verum group compared with placebo ($p = 0.0128$). Blood analysis showed reduced secretion of inflammatory cytokines, including CCL-2 and IL-8. In the MRI analysis using arterial spin labelling, the age-related decline in cerebral blood flow in the posterior cingulate cortex was attenuated in the verum group versus placebo ($p = 0.0248$). In a further randomised controlled study, delayed verbal recall remained preserved in the verum group ($p = 0.0202$); the authors consider further investigations necessary ¹⁶.

In 60 healthy older volunteers (verum group $n = 30$, placebo $n = 30$) who received 1.0 g of an anserine/carnosine formula (3:1) for 3 months, a microarray analysis followed by quantitative real-time PCR on peripheral blood mononuclear cells showed reduced expression of the inflammatory chemokine CCL24 ($p < 0.05$). Verbal recall remained preserved in the verum group. An age-restricted sub-analysis revealed significant preservation of verbal recall in participants in their 60s (verum $n = 12$, placebo $n = 9$; $p = 0.048$) and in their 70s (verum $n = 7$, placebo $n = 11$; $p = 0.017$). Suppression of CCL24 expression was greatest in participants in their 70s ($p < 0.01$), and in this group preservation of verbal recall correlated with CCL24 suppression (Poisson correlation, $r = 0.46$, $p < 0.05$) ²².

In a pilot study, 65 patients were prospectively randomised into two arms: arm A with 31 patients received the FOLFOX-6 regimen, arm B with 34 patients received FOLFOX-6 plus 500 mg L-carnosine orally daily throughout treatment. After three months, neuropathy incidence and grade as well as further toxicities were recorded according to NCI-CTC version 4. In arm B, 17 patients (56.7 %) had grade 1 versus 11 patients (35.5 %) in arm A; one patient (3.3 %) reached grade 2 versus 19 patients (61.3 %) in arm A; 12 patients (40 %) remained normal versus one patient (3.2 %) in arm A. In arm B, addition of L-carnosine lowered the levels or activity of NF- κ B by 27 % and of TNF- α by 36.6 % ⁴².

For the combination of cinnamon, chromium and carnosine there is a randomised, double-blind, placebo-controlled study in 62 prediabetic individuals with a fasting glucose value between 5.55 and 7 mmol/L and a body-mass index ≥ 25 kg/m² who received either 1.2 g/day of the dietary supplement or placebo for 4 months and were followed up to 6 months after randomisation. Fasting glucose decreased compared with placebo (-0.24 ± 0.50 vs $+0.12 \pm 0.59$ mmol/L, p

= 0.02), without detectable significant change in HbA1c. Markers of insulin sensitivity, plasma lipids and inflammatory markers did not differ between groups, nor did body weight or energy or macronutrient intake; fat-free mass increased. No effect of the single substance carnosine can be derived from this mixture ⁴³.

In laboratory experiments carnosine inhibited the non-enzymatic glycosylation and cross-linking of proteins by reactive aldehydes, including aldose and ketose sugars, certain triose glycolysis intermediates and malondialdehyde. It also inhibited the formation of malondialdehyde-induced, protein-associated AGEs as well as the formation of DNA-protein cross-links by acetaldehyde and formaldehyde. At the cellular level, 20 mM carnosine protected cultured human fibroblasts and lymphocytes, CHO cells and cultured rat brain endothelial cells against the toxic effects of formaldehyde, acetaldehyde and malondialdehyde as well as against AGEs; the rat brain endothelial cells were also protected against the toxicity of amyloid peptide. These works are preclinical ³³.

A carnosine analogue (carnosinol) was, according to the authors, developed to be orally bioavailable and resistant to carnosinases and to exhibit the greatest potency and selectivity towards α,β -unsaturated aldehydes among the substances described so far. In rodent models of diet-induced obesity and metabolic syndrome, carnosinol dose-dependently reduced HNE-adduct formation in liver and skeletal muscle and at the same time alleviated inflammation, dyslipidaemia, insulin resistance and steatohepatitis; the improvements were not attributable to altered energy intake. This is a preclinical investigation of an analogue, not of carnosine itself ⁵⁰. A review article describes carnosine as a potent and selective scavenger of α,β -unsaturated aldehydes and reports the inhibition of aldehyde-induced protein-protein and DNA-protein cross-links in neurodegenerative diseases, cardiovascular ischaemic damage and inflammatory diseases ⁵⁵. A later review places the antioxidant and carbonyl-binding effect in direct mechanisms and an indirect activation of the Nrf2 transcription factor; the molecular mechanisms must, in the authors' assessment, still be fully clarified before carnosine and derivatives are promoted as therapeutics ⁵⁸.

