

Almond

OVERVIEW

Almonds (*Prunus dulcis*) are perennial tree crops producing nutrient-dense seeds widely consumed globally for their health benefits and culinary versatility [Yada, Huang & Lapsley, 2013]. Almond cultivation in California began in the 1800s, likely from cultivars imported from southern France, and modern varieties largely descend from two unrelated founders, *Nonpareil* and *Mission*, selected from these early introductions [Yada, Huang & Lapsley, 2013].

The overwhelming majority of commercially grown almonds in Australia belong to the North American variety of almonds, and include the cultivars such as *Nonpareil*, *Carmel*, *Price*, and *Monterey*.

- As of 2024, 42% of Australian households regularly consume fresh almonds, with 880 g consumed *per capita* [Hort Innovation - AUS Statistics Handbook 24/25, 2026]. 153,500 tons of almonds worth \$870.3 million (AUD) were produced in 2023-2024 across Australia [Hort Innovation - AUS Statistics Handbook 24/25, 2026]. Victoria led almond production (44.9%), followed by South Australia (30.0%), and New South Wales (24.6%) [Hort Innovation - AUS Statistics Handbook 24/25, 2026].



Raw 'Sweet' Almond



Roasted Almond



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Almond Cultivation

Fenster, 2021, reported that regenerative almond orchards showed markedly better soil health and biodiversity compared to conventional orchard, with 62% higher soil organic matter (3.88% vs. 2.39%, $p = 0.03$), significantly greater labile carbon and nitrogen, and ~6× faster water infiltration ($p = 0.04$) [Fenster, Oikawa & Lundgren, 2021]. Microbial biomass was substantially higher, including *Gram+* and *Actinobacteria* populations ($p \leq 0.05$), and invertebrate biomass and species richness more than doubled ($p < 0.01$). Almond kernel yields and nutrient density was largely similar, except for magnesium, which was 9% higher in regenerative almonds ($p < 0.001$) [Fenster, Oikawa & Lundgren, 2021].

Organic vs Conventional Almond

Organic almonds exhibited a healthier lipid profile compared to conventional ones, with PUFA increased by 15% ($p \leq 0.05$) and linoleic acid rising by 12% ($p \leq 0.05$), while SFA decreased by 6.5% ($p \leq 0.05$), and MUFA and oleic acid showed no significant differences ($p > 0.05$) [García-Martínez, et al., 2025]. Irrigation strategy strongly influenced fatty acids where intense irrigation increased SFA content (8.8% increase, $p \leq 0.05$) and lowered PUFA, whereas dry land(water-stress) conditions boosted PUFA content (7.2% increase, $p \leq 0.05$), but reduced MUFA and oleic acid ($p \leq 0.05$) [García-Martínez, et al., 2025]. Plant cover significantly reduced SFA (5.07% decrease, $p \leq 0.05$) and improved thrombogenic index (5.24% decrease, $p \leq 0.05$), though it did not alter oleic or linoleic acid levels ($p > 0.05$) [García-Martínez, et al., 2025]. Overall, organic farming combined with support or dry irrigation and vegetation cover produced almonds with lower atherogenic (AI) and thrombogenic (TI) indices ($p \leq 0.05$) and higher beneficial fatty acid content for health (BFAH, $p \leq 0.05$) [García-Martínez, et al., 2025].

Organic almonds grown under rainfed conditions in southeastern Spain showed significantly higher levels of several bioactives compared to conventional almonds [Cárceles Rodríguez et al., 2023]. Oleic acid increased by 9% (12.1 vs 11.1 g per 100 g DW, $p < 0.001$) and linoleic acid by 9.1% (5.73 vs 5.25 g per 100 g DW, $p < 0.01$) in organic almonds. SFA rose by 8.6% ($p < 0.001$), MUFA by 8.0% ($p < 0.001$), and PUFA by 9.0% ($p < 0.01$) [Cárceles Rodríguez et al., 2023]. However, total phenolic content (TPC) was 15.8% lower in organic almonds overall (251.0 vs 298.0 mg GAE per 100 g, $p < 0.05$), though cultivar-specific effects were noted, where *Marcona* had 38.6% higher TPC under organic management ($p < 0.05$), *Desmayo* and *Largueta* showed no significant difference ($p > 0.05$) [Cárceles Rodríguez et al., 2023]. These results suggest that organic farming enhances fatty acid content but may variably affect total phenolic content depending on cultivar.

Almond Cultivars

In almonds, oleic acid (MUFA (18:1)) was the dominant bioactive lipid across all cultivars, with *Nonpareil*, *Carmel*, and *Price* cultivars showing

distinct profiles and seasonal shifts [Abdallah, Ahumada & Gradziel, 1998]. *Nonpareil* almonds are reported to contain 25.14 g oleic and 9.41 g linoleic acid per 100 g FW, *Carmel* is reported to contain 26.35 g oleic and 10.09 g linoleic acid per 100 g FW, *Price* almonds reported 26.7 g oleic and 7.44 g linoleic acid per 100 g FW [Abdallah, Ahumada & Gradziel, 1998]. Across all 21 almond cultivars listed in Abdallah, 1998, the mean Oleic : Linoleic (MUFA (18:1): PUFA (18:2)) acid ratio was 3.09 for the 1995 harvest, and 3.21 for the 1996 harvest, an increase of 3.7%, but not statistically significant ($p > 0.05$) [Abdallah, Ahumada & Gradziel, 1998]. ANOVA confirmed significant genotype effects for oil and major fatty acids in almond cultivars ($p < 0.01$), with additional year and 'genotype x year' interactions ($p < 0.01$ for oil, palmitic, oleic, and linoleic acids) [Abdallah, Ahumada & Gradziel, 1998].

Almond skins are the primary source of phenolic content in whole almonds [Bolling, 2017]. Across seven North American California cultivars, total phenolic content in almond skins ranged from 58.0 to 159.0 mg GAE per 100 g FW [Bolling et al., 2010]. Seasonally, when 2005 cultivated almonds are compared to 2007 cultivated almonds total phenolic content phenols and FRAP antioxidant activity did not significantly differ by year ($p > 0.05$) in *Carmel*, *Nonpareil* and *Butte* cultivars [Bolling et al., 2010]. A scoping review of almonds (Barral-Martínez, 2021) reported that total phenolic content among almond cultivars vary widely, ranging from 30.0 to 16371.0 mg GAE per 100 g DW [Barral-Martínez et al., 2021]. The Italian almond cultivar '*Duro*' exhibited the highest phenolic content at 16371.0 mg per 100 g DW, followed by the Portuguese cultivar [Barreira et al., 2008]. In comparison, North American Californian cultivars such as *Nonpareil* (530.0 mg per 100 g DW) and *Carmel* (610.0 mg per 100 g DW), and other European almond cultivars such as the *Pegarinhos* (922.0 mg per 100 g DW), which can sometimes be 'two-seeded' rather than the standard 'one-seeded' almond, reported much lower range of total phenolic content (30.0 – 1100.0 mg per 100 g DW) [Barreira et al., 2008; Barral-Martínez et al., 2021].

The *Duro* almond cultivar reported stronger antioxidant activity compared to other European almonds, with significantly lower EC50 values across multiple antioxidant assays ($p < 0.05$) [Barreira et al., 2008]. In the TBARS (Thiobarbituric Acid Reactive Substances) lipid peroxidation assay, *Duro* reported an EC50 of 0.18 mg/mL, compared to 4.29 mg/mL for *Pegarinhos* (two-seeded), representing a 2283% increase in antioxidant efficiency. In the DPPH (2,2-diphenyl-1-picrylhydrazyl) radical scavenging assay, *Duro* reported an EC50 of 0.70 mg/mL, while *Pegarinhos* reported 4.74 mg/mL, a 577% improvement over *Pegarinhos* [Barreira et al., 2008]. The *Duro* almond cultivar consistently outperformed all other European cultivars, and these differences were statistically significant ($p < 0.05$), and strongly correlated with total phenolic content, with a negative linear correlation ($p < 0.008$) between phenolic concentration and EC50 values [Barreira et al., 2008]. Bolling, 2010, also reported that total phenolic content were significantly correlated with antioxidant capacity (FRAP,



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$R^2 = 0.85$, $p = 0.016$), underscoring the functional relevance of cultivar-driven differences in almond phenolic content [Bolling et al., 2010].

Almonds are also reported to contain a good levels of phytosterols (87.8 mg per 100 g FW), assuming an average oil content of 54.3% FW almonds [Kornsteiner-Krenn, Wagner & Elmadfa, 2013]. The dominant sterol is β -sitosterol, contributing 77.5 mg per 100 g FW, followed by ergosterol at 8.0 mg per 100 g FW, and campesterol at 2.0 mg per 100 g FW [Kornsteiner-Krenn, Wagner & Elmadfa, 2013]. Almond is reported to have higher phytosterol content compared to other nuts (Brazil nuts, peanuts, walnuts, cashews) except for pistachios, which as 68% higher phytosterol content [Kornsteiner-Krenn, Wagner & Elmadfa, 2013].



NUTRITIONAL INFORMATION

The nutrient information panel (NIP) for the major types of almond, raw, and roasted, listed in the following table are sourced from the Australian Food Composition Database (AFCD) used by clinicians and dietitians [FSANZ - AFCD, 2025]. The AFCD dietary information is a standard guideline, and packaging for commercially sold almonds may contain different amounts of protein, total dietary fibre (TDF), and essential minerals depending on cultivation, harvest method, storage time and storage conditions.