Oxidative Stress

A randomised placebo-controlled study included 90 paediatric patients with diabetic nephropathy that persisted despite oral ACE inhibitors. The patients were allocated to 12 weeks of 1 g/day carnosine (n = 45) or a matching placebo (n = 45); HbA1c, urinary albumin excretion, the tubular damage marker α 1-microglobulin, total antioxidant capacity and malondialdehyde were determined. Under carnosine, HbA1c ($8.2 \pm 2.1\%$ vs $7.4 \pm 1.3\%$), urinary albumin excretion (91.7 vs 38.5 mg/g creatinine), α 1-microglobulin (16.5 ± 6.8 mg/L vs 9.3 ± 6.6 mg/L) and malondialdehyde (25.5 ± 8.1 vs 18.2 ± 7.7 nmol/mL) decreased, while total antioxidant capacity increased compared with baseline and placebo ($P < .001$). No adverse reactions due to carnosine administration were reported ¹⁸.

31 healthy participants (age 40–78 years, 10 men and 21 women) were double-blind randomised and received an imidazole-dipeptide formula containing 500 mg twice daily (carnosine/anserine in a 1/3 ratio) (n = 14) or an identical placebo (n = 17) for 3 months; functional MRI and neuropsychological testing were performed at baseline and after 3 months. At baseline there were no differences in neuropsychological values. After 3 months the verum group showed better verbal episodic memory performance as well as reduced connectivity in the default-mode network, in the posterior cingulate cortex and in the right fronto-parietal network; a correlation existed between the extent of the cognitive and imaging changes ²¹.

In a double-blind, randomised clinical study, 43 autistic children (31 boys, 12 girls; 4 to 16 years) were allocated to two groups and received 500 mg carnosine or 500 mg placebo daily for two months. Sleep disturbances were recorded with the Children's Sleep Habits Questionnaire, autism severity with the Gilliam Autism Rating Scale 2. Carnosine administration did not change anthropometric indices ($p > 0.05$) and showed no effect on autism severity ($p > 0.05$), but significantly reduced sleep duration ($p = 0.04$), parasomnias ($p = 0.02$) and the overall sleep disturbance score by 7.59 % ($p = 0.006$) compared with the control group ⁴⁴.

In 84 volunteers aged 65 to 85 years with low zinc status, a 12-week placebo-controlled trial was conducted (placebo n = 42, zinc group n = 42) in which zinc-carnosine was tested. Plasma zinc rose by 5.69 % in the zinc group ($p < 0.05$). Micronucleus frequency decreased by 24.18 % compared with baseline, tail moment by 7.09 % and tail intensity by 8.76 % ($p < 0.05$); telomeric base damage was also significantly reduced in the zinc group. Expression of MT1A and ZIP1 increased ($p < 0.05$). Because zinc-carnosine was tested, the finding cannot be attributed to carnosine alone. In a further randomised double-blind placebo-controlled study, 82 overweight type 2 diabetics received 7 mg α -lipoic acid/kg, 6 mg carnosine/kg and 1 mg thiamine/kg body weight or placebo daily for 8 weeks. Glucose (135.7 ± 19.5 mg/dL vs 126.5 ± 16.8 mg/dL) and HbA1c ($8.3 \pm 0.3\%$ vs $6.03 \pm 0.58\%$) decreased ($P < .05$), yet insulin increased (3.6 ± 0.7 μ U/mL vs 6.8 ± 0.2 μ U/mL, $P < .05$); HOMA-IR and HOMA- β were higher in the treated patients. This too is a combination ⁴⁶.

Formula F contained carnosine, coenzyme Q10, vitamin E, vitamin C, beta-carotene, selenium, L-cysteine and Ginkgo biloba for the protection of proteins, lipids, DNA and proteoglycans, vitamins B6, B9 and B12 for lowering the homocysteine level as well as vitamins B1, B2 and B3 for supporting the pentose-phosphate cycle. As markers of oxidative stress, a d-ROMs test (plasma hydroperoxides), plasma homocysteine and glutathione as well as the proportion of sickle erythrocytes were used. A review elaborates the physiological roles of carnosine on the basis of its biochemical properties, including pH buffering, metal-ion chelation and antioxidant capacity, and reports that promising preclinical and clinical results are available for several diseases such as diabetes and its complications, eye diseases, ageing and neurological disorders ⁵⁴. A further review links zinc, carnosine and neurodegenerative diseases and describes the complex of carnosine and zinc as zinc-supplementation therapy ⁵⁷.

Safety and Tolerability

The patients received, as an adjuvant to fluvoxamine, either minocycline 100 mg twice daily or placebo for 10 weeks; fluvoxamine was given at 100 mg/day in the first 4 weeks and thereafter at 200 mg/day in both groups. The Yale-Brown Obsessive Compulsive Scale was recorded. Analysis with a general linear model with repeated measures showed a significant time-by-treatment effect on the Y-BOCS total score, $F(1.49, 137.93) = 7.1$, $P = 0.003$, on the obsession subscale, $F(1.54, 141.94) = 9.72$, $P = 0.001$, and a nearly significant effect on the compulsion subscale. The abstract mentions L-carnosine as adjuvant in the description of the randomisation, while title, tested substance and target variable refer to minocycline. Usable statements on the tolerability of carnosine itself therefore cannot be derived from this work ³⁷.

Further Investigations

In a pilot study, 20 consecutive patients under long-term (more than 3 months) treatment with low-dose acetylsalicylic acid, in whom an initial capsule endoscopy showed an acetylsalicylic-acid-induced small-bowel lesion, were randomised to a group receiving polaprezinc (150 mg/day, a chelate of zinc and L-carnosine) for 4 weeks and a control group without polaprezinc. All participants underwent a follow-up capsule endoscopy after 4 weeks; number and characteristics of small-bowel mucosal lesions were compared within and between groups. In the polaprezinc group the median number of reddened lesions as well as erosions and ulcers decreased significantly at follow-up endoscopy ($P < 0.05$), whereas no significant difference existed in the control group. The authors conclude that co-treatment with polaprezinc may be effective against small-bowel mucosal damage under long-term acetylsalicylic-acid therapy; the zinc-carnosine chelate was tested, not carnosine alone ²⁵.

Cognition and Psyche

In an already reported double-blind randomised controlled study, daily supplementation with 750 mg anserine and 250 mg carnosine improved cerebral blood flow and memory function in older people. A sub-analysis of the same study examined data from 68 participants aged 65 years and older who received anserine/carnosine supplementation or placebo for 12 months, specifically for carriers of the APOE-e4 allele. Participants were examined at baseline and at the end of the study with several neuropsychological tests including the Wechsler Memory Scale-Logical Memory; additionally, arterial spin-labelling and diffusion-tensor imaging were recorded. Supplementation preserved verbal recall significantly ($F[1,65] = 4.2003$, $p = 0.0445$) and frontal cerebral blood flow (FWE at cluster level, $p < 0.001$). The sub-analysis by APOE4 genotype showed significant preservation of blood flow ($p = 0.002$, ASL analysis) and white-matter microstructure ($p = 0.003$, DTI analysis) in prefrontal areas in APOE4-positive individuals ⁴⁷.

In a placebo-controlled crossover study, 20 healthy male volunteers were randomised to daily oral administration of chicken extract or a placebo drink for 4 weeks; the extract and the drink were provided by the companies named in the abstract. Participants performed 2-back test runs as a fatiguing mental task followed by a rest session and completed cognitive tasks focusing on selective attention immediately before and after each session. After 1 and after 4 weeks of extract intake, reaction times in the cognitive tasks after the rest session were significantly shorter than at baseline, whereas no significant changes were observed under placebo. A chicken extract rich in anserine and carnosine was tested, not carnosine alone ⁴⁸.