The proportion of soluble dietary fibre (SDF) & insoluble dietary fibre (IDF) in almond is estimated using a published analytical method [Chen, Lapsley & Blumberg, 2006]. Note that cooking almonds may change the proportion of SDF:IDF, but specific information is currently unavailable. An average adult human male requires 31.0 g while an average human female requires 25.0 g of TDF intake per day for healthy gut function [Cleveland Clinic - Fiber, 2025].

Maximum allowed levels of natural toxicants Arsenic (As) and Cadmium (Cd) present in almond are indicated based on FSANZ Food Standards Code [FSANZ - Food Standards Code, 2025]. Average almond weight and serving are cited from the Harvard T.H. Chan School of Public Health [Harvard T.H. Chan School of Public Health - Almond2, 2025].

NIP Values shown for: 100.0 g.

Average almond weight: 1.2 g

One serve of almond (23 in a cup): 28.3 g (1 ounce)

Information regarding Recommended Dietary Intake (RDI) for vitamins and essential minerals are sourced from NIH Fact Sheets [NIH - Office of Dietary Supplements, 2025]. Please consult an expert clinician or certified dietitian to address your specific needs, or seek information via Healthdirect Australia [Healthdirect Australia, 2025].

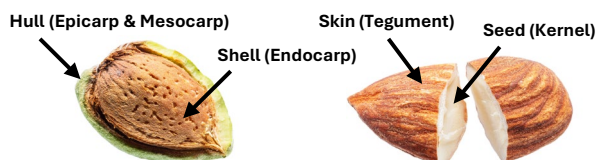


SEASONALITY IN AUSTRALIA

Fresh almond harvest in Australia occurs in the three months of Feb-Apr, and almond is primarily cultivated in the southern regions of Australia [Hort Innovation - AUS Statistics Handbook 24/25, 2026].

The primary cultivars planted in Australia are as follows:

Almond: Nonpareil, Carmel, Price, Monterey (all are North American almond cultivars)



Nutrient Information Panel (NIP) for Almond ^(a)

Citations: FSANZ - AFCD, 2025; NIH - Office of Dietary Supplements, 2025; Healthdirect Australia, 2025

	Almond Kernel + Skin (Unsalted) (Raw)	Almond Kernel + Skin (Unsalted) (Roasted)	Almond Kernel + Skin (Meal)
Basic Nutrients (per 100 g)			
Energy	2385 kJ	2461 kJ	2568 kJ
Total Protein (incl. Amino acids)	19.7 g	19.7 g	20.5 g
Available carbohydrate, with sugar alcohols	5.4 g	5.4 g	5.1 g
Total Sugars (Free + Added)	5.2 g	5.2 g	3.9 g
Tryptophan	228.0 mg	228.0 mg	228.0 mg
Dietary Fibre (per 100 g), (%RDI, range shown Male - Female adults, or both) ^(b)			
Total Dietary Fibre (TDF) RDI: 31.0 - 25.0 g	10.9 g (35.2 - 43.6%)	10.9 g (35.2 - 43.6%)	8.9 g (28.7 - 35.6%)
Soluble Dietary Fibre (SDF)	2.2 g	2.2 g	1.8 g
Insoluble Dietary Fibre (IDF)	8.7 g	8.7 g	7.1 g
Vitamins (per 100 g), (%RDI, range shown Male - Female Adults, or both) ^(b)			
Vit. B1 (Thiamin) RDI: 1.2 - 1.1 mg	0.19 mg (15.8 - 17.3%)	0.162 mg (13.5 - 14.7%)	0.14 mg (11.7 - 12.7%)
Vit. B2 (Riboflavin) RDI: 1.3 - 1.1 mg	0.07 mg (5.4 - 6.4%)	0.066 mg (5.1 - 6.0%)	0.9 mg (69.2 - 81.8%)
Vit. B3 (Niacin Equiv.) RDI: 16.0 - 14.0 mg	4.3 mg (26.9 - 30.7%)	4.08 mg (25.5 - 29.1%)	3.8 mg (23.8 - 27.1%)
Vit. B5 (Pantothenic acid) RDI: 5.0 mg	0.25 mg (5.0%)	N.R.	0.3 mg (6.0%)
Vit. B6 (Pyridoxine) RDI: 1.3 mg	0.09 mg (6.9%)	0.08 mg (6.2%)	0.14 mg (10.8%)
Vit. B7 (Biotin) RDI: 30.0 µg	N.R.	N.R.	56.0 µg (186.7%)
Vit. B9 (Folate) RDI: 400.0 µg	37.0 µg (9.3%)	30.0 µg (7.5%)	43.0 µg (10.8%)
Vit. E (Tocopherols) RDI: 15.0 mg	31.42 mg (209.5%)	28.58 mg (190.5%)	22.62 mg (150.8%)
Essential Minerals (per 100g), (%RDI, range shown Male - Female adults, or both) ^(b)			
Calcium (Ca) RDI: 1000.0 mg	265.0 mg (26.5%)	265.0 mg (26.5%)	220.0 mg (22.0%)
Potassium (K) RDI: 3400.0 - 2600.0 mg	796.0 mg (23.4 - 30.6%)	796.0 mg (23.4 - 30.6%)	640.0 mg (18.8 - 24.6%)
Sodium (Na) RDI: 920.0 mg	0.0 mg (0.0%)	0.0 mg (0.0%)	5.0 mg (0.5%)
Magnesium (Mg) RDI: 420.0 - 320.0 mg	266.0 mg (63.3 - 83.1%)	266.0 mg (63.3 - 83.1%)	260.0 mg (61.9 - 81.3%)
Manganese (Mn) RDI: 2.3 - 1.8 mg	2.95 mg (128.3 - 163.9%)	N.R.	1.9 mg (82.6 - 105.6%)
Iron (Fe) RDI: 8.0 - 18.0 mg	3.75 mg (46.9 - 20.8%)	3.75 mg (46.9 - 20.8%)	3.1 mg (38.9 - 17.2%)
Zinc (Zn) RDI: 14.0 - 8.0 mg	3.63 mg (25.9 - 45.4%)	3.63 mg (25.9 - 45.4%)	3.4 mg (24.3 - 42.5%)
Phosphorus (P) RDI: 700.0 mg	525.0 mg (75.0%)	420.0 mg (60.0%)	470.0 mg (67.1%)
Copper (Cu) RDI: 0.9 mg	0.85 mg (94.4%)	N.R.	1.0 mg (111.1%)
Chromium (Cr) RDI: 35.0 - 25.0 µg	18.8 µg (53.7 - 75.2%)	N.R.	N.R.
Selenium (Se) RDI: 55.0 µg	1.5 µg	1.5 µg	2.0 µg
Arsenic (As) Maximum allowed level: 1.0 mg / 1.0 kg of almond	3.0 µg (0.3% of maximum level)	N.R.	N.R.
Cadmium (Cd) Maximum allowed level: 0.1 mg / 1.0 kg of almond	0.0 µg (0.0% of maximum level)	N.R.	N.R.

- Data shown here per 100 g of raw almond (kernel), roasted almond (kernel), and almond meal (kernels boiled in hot water, skin removed, and ground into a meal), please note that 1 standard serving of almond ~28 g.
- Single value indicates %RDI for both Male and Female adults. Consult NIH Dietary Supplement Fact Sheets for specific %RDI values for children (below Age 18), older adults (above Age 50), and adults with specific needs & disease conditions, alternatively, consult Healthdirect Australia for relevant information [NIH - Office of Dietary Supplements, 2025; Healthdirect Australia, 2025].
- N.R. or 'Not Reported' in the databases.



STORAGE AND COOKING

Post-harvest Storage

Storage conditions significantly influenced microbial growth and mycotoxin contamination in raw almonds stored over nine months [Rodrigues et al., 2023]. Rodrigues, 2023, reports that both tested raw almond cultivars, *Guara* and *Lauranne*, reported the lowest mold counts at under 60% RH (relative humidity) at 25 °C, while high RH (70%) and prolonged storage markedly increased fungal diversity and aflatoxin risk ($p < 0.05$) [Rodrigues et al., 2023]. At 70% RH, aflatoxin B1 reached 53.9 µg/kg and total aflatoxins 264.1 µg/kg, far exceeding European Union (EU) safety limits, whereas 60% RH maintained much lower levels (AFB1 = 2.7 µg/kg) [Rodrigues et al., 2023]. *Lauranne* exhibited extreme *Penicillium* toxin accumulation at 4 °C after six months, highlighting that refrigeration alone does not guarantee almond storage safety [Rodrigues et al., 2023]. Overall, 60% RH at 25 °C was the reported as the effective condition for minimizing microbial growth and mycotoxin accumulation during long-term almond storage [Rodrigues et al., 2023].