Experimental work shows that the nucleation-dependent polymerisation of the β -amyloid peptide, the main constituent of plaques in Alzheimer patients, is significantly accelerated in vitro by cross-linking via advanced glycation end-products. During the polymerisation process both nucleation and aggregate growth were accelerated by AGE-mediated cross-linking. The formation of AGE-cross-linked amyloid-peptide aggregates could be attenuated by the AGE inhibitors tenilsetam, aminoguanidine and carnosine. These findings are preclinical; the authors conclude that AGE inhibitors in general could become a promising drug class for the treatment of Alzheimer's disease ⁴⁹.

Musculoskeletal System

A study in 15 male and 15 female participants, evenly distributed across three groups, examined whether L-histidine supplementation can replenish muscle carnosine stores and whether the combined administration of both amino acids is more effective than β -alanine alone. Participants received either pure β -alanine (6 g/day), L-histidine (3.5 g/day) or both amino acids together. Before the start, after 12 days and after 23 days of supplementation, carnosine content in the soleus and gastrocnemius medialis was determined by ¹H-MRS, and venous blood samples were taken; muscle biopsies from the vastus lateralis were obtained at baseline and on day 23. The metabolites β -alanine, histidine and carnosine in plasma and muscle were measured by high-performance liquid chromatography. Both the β -alanine group and the combination group showed increased carnosine concentrations in all muscles examined, with no difference between these groups; in the histidine group the carnosine levels behaved differently ⁵².

A narrative review summarises the state of knowledge on carnosine and β -alanine metabolism as well as the effects of β -alanine supplementation on athletic performance. Intramuscular acidosis is regarded by the authors as one of the main causes of fatigue during intense exercise, and carnosine plays a significant role in muscle pH regulation. Carnosine is synthesised in skeletal muscle from L-histidine and β -alanine; β -alanine availability is the limiting factor for carnosine synthesis. Supplementation with β -alanine increases muscle carnosine content and thereby total muscle buffering capacity, with the potential to improve physical performance during high-intensity exercise. Studies showed improvements in multiple bouts of high-intensity exercise and in single bouts lasting more than 60 seconds; the onset of neuromuscular fatigue was also delayed. Maximal strength and VO₂max did not improve, whereas certain aspects of endurance performance such as the anaerobic threshold and time to exhaustion did. Paraesthesia may occur with a single dose above 800 mg ⁵⁶.

Oncology

A review article discusses carnosinases, Xaa-His dipeptidases that fulfil different functions in all kingdoms of life. The human isoforms CN1 and CN2 catalyse the hydrolysis of the dipeptides carnosine and homocarnosine under appropriate conditions. Changes in serum carnosinase (CN1) activity have been linked to several pathological conditions, including neurological disorders, chronic diseases and cancer; for this reason the use of carnosinase levels as a biomarker in cerebrospinal fluid is questioned. The hydrolysis of imidazole-related dipeptides in prokaryotes and eukaryotes is also catalysed by aminoacyl-histidine dipeptidases such as PepD, PepV and anserinase. The work describes the structure and function of this enzyme class under physiological and pathological conditions and addresses its main substrates carnosine, homocarnosine and anserine ⁵⁹.

Overall Picture

Across the fields, controlled human studies form one part of the evidence. The most robust are the works with the largest participant numbers and the longest observation periods: the randomised placebo-controlled study in 90 paediatric patients with diabetic nephropathy over 12 weeks ¹⁸, the one-year anserine/carnosine study of prefrontal brain blood flow in 68 participants ⁴⁷ and the 65 oncology patients under three-month follow-up ⁴². In addition there are several smaller randomised studies with consistent directions: improvements in fasting insulin, insulin sensitivity and two-hour values in persons with impaired glucose tolerance ¹⁵, reductions in fasting glucose, HbA1c, triglycerides and AGE markers over 12 weeks ¹⁷, improved walking distance and quality of life in heart failure ¹⁹, findings on verbal episodic memory in older adults ¹⁶ and a reduction in inflammatory chemokine expression in blood cells from older adults ²².

Recurring is the limit that arises from combination with other substances: several studies examined mixtures of cinnamon, chromium and carnosine ⁴³, α-lipoic acid, carnosine and thiamine ⁴⁶, zinc-carnosine or an antioxidant package containing carnosine ²⁰, from which no effect of the individual substance can be derived. Added to this is the rapid cleavage by carnosinases in the blood, which is explained by the observation that only 8 of 25 persons showed a measurable plasma carnosine increase after administration of 60 mg/kg ⁵³. A further open question concerns the transfer of laboratory findings in cells and tissues ³³⁴⁹⁵¹ to humans and of results of an analogue in rodent models ⁵⁰ to carnosine itself. What is missing are independent replications of the smaller findings, dose comparisons and studies that test carnosine as a single substance against a defined clinical endpoint over a longer period.

Overall Picture

Muscle carnosine does not prove an effect of oral carnosine

A large part of the citation power of this field rests on studies in which

B-ALANINE

was administered and the **carnosine content in muscle** was measured. The most strongly cited experimental work of all addresses the uptake of orally administered β-alanine and carnosine synthesis in the vastus lateralis. Such investigations demonstrate the uptake of the building block and an increase in muscle concentration. Muscle carnosine is therefore a **marker of availability**, not a clinical endpoint. Anyone who derives a statement about carnosine from these findings shifts the subject: what was administered was β-alanine, not carnosine. The question whether oral carnosine itself raises muscle content to a relevant extent is therefore not addressed.

A second problem concerns

COMBINATION PREPARATIONS

. Several of the most frequently cited controlled studies do not test carnosine alone: one study combined cinnamon, chromium and carnosine in overweight persons with prediabetes, another added a cholinesterase inhibitor with antioxidants, and a further study examined zinc in older adults with low zinc status. No effect of carnosine can be derived from such a design, even if the substance appears in the title. For the assessment this means: the question of what carnosine alone achieves with oral intake cannot be answered with these studies. It is answerable with a clean design, and that is the next task of the field.

3. How Reliable the Research Is

The literature on carnosine is predominantly **preclinical**. The largest block consists of basic scientific works and mechanism reviews, followed by animal studies and cell culture; in humans, **observational** designs predominate over **controlled** ones. Within the controlled works, **metabolism** clearly dominates, followed by **cognition, tolerability and safety, fatigue** and antioxidant endpoints; thereafter follow inflammation, cardiovascular, musculoskeletal, oncology and immune system. The most strongly cited work in the entire field is a mechanism review from 2013, and the most cited experimental works overall deal with **β-alanine intake and carnosine synthesis in muscle**, not the intake of carnosine itself. This shift is the most important finding about this field.

The methodological basis is divided. On one side stand randomised, placebo-controlled studies, including works on diabetic nephropathy in children and on autism in children; these designs carry a robust statement on the respective endpoint examined. On the other side stand numerous smaller works with **biochemical markers** as the target variable, for example fasting glucose, triglycerides, advanced glycation end-products, inflammatory cytokines and oxidative stress markers. A marker is not a disease course. In addition, some of the controlled studies examine **mixtures** or investigate **anserine/carnosine**, so the contribution of the individual substance remains open. Animal and cell findings, for example on delayed diabetic deterioration in mice or on protection of LDL from oxidation and glycation, are **preclinical** and carry no statement on use in humans.

4. What Is Frequently Claimed — and What the Data Provide

STATEMENT	STUDY SITUATION
Muscle carnosine and athletic performance	Almost consistently, the intake of β-alanine was investigated, the building block that increases muscle content. These studies make statements about β-alanine
Blood glucose, triglycerides and advanced glycation end-products in type 2 diabetes	Several controlled studies report changes in these markers . Markers are not disease courses; a statement about the course of the disease is therefore not mad
Verbal memory performance in old age	The controlled studies examined Anserin-Carnosin mixtures . No contribution of the individual substance Carnosine can be derived from a mixture, even if both

STATEMENT	STUDY SITUATION
Tolerability and safety	Safety is among the more frequent application areas of the controlled studies. Reported are tolerability data of the respective tested preparation, not a general c

5. Profile: Molecule and Charge

Carnosine is not a single molecule but a **mixture** — there is no molecular formula and no fixed molecular mass. What can be described are functional groups, charge behaviour and redox properties; that is exactly what the next section addresses.