High-barrier bags (HBB) preserved dry-roasted *Nonpareil* almond quality in long-term storage (>1 year) better compared to polypropylene bags (PPB) ($p < 0.05$) [Cheely et al., 2018]. Cheely, 2018, reports that the earliest rejection of stored almonds was at 12 months for PPB stored almonds stored at 35 °C & 65% RH, whereas HBB almonds stored at 4–25 °C maintained hedonic scores above 7 and low rejection rates [Cheely et al., 2018]. Oxidation markers such as rancid and 'painty' odours and flavours increased with temperature ($p < 0.05$), and crunchiness declined sharply under high temperature and humidity, especially in PPB. Instrumental force peaks (almond texture assessment) were the strongest predictor of overall almond acceptability ($R^2 = 68.5\%$), highlighting the importance of cool storage and HBB for long-term quality retention [Cheely et al., 2018].

Over 12 months of storage, raw almonds reported a significant decline in bioactive compounds and slight shifts in fatty acid composition. El Bernoussi, 2024, reported that total phenolic content dropped by about 34% (from 1951.0 to 1293.0 mg GAE per 100 g, $p < 0.05$), reducing antioxidant potential [El Bernoussi et al., 2024]. SFA increased by 15%, MUFA decreased by 5%, primarily due to a reduction in oleic acid content (5.3% loss) and a rise in linoleic acid (14% increase) (both $p < 0.05$), slightly increasing susceptibility to oxidation [El Bernoussi et al., 2024]. PUFA also rose by 14%, shifting the MUFA/PUFA balance. α -Tocopherol remained the dominant vitamin E form, but total tocopherols decreased by 12.5% ($p < 0.05$), indicating reduced oxidative stability [El Bernoussi et al., 2024]. Overall, sweet almonds retained good nutritional quality, but the loss of phenolic and tocopherol content combined with fatty acid changes suggests a gradual decline in antioxidant potential during long-term storage.

Blanching Almonds

Blanching significantly affects the total phenolic content (TPC) of almonds, particularly in the skin where most phenolics are concentrated (65.0% of TPC in *Nonpareil*, 50.1% in *Carmel*, 64.4% in *Price* almond cultivars) [Milbury et al., 2006], and blanching removes this layer, leading to a major reduction in phenolic content [Chang et al., 2016]. Hot water blanching removes up to 90% of extractable phenolics within 10 min, with 73% lost in just 2 minutes, leading to a substantial reduction in total phenolic content in the final product [Bolling, 2017].

Raw almond skins contain significantly more phenolics than blanched almond skins, with total phenolic content of 3474.1 mg per 100 g FW reported in natural skin vs 278.9 mg per 100 g FW in blanched skins, representing a 92% reduction due to blanching ($p < 0.01$) [Mandalari et al., 2016]. During simulated digestion, raw almond skins released up to 68.5% of phenolic acids and 65.6% of flavonols, while blanched almond skins released 64.7% and 52.3%, respectively, showing significantly lower bioaccessibility across all phenolic classes ($p < 0.01$) [Mandalari et al., 2016]. The food matrix also strongly influenced bioactive release, where water and crispbread enhanced phenolic bioaccessibility from raw almond skins, while biscuits and crispbread were optimal for blanched almond skins ($p < 0.01$) [Mandalari et al., 2016]. Full-fat milk consistently reduced phenolic recovery and antioxidant activity due to protein–phenolic binding, with significantly lower free phenol levels and DPPH (2,2-diphenyl-1-picrylhydrazyl) scavenging capacity ($p < 0.01$) [Mandalari et al., 2016].

Blanching caused significant shifts in almond fatty acid content, on average, oleic acid (C18:1) decreased from 686.7 mg per 100 g FW in raw

almonds to 533.6 mg (22.3% decrease, $p < 0.05$) after blanching [Oliveira et al., 2020]. Linoleic acid (C18:2) dropped sharply from 108.5 mg per 100 g FW to 5.4 mg (95.0% decrease) with blanching ($p < 0.05$) [Oliveira et al., 2020]. MUFA content fell by 16.1% with blanching, while PUFA increased dramatically, rising from 193.2 mg in raw almonds to 278.2 mg (44.0% increase) after blanching ($p < 0.05$) [Oliveira et al., 2020]. SFA showed minor changes, increasing slightly with blanching (7.5% increase, $p < 0.05$) [Oliveira et al., 2020].

Roasting Almonds

Almonds are typically roasted using the 'Hot-Air Oven' method, where oven temperatures between 129.5 °C to 154.5 °C are applied on almonds for 10–30 min to trigger a *Maillard* reaction [Pedron, Jaouhari & Bordiga, 2025]. Roasting significantly altered the bioactive content of raw almonds, with longer and hotter roasting conditions leading to substantial increases in total phenolic content (TPC) from 111.5 mg per 100 g FW in raw almonds to 276.6 mg per 100 g FW in roasted almonds, reported after roasting at 200 °C for 20 minutes (148% increase, $p < 0.05$), while total flavonoids increased from 36.1 to 78.5 mg per 100 g FW (117% increase, $p < 0.05$) [Lin et al., 2016]. Proanthocyanidins also reported a large increase from 5.1 to 36.5 mg per 100 g FW (621% increase, $p < 0.05$). Roasting also transformed glycosylated flavonoids into aglycones, with compounds like isorhamnetin and quercetin increasing more than 3000% ($p < 0.05$) [Lin et al., 2016]. These bioactive increases were strongly correlated with antioxidant activity, with total phenolics and phenolic acids showing $R^2 > 0.92$ against TEAC (Trolox Equivalent Antioxidant Capacity) and DPPH (2,2-diphenyl-1-picrylhydrazyl) assays ($p < 0.001$) [Lin et al., 2016]. Roasting has a biphasic effect where initial heating (5–10 min) reduces flavonoids and phenolic acids, but extended roasting (20 min at 200 °C) increases their bioaccessibility, resulting in up to 25% higher recovery compared to raw almonds [Bolling, 2017]. Roasting at moderate conditions (150–180 °C for 10–20 min) can increase extractable phenolics by up to 25%, likely due to the breakdown of polymerized compounds and hydrolysis of glycosides, while excessive roasting may degrade thermolabile phenolics [Chang et al., 2016].

Roasting also caused significant shifts in almond fatty acid content, on average, oleic acid (C18:1) decreased from 686.7 mg per 100 g FW in raw almonds to 478.5 mg (30.3% decrease, $p < 0.05$) after roasting [Oliveira et al., 2020]. Linoleic acid (C18:2) dropped sharply from 108.5 mg per 100 g FW to 18.4 mg (83.0% decrease) with roasting ($p < 0.05$) [Oliveira et al., 2020]. MUFA content fell by 28.6% with roasting, while PUFA increased dramatically, rising from 193.2 mg in raw almonds to 465.9 mg (141.1% increase) after roasting ($p < 0.05$) [Oliveira et al., 2020]. SFA showed minor changes, decreasing slightly with roasting (3.3% decrease, $p < 0.05$) [Oliveira et al., 2020].

Grundy, 2016, reports that roasting significantly alters almond composition and reduces moisture content by 40.7–59.1%, disrupts cell wall integrity, and causes lipid coalescence, which affects bioactive release [Grundy, Lapsley & Ellis, 2016]. Despite these changes, roasted almonds do not show significant differences in gastric lipid release or duodenal digestion rates compared to raw almonds, suggesting that roasting does not significantly enhance lipid bioaccessibility *in vivo* [Grundy, Lapsley & Ellis, 2016]. Grundy, 2016, also reports statistically significant changes in almond structure and phenolic loss due to roasting ($p < 0.05$), but no significant differences in postprandial lipid digestion or plasma lipid levels between raw and roasted forms ($p > 0.05$) [Grundy, Lapsley & Ellis, 2016].

Kong, 2008, also reported that roasted almonds disintegrated significantly faster than raw almonds under simulated gastric conditions, with the half-time for 50% mass loss reduced from 11 h in raw almonds to 7.4 hours in roasted samples ($p < 0.05$) [Kong & Singh, 2009]. After 5 h, roasted almonds retained less weight (0.77 g/g initial vs. 0.93 g/g initial) and dry mass (0.52 g/g initial vs. 0.61 g/g initial) than raw almonds, indicating greater nutrient and bioactive release [Kong & Singh, 2009]. Soaking in gastric juice softened both types of almonds, but raw almonds remained more rigid, showing higher stiffness (67.23 N/mm vs. 49.68 N/mm for roasted, $p < 0.05$), which correlated with slower breakdown [Kong & Singh, 2009]. Raw almonds absorbed more gastric fluid and swelled more than roasted (103% v. 69% moisture after 16 h; volume increase 44% vs 32%, $p < 0.05$), contributing to prolonged satiety [Kong & Singh, 2009]. Microstructural analysis revealed that roasting created porous channels and disrupted cell walls, enhancing gastric juice penetration and accelerating digestion compared to raw almonds [Kong & Singh, 2009].