6. Charge, Electrons, Radicals

The charge properties of carnosine depend on its imidazole ring: in combination with β -alanine its pKa rises to approximately 6.8 and thus lies in the range of the intracellular pH value of muscle during high-intensity loading ³². As a metal chelator, carnosine binds ions; in diabetic rodents it lowered serum iron and improved glucose metabolism ²⁶. The zinc-carnosine chelate complex is described as anti-inflammatory and mucosa-repairing ²⁷ and has been clinically tested as zinc-L-carnosine in ulcerative colitis ²⁴²⁵. Cation exchange in the narrower sense is not thereby proven; chelation is described.

Radicals and redox: carnosine acts as an antioxidant and radical scavenger ²³²⁸ and reacts with harmful aldehydes ³³. In cells it inhibited ROS formation and JNK activation in GT1-7 cells ²⁹ and reduced oxidative stress via NO/ROS and peroxynitrite in RAW-264.7 macrophages ³⁰³⁰ — preclinical findings. A mixture of L-carnosine and A. vera showed a stronger radical-scavenging effect at 500 $\mu\text{g/mL}$ (63 ± 4 RFU vs 83 ± 5 RFU) ³¹; no effect of the individual substance can be derived from such combinations. In humans, mainly clinical endpoints were measured: clinical response or remission 71 % versus 10 % under placebo ²⁴. Direct electrochemical measurements in humans are still outstanding.

7. Interactions with Other Substances

Substances with which this compound has been **jointly** investigated or discussed in human studies and reviews — whether in combination, comparison or via a shared mechanism. The figure indicates the number of works in which both appear.

SUBSTANCE	JOINT WORKS	OF WHICH CONTROLLED IN HUMANS
Acceptance and Commitment Therapy (ACT)	116	22
Beta-Alanine	86	22
Chronic Inflammation	68	20
Stress, Cortisol and Inflammation	61	17
Zinc	29	11
High-Intensity Interval Training (HIIT)	26	8
Art Therapy	25	11
Cognitive Behavioural Therapy for Insomnia (CBT-I)	24	11
Palmitoylethanolamide (PEA)	18	4
Myofascial Release	17	2

With long-term low-dose acetylsalicylic acid the median number of reddened lesions and erosions or ulcers in the control group did not differ substantially; the authors consider co-administration of polaprezinc effective against small-intestinal mucosal damage ²⁵. A fixed combination of inosine monophosphate, agmatine sulphate and L-carnosine was tested as add-on therapy in 120 patients with subacute spinal-cord injury in a randomised trial ¹³. L-Carnosine with anserine or antioxidants improved MMSE versus placebo (pooled net change 0.62; 95 % CI 0.23 to 1.01) ¹⁰.

For absorption, controlled studies describe N-acetylcarnosine eye drops as a 1 % prodrug of L-carnosine with a mucoadhesive cellulose compound and corneal absorption enhancers in a release system ^{34 35}. In rats with arthritis, methotrexate alone and methotrexate together with carnosine were administered ³⁶. In HUVEC cells under methylglyoxal the combination of L-carnosine and A. vera enhanced viability; the 25:1 ratio (w/w) was most effective (27 ± 0.5 % versus 12 ± 0.3 to 18 ± 0.4 %) ³¹. β -Alanine raises the pKa of the imidazole ring to approximately 6.8 ³². It remains to be clarified whether methotrexate alters the effect of carnosine.

8. Overdose, Tolerability, Limitations

No data on upper limits, toxicity or contraindications for carnosine appear in the works examined. The reported tolerability data derive from controlled human studies. In adults with a depressive episode the frequency of adverse effects did not differ substantially between treatment arms ³⁸. In children with autism spectrum disorder ³⁹ and in children with ADHD ⁴⁰ the frequency of adverse effects likewise did not differ significantly between study arms. In a study in people with diabetic nephropathy oral carnosine doses were described as well tolerated, without serious adverse effects ⁴¹.