BIOACTIVES IN ALMOND

Almond Nutrients

Almond is an abundant source of vitamin E (Tocopherols) and also contains smaller amounts of vitamin B₁ (Thiamin), vitamin B₂ (Riboflavin) and vitamin B₃ (Niacin equiv.) [Asad & Malik, 2024; FSANZ - AFCD, (2025)]. Almond is also a good source of many essential minerals, including calcium, potassium, magnesium, manganese, iron, zinc, phosphorus, and copper [FSANZ - AFCD, (2025)]. Almond is great source of both insoluble (IDF) and soluble dietary fibre (SDF). Almond cell walls are primarily composed of insoluble fibre cellulose microfibrils embedded in a matrix of soluble fibre pectic polysaccharides rich in arabinose and galacturonic acid [Mandalari et al., 2008]. Minor sugars such as xylose and galactose are also present, along with some glucose, and this structure makes almond polysaccharides resistant to digestion in the upper gastrointestinal tract, allowing them to reach the colon for fermentation and potential prebiotic effects [Mandalari et al., 2008]. Almonds provide multiple health benefits, help lower LDL cholesterol, support weight management through satiety and partial fat malabsorption, improve postprandial glucose control, and offer antioxidant and anti-inflammatory effects that reduce oxidative stress and inflammatory biomarkers, contributing to cardiovascular and metabolic health [Kamil & Chen, 2012].

Almond Fatty Acids & Polyphenols

Almonds have a high lipid content, typically ranging from 43% to 51%, with variations depending on cultivar and growing conditions [Gonçalves et al., 2023]. Their fatty acid profile is dominated by monounsaturated fatty acids (MUFA, ~60%), primarily oleic acid (C18:1), followed by polyunsaturated fatty acids (PUFA, ~30%), mainly linoleic acid (C18:2) [Gonçalves et al., 2023]. Saturated fatty acids (SFA) form a small fraction, with palmitic (C16:0) and stearic (C18:0) acids being the most common [Gonçalves et al., 2023]. This composition gives almonds a favourable unsaturated-to-saturated fat ratio, which is associated with cardiovascular health benefits. Overall, almonds are considered a rich source of heart-healthy fats, contributing to improved lipid profiles when included in the diet. Almond oils are also rich in phytosterols, with total sterol content varying depending on cultivar [Bertrand & Özcan, 2020]. The dominant phytosterol in almond is β -sitosterol, while δ -5-avenasterol, campesterol and stigmasterol contribute smaller amounts to overall phytosterol content [Bertrand & Özcan, 2020].

Almonds are a significant source of phenolics, mainly concentrated in the skins, which contain 70–100% of the total phenols in the fruit [Barral-Martinez et al., 2021]. Key compounds include flavonoids (catechin, epicatechin, naringenin glycosides), phenolic acids, and tannins [Barral-Martinez et al., 2021]. Whole kernels have variable total phenolic content depending on cultivar (e.g., *Nonpareil* reports 530.0 mg per 100 g DW, *Carmel* reports 610.0 mg per 100 g DW), while blanched skins show high levels of flavan-3-ols and flavonol glycosides [Barral-Martinez et al., 2021]. Almond phenolics contribute to strong antioxidant potential and health-promoting properties [Barral-Martinez et al., 2021].



FOOD SAFETY & ALLERGEN INFORMATION

Almond is generally safe to consume, unless contaminated or expired. Please consult an expert clinician or certified dietitian if you require specific health information tailored to your daily dietary intake. Major almond allergens include *Pru du 3*, *Pru du 4*, *Pru du 5*, *Pru du 6*, *Pru du 8* and *Pru du 10*, which have been included in the WHO-IUIS list of allergens [Bezerra, Ribeiro & Iglejas, 2021]. When screening for allergies, please consult the FSANZ database [FSANZ - Food Allergen Portal, 2025].

Concentration of Bioactives in Almond Fresh Weight (FW) ^(a) & Dry Weight (DW) ^(b) (per 100 g)

Citations: (1) [Abdallah, Ahumada & Gradziel, 1998]; (2) [Asad & Malik, 2024]; (3) [Barreira et al., 2008]; (4) [Barral-Martinez et al., 2021]; (5) [Bertrand & Özcan, 2020]; (6) [Bolling et al., 2010]; (7) [Bolling, 2017]; (8) [Chang et al., 2016]; (9) [Moreno-Gracia et al., 2021]; (10) [Grundy, Lapsley & Ellis, 2016]; (11) [Kornsteiner-Krenn, Wagner & Elmadafa, 2013]; (12) [Lin et al., 2016]; (13) [Lipan et al., 2020]; (14) [Milbury et al., 2006]; (15) [Oliveira et al., 2020]; (16) [Özcan, 2023]; (17) [Pedron, Jaouhari & Bordiga, 2025]; (18) [Temiz et al., 2025]; (19) [Yada, Huang & Lapsley, 2013]; (20) [Zhu, Wilkinson & Wirthensohn, 2015]; (21) [Gonçalves et al., 2023];

	Almond Kernel + Skin (Raw)	Roasted Almond Kernel + Skin (Raw)
β -sitosterol	103.00-206.00 mg ^(a) ; 1.92-288.96 mg ^(b)	L.S.I. ^(c)
δ -5-avenasterol	0.25-43.01 mg ^(b)	L.S.I. ^(c)
Campesterol	4.10-11.80 mg ^(b) ; 0.06-12.74 mg ^(b)	L.S.I. ^(c)
Stigmasterol	1.30-9.80 mg ^(a) ; N.P. ^(d) , 7.33 mg ^(b)	L.S.I. ^(c)
Total Phytosterols ^(e)	270.00 mg ^(a) ; 69.34-105.30 mg	L.S.I. ^(c)
Oleic acid	31.29-31.53 g ^(a) ; 12.54-62.11 g ^(b)	28.51-37.65 g ^(a) ; 29.08-41.66 g ^(b)
Linoleic acid	9.40-15.10 g ^(a) ; 1.55-27.43 g ^(b)	8.23-12.55 g ^(a) ; 10.52-15.19 g ^(b)
α -Linolenic acid	0.003 g ^(a) ; 0.01-8.21 g ^(b)	L.S.I. ^(c)
Palmitoleic acid	0.227 g ^(a) ; 0.01-1.85 g ^(b)	L.S.I. ^(c)
Saturated fatty acids (SFA)	3.06-4.70 g ^(a) ; 2.47-5.67 g ^(b)	2.61-4.11 g ^(a) ; 3.34-4.64 g
Monounsaturated fatty acids (MUFA)	24.90-36.10 g ^(a) ; 24.48-48.80 g ^(b)	28.54-37.80 g ^(a) ; 30.05-41.66 g ^(b)
Polyunsaturated fatty acids (PUFA)	9.40-15.10 g ^(a) ; 7.24-19.37 g ^(b)	8.23-12.55 g ^(a)
Total Fatty Acids ^(e)	44.70-47.70 g ^(a) ; 35.96-63.64 g ^(b)	39.38-54.46 g ^(a)
Cyanidin (aglycone)	N.P. ^(d) , 4.40 mg ^(a)	L.S.I. ^(c)
Total Anthocyanins ^(e)	L.S.I. ^(c)	L.S.I. ^(c)
Squalene	9.64-11.31 mg ^(a)	L.S.I. ^(c)
Total Triterpenes ^(e)	L.S.I. ^(c)	L.S.I. ^(c)
Tyrosol	0.14 mg ^(a)	L.S.I. ^(c)
Hydroxytyrosol	0.20 mg ^(a)	L.S.I. ^(c)
Apigenin	0.010-1.047 mg ^(a)	L.S.I. ^(c)
Caffeic acid	0.04-0.28 mg ^(a)	L.S.I. ^(c)
Chlorogenic acid	N.P. ^(d) , 23.92 mg ^(a) ; 161.00 mg ^(b)	5.85-46.34 mg ^(a) ; 40.00-322.00 mg ^(b)
Catechin	0.10-36.60 mg ^(a) ; 0.95-20.00 mg ^(b)	N.P. ^(d) , 9.93 mg ^(a) ; N.P. ^(d) , 69.00 mg ^(b)
Epicatechin	0.01-26.60 mg ^(a) ; 0.32-16.00 mg ^(b)	N.P. ^(d) , 13.24 mg ^(a) ; N.P. ^(d) , 92.00 mg ^(b)
Protocatechuic acid	0.03-13.08 mg ^(a) ; 0.13-88.00 mg ^(b)	3.57-22.88 mg ^(a) ; 23.00-159.00 mg ^(b)
p-Coumaric acid	N.P. ^(d) , 1.34 mg ^(a) ; 9.00 mg ^(b)	0.44-0.04 mg ^(a) ; 3.00-42.00 mg ^(b)
Ellagic acid	0.002-0.035 mg ^(a)	L.S.I. ^(c)
Ferulic acid	0.01-2.15 mg ^(a)	L.S.I. ^(c)
Gallic acid	0.05-4.00 mg ^(a) ; 4.00 mg ^(b)	1.72-300.00 mg ^(a) ; 11.00-102.00 mg ^(b)
p-Hydroxybenzoic acid	N.P. ^(d) , 1.23 mg ^(a)	L.S.I. ^(c)
Quercetin (aglycone)	N.P. ^(d) , 3.58 mg ^(a) ; 0.02-1.00 mg ^(b)	N.P. ^(d) , 5.47 mg ^(a) ; N.P. ^(d) , 38.00 mg ^(b)
Quercetin-3-O-galactoside	0.24-1.37 mg ^(a) ; 0.04-2.00 mg ^(b)	L.S.I. ^(c)
Quercetin-3-O-glucoside	0.04-0.16 mg ^(a) ; 0.24-1.03 mg ^(b)	L.S.I. ^(c)
Isorhamnetin (aglycone)	0.01-3.20 mg ^(a) ; 0.09-4.00 mg ^(b)	N.P. ^(d) , 20.29 mg ^(a) ; N.P. ^(d) , 141.00 mg ^(b)
Isorhamnetin-3-O-rutinoside ^(e)	1.12-74.10 mg ^(a) ; 9.86-19.07 mg ^(a) , ^(b)	L.S.I. ^(c)
Isorhamnetin-3-O-glucoside ^(e)	0.01-19.07 mg ^(a) , ^(b) ; 9.86-19.07 mg ^(a) , ^(b)	L.S.I. ^(c)
Kaempferol (aglycone)	N.P. ^(d) , 0.49 mg ^(a) ; 0.01-2.00 mg ^(b)	N.P. ^(d) , 13.53 mg ^(a) ; N.P. ^(d) , 94.00 mg ^(b)
Kaempferol-3-O-rutinoside	0.11-23.30 mg ^(a) ; 0.71-28.00 mg ^(b)	L.S.I. ^(c)
Luteolin	0.034-0.427 mg ^(a)	L.S.I. ^(c)
Naringin	0.004-0.011 mg ^(a)	L.S.I. ^(c)
Naringenin (aglycone)	0.01-9.74 mg ^(a) ; 0.02-28.00 mg ^(b)	N.P. ^(d) , 9.21 mg ^(a) ; N.P. ^(d) , 64.00 mg ^(b)
Naringenin-7-O-glucoside	0.07-5.88 mg ^(a) ; 0.09-0.38 mg ^(b)	L.S.I. ^(c)
Rutin	0.00-1.66 mg ^(a) ; 0.06-28.00 mg ^(b)	L.S.I. ^(c)
Resveratrol	0.01-0.15 mg ^(a)	L.S.I. ^(c)
Vanillin	0.08-0.18 mg ^(a)	L.S.I. ^(c)
Vanillic acid	0.03-13.97 mg ^(a) ; 0.10-94.00 mg ^(b)	2.93-35.54 mg ^(a) ; 20.00-247.00 mg ^(b)
Sinapic acid	0.06-0.35 mg ^(a)	L.S.I. ^(c)
Total Phenolics ^(e) , ^(f)	2.00-1340.00 mg ^(a) ; 30.00-16400.00 mg ^(b)	25.46-1920.00 mg ^(a)
Glutamic acid	3558.70-6764.80 mg ^(a)	L.S.I. ^(c)
Tryptophan	211.00 mg ^(a)	L.S.I. ^(c)

- Indicates a 'range of mean values' reported for 'fresh weight' (FW) from multiple publications. In FW publications, water content is retained prior to chemical analysis of bioactives.
- Indicates a 'range of mean values' reported for 'dry weight' (DW) from multiple publications. In DW publications, almond is freeze-dried (lyophilized), and all water content removed prior to chemical analysis of bioactives.
- L.S.I. or 'Lack of Substantial Information' in scientific literature.
- N.P. or 'Not Present', or 'Trace' indicates absence of the bioactive actively confirmed in a scientific publication.
- 'Range of mean values', from multiple publications.
- Total phenolics reported in gallic acid equivalents (GAE) or chlorogenic acid equivalents (CGA).
- Reported alongside either isorhamnetin-3-O-rutinoside or isorhamnetin-3-O-glucoside in some instances.

HEALTH CLAIMS FOR MAJOR BIOACTIVES

The evidence of health claims for major bioactives in this BMF will be separated into three categories:



Multiple human clinical studies & observational studies, with meta-analyses & systematic reviews



Individual human clinical studies and/or animal-model studies available



Only laboratory 'in vitro' or 'in vivo' cell-line studies available



BIOACTIVE FOCUS: Almond



CLAIM [Lipid Profile, *N* = 36 Randomised controlled trial arms, Meta-analysis] [Asbighi et al., 2021]: Almond intake significantly reduced triglycerides (TC) (MD = -6.68 mg/dL, *p* = 0.008, *I*² = 64.9%), Total Cholesterol (TC) (WMD = -4.92 mg/dL, *p* = 0.001, *I*² = 53.9%), and LDL-C (WMD = -5.65 mg/dL, *p* < 0.001, *I*² = 82.5%). No significant effect was observed for HDL-C (WMD = -0.21 mg/dL, *p* = 0.697, *I*² = 48.2%). Sub-group analyses showed greater TG reduction in participants <50 years (WMD = -14.18 mg/dL, *p* = 0.017, *I*² = 80.9%), with almond doses ≥45 g/d (WMD = -10.91 mg/dL, *p* = 0.031, *I*² = 72.9%), and in healthy individuals (WMD = -10.92 mg/dL, *p* = 0.001, *I*² = 75.0%). TC reduction was more pronounced in participants <50 years (WMD = -8.42 mg/dL, *p* < 0.001, *I*² = 41.2%) and with doses ≥45 g/d (WMD = -8.41 mg/dL, *p* < 0.001, *I*² = 36.2%). LDL-C reduction was significant in participants with elevated baseline LDL-C (WMD = -8.73 mg/dL, *p* < 0.001, *I*² = 62.0%) and with trial durations ≥6 weeks (WMD = -6.28 mg/dL, *p* = 0.007, *I*² = 88.0%). Heterogeneity reported individually for each measurement, with moderate to high heterogeneity for TG, TC, and LDL-C (*I*² = 53.9–82.5%) and moderate heterogeneity for HDL-C (*I*² = 48.2%).

EFFECTIVE DOSAGE [Asbighi et al., 2021]: Almond doses ranged from 10 g/d to 168 g/d, with intervention durations from 3 to 77 weeks. Sub-group findings suggest ≥45 g/d for ≥6 weeks may optimize reductions in TG, TC, and LDL-C. No dose-response threshold was established for HDL-C.

CLAIM [Blood Pressure, *N* = 23 Randomised controlled trial arms, Meta-analysis] [Eslampour, Asbighi et al., 2020]: Almond intake significantly reduced diastolic blood pressure (WMD = -1.30 mmHg, *p* = 0.01, *I*² = 0%), while no significant effect was observed for systolic blood pressure (WMD = -0.83 mmHg, *p* = 0.34, *I*² = 58.9%). Sub-group analyses showed greater SBP reduction in participants with baseline SBP <120 mmHg (WMD = -2.01 mmHg, *p* < 0.001, *I*² = 86.7%), with almond doses <45 g/d (WMD = -1.83 mmHg, *p* < 0.001, *I*² = 66.7%), and in healthy individuals (WMD = -1.76 mmHg, *p* < 0.001, *I*² = 83.3%). SBP reduction was also more pronounced in participants <50 years (WMD = -1.74 mmHg, *p* < 0.001, *I*² = 89.1%) and with trial durations <6 weeks (WMD = -1.84 mmHg, *p* < 0.001, *I*² = 72.2%). Heterogeneity reported individually for each measurement, with negligible heterogeneity for DBP (*I*² = 0%) and moderate to high heterogeneity for SBP (*I*² = 58.9–89.1%).

EFFECTIVE DOSAGE [Eslampour, Asbighi et al., 2020]: Almond doses ranged from 10 g/d to 73 g/d, with intervention durations from 3 to 77.4 weeks. Sub-group findings suggest <45 g/d for <6 weeks may optimize SBP reduction in healthy adults with lower baseline SBP. No dose-response threshold was established for DBP.

CLAIM [Anthropometric Indices (Body Weight, BMI, Waist Circumference, Fat Mass, Fat-Free Mass), *N* = 37 Randomised controlled trial arms, Meta-analysis] [Eslampour, Moodi et al., 2020]: Almond intake significantly reduced body weight (WMD = -0.38 kg, *p* = 0.007, *I*² = 30.5%) and fat mass (WMD = -0.58 kg, *p* < 0.001, *I*² = 4.9%). No significant effect was observed for BMI (WMD = -0.30 kg/m², *p* = 0.101, *I*² = 62.6%), waist circumference (WMD = -0.60 cm, *p* = 0.078, *I*² = 0.0%), or fat-free mass (WMD = 0.23 kg, *p* = 0.097, *I*² = 49.5%). Sub-group analyses showed greater body weight reduction in trials ≥6 weeks (WMD = -0.38, *p* = 0.006, *I*² = 58.9%), with almond doses ≥45 g/d (WMD = -0.31, *p* = 0.049, *I*² = 62.6%), and in healthy (WMD = -0.50, *p* = 0.005, *I*² = 66.0%) or overweight participants (WMD = -1.82, *p* = 0.001, *I*² = 0.0%). Fat mass reduction was more pronounced in trials ≥6 weeks (WMD = -0.58, *p* < 0.001, *I*² = 38.8%), with doses ≥45 g/d (WMD = -0.58, *p* < 0.001, *I*² = 56.2%), and in healthy (WMD = -0.60, *p* < 0.001, *I*² = 47.4%) or normal BMI participants (WMD = -0.57, *p* = 0.001, *I*² = 0.0%). BMI reduction was significant only in trials ≥6 weeks (WMD = -0.16, *p* = 0.013, *I*² = 76.1%), doses ≥45 g/d (WMD = -0.17, *p* = 0.009, *I*² = 79.5%), and in older (≥50 years) or overweight participants. Heterogeneity reported individually for each measurement, with low heterogeneity for fat mass (*I*² = 4.9%) and waist circumference (*I*² = 0.0%), and moderate to high heterogeneity for BMI and fat-free mass (*I*² = 49.5–62.6%).