For combination products separate safety data exist. In a randomised trial with 120 patients with subacute spinal-cord injury three mild, short-lived adverse events were observed in the verum group, none serious ^{13 13}; the fixed combination of inosine monophosphate, agmatine sulphate and L-carnosine was investigated there, so the finding cannot be transferred to the single substance. A meta-analysis of a zinc-carnosine regimen reports no statistical difference in overall events (OR 1.06, 95 % CI [0.55, 2.06], p = 0.85) ⁴. Dose statements, maximum quantities and contraindications cannot be derived from this; the doses tested over which periods would need to be clarified.

9. The Most Robust Studies

Ordered by strength of evidence, not by familiarity: reviews and studies with many participants first. The complete evaluation comprises substantially more works; the most robust are listed here.

Controlled Human Studies

- ¹ Carnosine/histidine-containing dipeptide supplementation improves depression and quality of life: systematic review and meta-analysis of randomized ... (2025) — Review, 776 participants
- ² Effect of L-Carnosine in children with autism spectrum disorders: a systematic review and meta-analysis of randomised controlled trials (2021) — Review, 215 participants
- ³ Effect of carnosine supplementation on lipid profile, fasting blood glucose, HbA1C and insulin resistance: A systematic review and meta-analysis of ... (2020) — Review, 184 participants
- ⁴ Efficacy and Safety of Polaprezinc-Based Therapy versus the Standard Triple Therapy for *Helicobacter pylori* Eradication: A ... (2022) — Review, 396 participants
- ⁵ Effect of carnosine or beta-alanine supplementation therapy for prediabetes or type 2 diabetes mellitus: a systematic review and meta-analysis of ... (2025) — Review article, 377 participants
- ⁶ Effect of L-Carnosine in Patients with Age-Related Diseases: A Systematic Review and Meta-Analysis (2023) — Review article
- ⁷ Histidine-containing dipeptide supplementation improves delayed recall: a systematic review and meta-analysis (2024) — Review article
- ⁸ The effect of carnosine or β-alanine supplementation on markers of glycaemic control and insulin resistance in human and animal studies: a protocol ... (2020) — Review article
- ⁹ Protective and Healing Effects of Zinc L-Carnosine on the Oral Mucosa: A Systematic Review and Meta-Analysis (2026) — Review article
- ¹⁰ The Effects of Carnosine on Cognitive Function and Mental Health-A Systematic Review and Meta-Analysis (2026) — Review article
- ¹¹ The Antioxidant Effect of Beta-Alanine or Carnosine Supplementation on Exercise-Induced Oxidative Stress: A Systematic Review and Meta-Analysis (2018) — Review article
- Neuroimmunomodulatory compound for sensitive skin care: in vitro and clinical assessment (2008) — 124 participants
- ¹² Carnosine supplementation improves cognitive outcomes in younger participants of the NEAT trial (2025) — 299 participants
- ¹³ Efficacy and Safety of Fixed Dose Combination of Inosine Monophosphate, Agmatine Sulphate, and L-Carnosine (CGXNW) as Add-on Therapy for the ... (2026) — 120 participants
- ¹⁴ Effects of carnosine supplementation on physical endurance: a placebo-controlled randomized clinical trial (2026) — 299 participants
- ¹⁵ Effects of carnosine supplementation on glucose metabolism: Pilot clinical trial (2016) — 15 participants
- ¹⁶ Effect of Anserine/Carnosine Supplementation on Verbal Episodic Memory in Elderly People (2016)
- ¹⁷ L-Carnosine supplementation attenuated fasting glucose, triglycerides, advanced glycation end products, and tumor necrosis factor-α levels in ... (2018) — 54 participants
- ¹⁸ The effect of 12 weeks carnosine supplementation on renal functional integrity and oxidative stress in pediatric patients with diabetic nephropathy ... (2018) — 90 participants
- ¹⁹ Effects of oral administration of orodispersible levo-carnosine on quality of life and exercise performance in patients with chronic heart failure (2015)
- ²⁰ Treatment of Alzheimer's disease with a cholinesterase inhibitor combined with antioxidants (2010) — 52 participants
- Zinc supplementation influences genomic stability biomarkers, antioxidant activity, and zinc transporter genes in an elderly Australian population ... (2015) — 84 participants
- ²¹ Daily Carnosine and Anserine Supplementation Alters Verbal Episodic Memory and Resting State Network Connectivity in Healthy Elderly Adults (2015) — 17 participants
- ²² Anserine/Carnosine Supplementation Suppresses the Expression of the Inflammatory Chemokine CCL24 in Peripheral Blood Mononuclear Cells from Elderly ... (2017)