EFFECTIVE DOSAGE [Eslampour, Moodi et al., 2020]: Almond doses ranged from 10 g/d to 100 g/d, with intervention durations from 3 to 77.14 weeks. Sub-group findings suggest ≥45 g/d for ≥6 weeks may optimize reductions in body weight and fat mass. No dose-response threshold was established for BMI, waist circumference, or fat-free mass.

CLAIM [Anti-inflammation, *N* = 18 Randomised controlled trial arms, Meta-analysis] [Fatahi et al., 2021]: Almond intake significantly reduced C-reactive protein (CRP) (WMD = -0.25 mg/L, *p* = 0.009, *I*² = 0%) and interleukin-6 (IL-6) (WMD = -0.11 pg/mL, *p* = 0.029, *I*² = 19.9%). No significant effect was observed for tumor necrosis factor-alpha (TNF-α) (WMD = -0.05 pg/mL, *p* = 0.09, *I*² = 0%), intercellular adhesion molecule-1 (ICAM-1) (WMD = 6.39 ng/mL, *p* = 0.429, *I*² = 66.6%), or vascular cell adhesion molecule-1 (VCAM-1) (WMD = -8.31 ng/mL, *p* = 0.547, *I*² = 58.8%). Sub-group analyses showed greater CRP reduction at almond doses <60 g/d (WMD = -0.40, *p* = 0.259, *I*² = 20.7%), in nonobese participants (WMD = -0.32, *p* = 0.653, *I*² = 0%), and in healthy individuals (WMD = -0.27, *p* = 0.566, *I*² = 0%). IL-6 reduction was more pronounced in adults ≥50 years (WMD = -0.20, *p* = 0.397, *I*² = 3.9%), in nonobese participants (WMD = -0.14, *p* = 0.137, *I*² = 42.7%), and in those with normal blood glucose (WMD = -0.06, *p* = 0.602, *I*² = 0%). Heterogeneity reported individually for each measurement, with low heterogeneity for CRP and IL-6 (*I*² ≤ 19.9%) and moderate to high heterogeneity for ICAM-1 and VCAM-1 (*I*² = 58.8–66.6%).

EFFECTIVE DOSAGE [Fatahi et al., 2021]: Almond doses ranged from 41 g/d to 100 g/d, with intervention durations from 4 to 24 weeks. Sub-group findings suggest <60 g/d may optimize reductions in CRP and IL-6, particularly in healthy and nonobese adults. No dose-response threshold was established for TNF-α, ICAM-1, or VCAM-1.

CLAIM [Anti-inflammation, *N* = 25 Randomised controlled trial arms, Meta-analysis] [Hariri et al., 2023]: Almond intake significantly reduced interleukin-6 (IL-6) (WMD = -0.10 pg/mL, *p* < 0.001, *I*² = 77.0%), while no significant effect was observed for C-reactive protein (CRP) (WMD = 0.28 mg/L, *p* = 0.29, *I*² = 86.1%). Sub-group analyses showed greater CRP reduction in trials with crossover design (WMD = -0.85, *p* = 0.007, *I*² = 90.0%), intervention duration <84 days (WMD = -0.77, *p* = 0.016, *I*² = 91.5%), sample size ≥52 participants (WMD = -0.62, *p* = 0.046, *I*² = 85.3%), age <58 years (WMD = -0.90, *p* = 0.027, *I*² = 86.1%), and studies conducted in Europe (WMD = -1.64, *p* = 0.013, *I*² = 79.4%). IL-6 reduction was more pronounced in trials with parallel design (WMD = -0.13, *p* < 0.001, *I*² = 77.6%), almond dose ≥56 g/d (WMD = -0.08, *p* = 0.001, *I*² = 76.1%), intervention duration ≥84 days (WMD = -0.16, *p* = 0.001, *I*² = 83.2%), healthy participants (WMD = -0.13, *p* < 0.001, *I*² = 81.3%), and age ≥58 years (WMD = -0.22, *p* = 0.003, *I*² = 74.5%). Heterogeneity reported individually for each measurement, with high heterogeneity for CRP (*I*² = 86.1%) and IL-6 (*I*² = 77.0%), and moderate heterogeneity in select sub-groups (*I*² = 33.4–50.1%).

EFFECTIVE DOSAGE [Hariri et al., 2023]: Almond doses ranged from 42 g/d to 85 g/d, with intervention durations from 8 to 28 weeks. Sub-group findings suggest ≥56 g/d for ≥84 days may optimize IL-6 reduction, while CRP reduction was more evident in shorter trials (<84 days) and crossover designs. No dose-response threshold was established for either biomarker.

CLAIM [Anti-oxidation, *N* = 8 Randomised controlled trial arms and crossover trials, Meta-analysis] [Kolahi et al., 2025]: Almond supplementation significantly reduced 8-hydroxy-2'-deoxyguanosine (8-OHdG) (WMD = -5.83 ng/mL, *p* < 0.001, *I*² = 0%) and uric acid (UA) (WMD = -0.64 mg/dL, *p* = 0.009, *I*² = 55%), and significantly increased superoxide dismutase (SOD) activity (WMD = 2.02 U/mL, *p* = 0.008, *I*² = 92%). No significant effect was observed for glutathione peroxidase (GPx) (WMD = 13.04 U/L, *p* = 0.270, *I*² = 96%) or malondialdehyde (MDA) overall (WMD = -0.11 μmol/L, *p* = 0.141, *I*² = 42.8%). Sub-group analyses showed significant MDA reduction only at almond doses >60 g/d (WMD = -0.46 μmol/L, *p* = 0.002, *I*² = 0%), while lower doses showed no effect (WMD = -0.03 μmol/L, *p* = 0.372, *I*² = 0%). SOD improvement was also dose-dependent, with greater effects observed at higher doses. Heterogeneity reported individually for each measurement, with negligible heterogeneity for 8-OHdG (*I*² = 0%), moderate heterogeneity for UA and MDA (*I*² = 42.8–55%), and high heterogeneity for SOD and GPx (*I*² = 92–96%).

EFFECTIVE DOSAGE [Kolahi et al., 2025]: Almond doses ranged from 5 g/d to 168 g/d, with intervention durations from 4 to 24 weeks. Sub-group findings suggest >60 g/d may optimize reductions in MDA, 8-OHdG, and UA, and enhance SOD activity. No dose-response threshold was established for GPx.



BIOACTIVE FOCUS: Almond

CLAIM [Anti-oxidation, $N = 7$ Randomised controlled trial arms, Meta-analysis] [Luo *et al.*, 2023]: Almond intake significantly reduced malondialdehyde (MDA) levels (WMD = -6.63 nmol/mL, $p < 0.001$, $I^2 = 0\%$). No significant effect was observed for oxidized low-density lipoprotein (Ox-LDL) (SMD = 0.12 , $p = 0.28$, $I^2 = 0\%$). Sub-group analyses were not reported. Sensitivity analyses showed that removal of individual studies did not alter the overall findings. Heterogeneity reported individually for each measurement, with negligible heterogeneity for both MDA and Ox-LDL ($I^2 = 0\%$).

EFFECTIVE DOSAGE [Luo *et al.*, 2023]: Almond doses ranged from 30 g/d to 84 g/d, with intervention durations from 4 to 28 weeks. Significant MDA reduction was observed across this range. No dose-response threshold was established for Ox-LDL.

CLAIM [Cardiovascular Disease Risk, $N = 15$ Randomised controlled trial arms, Meta-analysis] [Lee-Bravatti *et al.*, 2019]: Almond intake significantly reduced total cholesterol (TC) (WMD = -10.69 mg/dL, $p < 0.001$, $I^2 = 67\%$), LDL-C (WMD = -5.83 mg/dL, $p < 0.001$, $I^2 = 61\%$), body weight (WMD = -1.39 kg, $p = 0.007$, $I^2 = 0\%$), and apolipoprotein B (WMD = -6.67 mg/dL, $p = 0.03$, $I^2 = 50\%$). HDL-C was also significantly reduced (WMD = -1.26 mg/dL, $p = 0.04$, $I^2 = 16\%$), while triglycerides (TG) showed no significant change (WMD = -11.63 mg/dL, $p = 0.06$, $I^2 = 71\%$). Sub-group analyses showed greater TC reduction with almond doses >42.5 g/d (WMD = -15.83 , $p < 0.001$, $I^2 = 80\%$) and with intervention durations ≤ 6 weeks (WMD = -7.18 , $p < 0.001$, $I^2 = 29\%$). LDL-C reduction was more pronounced in participants at risk of cardiovascular disease (WMD = -10.46 , $p < 0.001$, $I^2 = 61\%$) and with doses ≥ 42.5 g/d (WMD = -6.67 , $p = 0.05$, $I^2 = 50\%$). Body weight reduction was significant across all sub-groups ($I^2 = 0\%$). Heterogeneity reported individually for each measurement, with low heterogeneity for body weight and HDL-C ($I^2 \leq 16\%$) and moderate to high heterogeneity for TC, LDL-C, TG, and ApoB ($I^2 = 50\text{--}71\%$).

EFFECTIVE DOSAGE [Lee-Bravatti *et al.*, 2019]: Almond doses ranged from 25 g/d to 100 g/d, with intervention durations from 4 to 16 weeks. Sub-group findings suggest >42.5 g/d for ≤ 6 weeks may optimize reductions in TC, LDL-C, and body weight. No dose-response threshold was established for triglycerides or HDL-C.

CLAIM [Cardiometabolic Parameters in Type 2 Diabetes, $N = 9$ Randomised controlled trial arms and crossover trials, Meta-analysis] [Moosavian *et al.*, 2021]: Almond intake significantly reduced LDL-C (WMD = -5.28 mg/dL, $p = 0.026$, $I^2 = 9.2\%$). No significant effects were observed for triglycerides (TG) (WMD = 0.24 mg/dL, $p = 0.966$, $I^2 = 7.3\%$), total cholesterol (TC) (WMD = 1.70 mg/dL, $p = 0.556$, $I^2 = 17.2\%$), HDL-C (WMD = 0.12 mg/dL, $p = 0.871$, $I^2 = 0.0\%$), fasting plasma glucose (FPG) (WMD = 0.42 mg/dL, $p = 0.799$, $I^2 = 0.0\%$), insulin (WMD = 0.26 μ U/mL, $p = 0.749$, $I^2 = 0.0\%$), and HbA1c (WMD = 0.06% , $p = 0.362$, $I^2 = 2.7\%$). No significant changes were found for body mass index (BMI) (WMD = 0.02 kg/m², $p = 0.953$, $I^2 = 0.0\%$), body weight (WMD = 0.08 kg, $p = 0.818$, $I^2 = 0.0\%$), or body fat (WMD = 1.06% , $p = 0.362$, $I^2 = 0.0\%$). Systolic blood pressure (SBP) (WMD = 0.83 mmHg, $p = 0.749$, $I^2 = 0.0\%$) and diastolic blood pressure (DBP) (WMD = 1.20 mmHg, $p = 0.510$, $I^2 = 0.0\%$) were also unaffected. C-reactive protein (CRP) showed no overall change (WMD = 0.26 mg/dL, $p = 0.125$, $I^2 = 92.4\%$), although sub-group analysis indicated a significant reduction with almond intake ≤ 50 g/d (WMD = -0.55 mg/dL, $p < 0.05$, I^2 not reported). Sub-group analyses showed greater LDL-C reduction with almond doses >50 g/d (WMD = -7.12 mg/dL, $p < 0.05$, I^2 not reported) and baseline LDL-C <130 mg/dL (WMD = -5.27 mg/dL, $p < 0.05$, I^2 not reported). Heterogeneity reported individually for each measurement, with low heterogeneity for most outcomes ($I^2 = 0.0\text{--}17.2\%$) and high heterogeneity for CRP ($I^2 = 92.4\%$).

EFFECTIVE DOSAGE [Moosavian *et al.*, 2021]: Almond doses ranged from 29 g/d to 113 g/d, with intervention durations from 3 to 12 weeks. Sub-group findings suggest >50 g/d may optimize reductions in LDL-C and CRP. No dose-response threshold was established for other cardiometabolic outcomes.

CLAIM [Cardiometabolic Health, $N = 26$ Randomised controlled trial arms and crossover trials, Meta-analysis] [Morvaridzadeh *et al.*, 2022]: Almond intake significantly reduced total cholesterol (TC) (SMD = -0.29 , $p < 0.001$, $I^2 = 33.5\%$), triglycerides (TG) (SMD = -0.23 , $p < 0.001$, $I^2 = 14.6\%$), LDL-C (SMD = -0.29 , $p < 0.001$, $I^2 = 20.8\%$), non-HDL-C (SMD = -0.36 , $p < 0.001$, $I^2 = 0.0\%$), and very-low-density lipoprotein (VLDL) (SMD = -0.23 , $p < 0.001$, $I^2 = 0.0\%$). Diastolic blood pressure (DBP) was also significantly reduced (SMD = -0.17 , $p < 0.001$, $I^2 = 0.0\%$). No significant effects were observed for systolic blood pressure (SBP) (SMD = -0.06 , $p = 0.127$, $I^2 = 28.3\%$), HDL-C (SMD = 0.01 , $p = 0.803$, $I^2 = 39.9\%$), fasting blood glucose (SMD = 0.02 , $p = 0.799$, $I^2 = 71.9\%$), insulin (SMD = 0.19 , $p = 0.008$, $I^2 = 56.8\%$), HbA1c (SMD = -0.10 , $p = 0.362$, $I^2 = 0.0\%$), HOMA-IR (SMD = 0.07 , $p = 0.161$, $I^2 = 36.9\%$), C-peptide (SMD = 1.42 , $p = 0.000$, $I^2 = 88.1\%$), and hepatic enzymes (ALT, AST, GGT; SMDs = -0.16 to 0.02 , $p > 0.05$, $I^2 = 0.7\text{--}43.4\%$). Inflammatory markers including CRP, hs-CRP, IL-6, TNF- α , ICAM, VCAM, and homocysteine showed no significant changes overall. Sub-group analyses showed greater reductions in TC, TG, LDL-C, non-HDL-C, and VLDL with almond doses ≥ 50 g/d and durations ≥ 10 weeks. DBP reduction was more pronounced in unhealthy individuals, those with baseline DBP >75 mmHg, and with almond doses >50 g/d. IL-6 was significantly reduced in healthy participants, young adults, and with almond doses ≥ 50 g/d. Heterogeneity reported individually for each

measurement, with low to moderate heterogeneity for most lipid and blood pressure outcomes ($I^2 = 0.0\text{--}33.5\%$) and high heterogeneity for insulin, C-peptide, and IL-6 ($I^2 = 56.8\text{--}94.9\%$).

EFFECTIVE DOSAGE [Morvaridzadeh *et al.*, 2022]: Almond doses ranged from 5 g/d to 85 g/d, with intervention durations from 4 to 20 weeks. Sub-group findings suggest ≥ 50 g/d for ≥ 10 weeks may optimize reductions in TC, TG, LDL-C, non-HDL-C, VLDL, DBP, and IL-6. No dose-response threshold was established for HDL-C, glycemic markers, or hepatic enzymes.

CLAIM [Obesity & Metabolic Syndrome, $N = 43$ Randomised controlled trial arms, Meta-analysis] [Chahibakhsh *et al.*, 2024]: Almond supplementation significantly reduced body weight (WMD = -0.45 kg, $p = 0.026$, $I^2 = 33.7\%$), waist circumference (WC) (WMD = -0.66 cm, $p = 0.037$, $I^2 = 0.0\%$), fat mass (FM) (WMD = -0.66 kg, $p = 0.009$, $I^2 = 22.3\%$), and hunger score (WMD = -1.15 mm, $p = 0.006$, $I^2 = 0.0\%$). No significant effects were observed for body mass index (BMI) (WMD = -0.20 kg/m², $p = 0.122$, $I^2 = 50.9\%$), body fat percent (WMD = -0.39% , $p = 0.154$, $I^2 = 81.9\%$), fat-free mass (FFM) (WMD = -0.07 kg, $p = 0.741$, $I^2 = 85.1\%$), waist-to-hip ratio (WHR) (WMD = -0.04 , $p = 0.203$, $I^2 = 93.9\%$), visceral adipose tissue (VAT) (WMD = -0.33 cm, $p = 0.323$, $I^2 = 79.5\%$), fullness (WMD = 0.46 mm, $p = 0.517$, $I^2 = 0.0\%$), desire to eat (WMD = 0.98 mm, $p = 0.558$, $I^2 = 0.0\%$), and prospective food consumption (WMD = 1.08 mm, $p = 0.505$, $I^2 = 0.0\%$). Sub-group analyses showed greater reductions in body weight (WMD = -0.63 kg, $p = 0.027$, $I^2 = 60.2\%$), WC (WMD = -3.65 cm, $p < 0.001$, $I^2 = 84.6\%$), FM (WMD = -0.98 kg, $p = 0.024$, $I^2 = 49.4\%$), and hunger score (WMD = -1.15 mm, $p = 0.006$, $I^2 = 0.0\%$) with almond doses ≥ 50 g/d. Longer intervention durations (≥ 12 weeks) also enhanced reductions in body weight (WMD = -0.66 kg, $p = 0.040$, $I^2 = 64.7\%$), WC (WMD = -5.77 cm, $p < 0.001$, $I^2 = 74.7\%$), FM (WMD = -0.87 kg, $p = 0.031$, $I^2 = 68.5\%$), and body fat percent (WMD = -0.74% , $p = 0.020$, $I^2 = 0.0\%$). Heterogeneity reported individually for each measurement, with low to moderate heterogeneity for body weight, WC, FM, and hunger ($I^2 = 0.0\text{--}49.4\%$) and high heterogeneity for BMI, body fat percent, FFM, WHR, and VAT ($I^2 = 50.9\text{--}93.9\%$).

EFFECTIVE DOSAGE [Chahibakhsh *et al.*, 2024]: Almond doses ranged from 10 g/d to 100 g/d, with intervention durations from 3 weeks to 18 months. Sub-group findings suggest ≥ 50 g/d for ≥ 12 weeks may optimize reductions in body weight, WC, FM, and hunger. No dose-response threshold was established for BMI, FFM, WHR, VAT, or other appetite scores.