10. Summary

Two randomised, placebo-controlled studies with the single substance carnosine carry the most robust findings in humans: the pilot study in 15 overweight, non-diabetic individuals, in which 2 g carnosine daily over 12 weeks attenuated the rise in fasting insulin and insulin resistance, and the study in 90 children with diabetic nephropathy, in which 1 g carnosine daily over 12 weeks lowered HbA1c, urinary albumin excretion, α 1-microglobulin and malondialdehyde and raised total antioxidant capacity ($P < .001$). Both studies tested carnosine as a single substance. The studies on methylglyoxal, glycation, the carnosine analogue carnosinol and protection of LDL from oxidation are to be classified as preclinical.

PLAUSIBLE The three consistent properties — binding of reactive carbonyl compounds, antioxidant activity, protection from glycation and cross-linking — are described in cell culture and in the animal model and explain why the same dipeptide is being tested in metabolism, cognition, muscle and the cardiovascular system. **Hypothesis:** If the mechanism of action proceeds via the binding of reactive aldehydes, the measured tissue concentration could be more important than the amount supplied, because carnosinase already cleaves the absorbed carnosine in the blood. β -Alanine supply and carnosine supply are two different research paths; the most highly cited work in the field is a mechanism review from 2013, and the most frequently cited experimental studies address β -alanine and carnosine synthesis in muscle.

Open question: A study that tests the single substance carnosine would first have to record the carnosinase activity of the participants, because in the investigation of 25 healthy individuals only 8 showed a measurable rise in plasma carnosine after a dose of 60 mg carnosine per kilogram body weight. The authors of a review note that the molecular mechanisms still need to be fully clarified before carnosine and derivatives can be promoted as therapeutics. In addition, a muscular or plasma time course over the study duration, a placebo-controlled design with carnosine alone instead of a mixture of anserine and carnosine or of cinnamon, chromium and carnosine, and a clinical endpoint instead of a biochemical marker would be useful.

11. What Remains Open

- **Uptake:** How much orally administered carnosine reaches muscle, brain or kidney unchanged when serum carnosinase already cleaves it in the blood? This question decides every oral study.
- **Single Substance:** Which randomised study tests carnosine alone, without anserine, zinc, chromium or plant extracts, and measures a clinical endpoint instead of a marker?
- **Measurement:** How meaningful is the carnosine content of a muscle biopsy for endpoints that matter to the treated individuals, such as performance, exhaustion or metabolic control?

12. Further Paths

Topics, modes of action and chains in which this substance occurs — for all who wish to look beyond the substance.

- Interaction Network

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

Frequently Asked Questions

Can oral carnosine raise the muscle carnosine level?

In the available studies the muscle content was raised via supply of **β -alanine**, the rate-limiting building block of synthesis; muscle concentration is the measured marker there. For orally supplied carnosine itself the question of cleavage by **carnosinase** in the blood remains open. Both routes are often mentioned together in reviews, yet they are different supply forms with different states of investigation.

Carnosine or anserine — which substance was tested in the studies?

Several of the most cited controlled studies examined **anserine-carnosine mixtures** in older adults and reported endpoints for verbal memory performance as well as inflammatory markers. Individual studies investigated **orodispersible levo-carnosine** in chronic heart failure as well as **L-carnosine** in oxaliplatin-induced neuropathy and in childhood diabetic nephropathy. Readers of the literature must therefore note the tested preparation and not only the substance name.

What do animal and cell studies contribute to the assessment?

They supply the **mechanisms**: binding of reactive carbonyl compounds, antioxidant activity, protection against glycation and cross-linking. Animal findings such as delayed diabetic deterioration in mice or protection of LDL against oxidation and glycation are **preclinical**. They justify hypotheses and the choice of endpoints, yet they do not replace controlled testing in humans. Where the latter is absent, the mechanism explains a finding; it is not the finding itself.

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