BIOACTIVE FOCUS: Oleic Acid



CLAIM [Anti-inflammation, $N = 31$ Randomised controlled trial arms, Meta-analysis] [Wang *et al.*, 2022]: Oleic acid supplementation significantly reduced C-reactive protein (CRP) (SMD = 0.11 , $p = 0.038$, $I^2 = 7.9\%$). No significant effect was observed for TNF- α (SMD = 0.05 , $p = 0.534$, $I^2 = 0\%$), IL-6 (SMD = 0.01 , $p = 0.849$, $I^2 = 0\%$), fibrinogen (SMD = 0.08 , $p = 0.520$, $I^2 = 0\%$), PAI-1 activity (SMD = 0.11 , $p = 0.355$, $I^2 = 0\%$), sICAM-1 (SMD = 0.06 , $p = 0.595$, $I^2 = 0\%$), and sVCAM-1 (SMD = 0.04 , $p = 0.701$, $I^2 = 46.1\%$). Sub-group analyses showed greater CRP reduction in participants <50 years (SMD = 0.13 , $p < 0.05$, $I^2 = 8.1\%$), with intervention durations ≤ 12 weeks (SMD = 0.14 , $p < 0.05$, $I^2 = 1.4\%$), and in individuals with BMI <30 (SMD = 0.12 , $p < 0.05$, $I^2 = 3.3\%$). CRP reduction was also more pronounced in healthy participants (SMD = 0.21 , $p < 0.05$, $I^2 = 22.8\%$) and those with baseline CRP above the median (SMD = 0.19 , $p < 0.05$, $I^2 = 14.1\%$). Heterogeneity reported individually for each measurement, with low heterogeneity for most outcomes ($I^2 = 0\text{--}7.9\%$) and moderate heterogeneity for sVCAM-1 ($I^2 = 46.1\%$).

EFFECTIVE DOSAGE [Wang *et al.*, 2022]: Oleic acid doses ranged from 1.5 g/d to 37.1 g/d. Sub-group findings suggest interventions ≤ 12 weeks and doses providing a substantial difference in oleic acid intake compared to control may optimize CRP reduction. No dose-response threshold was established for TNF, IL-6, or adhesion molecules. Intervention time-range 4 to 24 weeks.

CLAIM [Metabolic Syndrome (MetS), $N = 67$ Randomised controlled trial arms, Meta-analysis] [Pastor, Bouzas & Tur, 2021]: Oleic acid supplementation showed no significant effect on overall MetS (SMD = 0.03 , $p = 0.150$, $I^2 = 0\%$). However, lipid profile improved modestly (SMD = 0.06 , $p = 0.050$, $I^2 = 0\%$). No significant effects were observed for body composition (SMD = 0.06 , $p = 0.300$, $I^2 = 34\%$), glycaemic profile (SMD = 0.04 , $p = 0.590$, $I^2 = 12\%$), or blood pressure (SMD = 0.02 , $p = 0.800$, $I^2 = 0\%$). Low heterogeneity for lipid profile ($I^2 = 0\%$), moderate for body composition ($I^2 = 34\%$), and negligible for other outcomes.

EFFECTIVE DOSAGE [Pastor, Bouzas & Tur, 2021]: Oleic acid provided via high-oleic oils or enriched diets. No clear dose-response threshold was established for MetS components. Intervention time-range 2 weeks to 6 months.



BIOACTIVE FOCUS: Oleic Acid

CLAIM [Gastrointestinal Function, $N = 14$ Adults, Randomised Double-Blind Crossover Trial] [Al-Humadi et al., 2024]: Microencapsulated oleic acid delivered to the distal small intestine significantly increased the number of bowel movements within 24 h compared to gastric delivery (2.5 vs 1.1 movements/day, $p = 0.009$) and improved stool consistency (Bristol Stool Scale: 4.0 vs 2.3 at 24 h, $p = 0.03$). A higher proportion of participants passed a bowel movement within 24 h in the distal release group (92.8% vs 64.3%, $p = 0.05$). No significant differences were observed for straining ($p = 0.65$), urgency ($p = 0.08$), accidental leakage ($p = 0.32$), hunger, fullness, nausea, or calorie intake (all $p > 0.05$). Mild, transient gastrointestinal symptoms (stomachache, nausea, burping, flatulence) were reported, with no severe adverse events.

EFFECTIVE DOSAGE [Al-Humadi et al., 2024]: A single dose of 21.25 g oleic acid (~400 kcal) encapsulated for distal small intestine release was tested against gastric release. Intervention duration was acute (24 h follow-up). Findings suggest targeted delivery to the distal small intestine optimizes laxative effect without impacting appetite or calorie intake. Long-term safety and efficacy require further investigation.



INTERESTING FACTS

Almond Hull for Biofuels Feedstock:

Almond hulls contain high levels of fermentable sugars (25–33% dry basis), primarily sucrose, glucose, and fructose, making them a promising feedstock for biofuel production [Offerman et al., 2014]. Among the six major California varieties studied, *Nonpareil* showed the highest sugar content, yielding up to 271 kg fermentable sugars per tonne of “as is” hulls and a theoretical ethanol output of about 176 L/tonne [Offerman et al., 2014]. Other varieties produced lower yields (130–148 L/tonne), with shell content significantly affecting sugar concentration [Offerman et al., 2014]. Industrial-scale processing would require hot water extraction and syrup concentration for fermentation, but the spent hulls retain ~93% moisture, posing challenges for drying and economic utilisation [Offerman et al., 2014].

Almond Hull as Mulch for Avocado Cultivation:

Applying almond shells as mulch in organic avocado orchards created a new organic soil horizon and more than doubled soil organic carbon in the top 25 cm compared to conventional management [López et al., 2014]. Despite slow decomposition due to high lignin content, nutrient mineralization was sufficient to maintain or even improve avocado yields without synthetic fertilizers [López et al., 2014]. Mulching significantly enhanced soil enzyme activities and microbial activity, indicating improved biological quality, while also increasing phosphorus and nitrogen availability [López et al., 2014]. These findings highlight almond shells as an effective, sustainable mulch that improves soil health, supports carbon sequestration, and reduces input costs in avocado production [López et al., 2014].

Almond Bagasse for Food Functionalisation:

Almond bagasse (almond by-processing waste pulp) is a rich source of phenolics such as epicatechin, vanillic acid, and rutin, which contribute to strong antioxidant properties [Duarte et al., 2023]. Dehydration methods (hot air drying and lyophilization) reduce phenolic content and antiradical capacity, though lyophilization generally preserves more bioactives than hot air drying [Duarte et al., 2023]. *In vitro* digestion and colonic fermentation enhance the release of phenolics and restore antioxidant activity, while fermentation promotes beneficial short-chain fatty acid producing bacteria like *Butyrivibrio* and *Ruminococcus* [Duarte et al., 2023]. These findings indicate stabilised almond bagasse powders as sustainable functional ingredients for improving gut health and antioxidant intake in food formulations [Duarte et al., 2023].



INTERESTING FACTS

Bitter Almond?

Bitter almonds differ from sweet almonds mainly in their high content of the cyanogenic diglucoside amygdalin, which releases toxic hydrogen cyanide upon hydrolysis, whereas sweet almonds contain negligible amounts [Sánchez-Pérez et al., 2008]. This difference is genetically controlled by a single recessive gene and is linked to the presence or absence of β -glucosidase activity in the tegument: sweet almonds degrade prunasin before it reaches the cotyledon, preventing amygdalin formation [Sánchez-Pérez et al., 2008]. Bitter almonds accumulate prunasin in the tegument and convert it into amygdalin in the cotyledons during development, serving as a defence mechanism against herbivores [Sánchez-Pérez et al., 2008].

'Bitter' Almond Toxins as a Nematocidal:

Temiz, 2025, reported that ultrasound-assisted extraction (BAU) of 'bitter' almond by-products yielded significantly higher amygdalin content (1213 mg per 100 g of extract) compared to conventional extraction (BAC) (974 mg per 100 g of extract) ($p < 0.05$) [Temiz et al., 2025]. Amygdalin is a highly toxic compound, making bitter almonds unsafe for human consumption, due to its conversion to poisonous cyanide when ingested [TGA – Amygdalin, 2020]. The Therapeutic Goods Administration (TGA) reported that the lethal dose of amygdalin in adults is estimated to be 9.0 – 60.0 mg/kg body weight (540.0 – 3600.0 mg for a 60 kg adult) [TGA – Amygdalin, 2020].

Both extracts demonstrated dose-dependent nematocidal activity against the pathogenic nematode *Ditylenchus dipsaci*, that often infects onion and garlic plants, with UAE bitter almond extracts achieving 90.38% inhibition at 1 mg/mL, outperforming the 'Fenamiphos' organophosphate nematocide (61.97% inhibition) ($p < 0.05$) [Temiz et al., 2025]. At a 2 mg/mL dose, BAU and BAC exceeded 93% inhibition, while authentic amygdalin alone exhibited limited efficacy (26.83%) [Temiz et al., 2025]. EC_{50} values indicated BAU was about twice as potent as BAC (3.81 vs 7.64 mg/mL). These results suggest nematocidal activity is driven by synergistic effects of amygdalin and phenolics rather than amygdalin alone, highlighting bitter almond extracts as promising eco-friendly bio-nematicides for integrated pest management [Temiz et al., 2025].

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