Calcium

bw	body weight
CNS	Chinese Nutrition Society
CRN	Council for Responsible Nutrition
DRI	dietary reference intake
EC SCF	European Commission Scientific Committee on Food
EFSA	European Food Safety Authority
EVM	Expert Group on Vitamins and Minerals
ICMR-NIN	Indian Council of Medical Research - National Institute of Nutrition
IOM	Institute of Medicine
IU	international unit
JECFA	Joint FAO/WHO Expert Committee on Food Additives
KNS	Korean Nutrition Society
LOAEL	lowest observed adverse effect level
LOEL	lowest observed effect level
NDA	EFSA Panel on Nutrition, Novel Foods and Food Allergens
NIH	National Institute of Health
NOAEL	no observed adverse effect level
NOEL	no observed effect level
RCT	randomized clinical trial
SUL	safe upper level
UF	uncertainty factor
UL	tolerable upper intake level

Introduction

Calcium is a nutrient most often associated with the formation, metabolism, strength, and health of bones and teeth (IOM 2011). Over 99 percent of calcium in the body resides in bones and teeth as calcium hydroxyapatite (IOM 2011; EFSA 2012; EFSA 2015). This mineral also plays a significant role as a second messenger in cell-signaling pathways, including the regulation of vascular contraction and dilation, muscle function, nerve transmission, intracellular signaling, and hormonal secretion functions (NIH 2025; Delage 2017; SCF 2003). Additionally, calcium

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has a role in protein regulation through protein stabilization and enhancing enzymatic activity (Delage 2017). In its structural roles, calcium has a substantial impact on presence or absence of osteoporosis. Calcium absorption and utilization may be dependent on and influenced by dietary intakes of phosphorus and vitamin D, as well as other factors such as parathyroid hormone, the peptide calcitonin, and estrogen (IOM 2011). Calcium homeostasis is maintained by hormones, such as parathyroid hormone, hydroxylated vitamin D3 and calcitonin, and these hormones help regulate calcium transport in the intestines, kidneys, and bones (Yu and Sharma 2023).

The role of dietary calcium and vitamin D in reducing the risk or delaying the onset of osteoporosis is now well recognized (FDA 1994). Because bone loss often accompanies the aging process, sufficient calcium intake during early adulthood increases peak bone mass, thereby reducing the risk of osteoporosis decades later (Heaney et al. 2000). Increases in calcium intake in postmenopausal women delay calcium loss from bone, thus lowering the risk of declines in bone mineral density to osteoporotic levels. Calcium intakes of 1,000 to 2,000 mg per day have been shown to increase or slow the decline in bone density and to reduce the risk of osteoporosis (FDA 1994; Amarnath et al. 2023).

The main dietary sources of calcium are dairy products, as well as dark green vegetables, legumes, nuts, fish with soft bones, calcium-fortified foods, and hard water (EFSA 2015). The most frequently utilized forms of calcium in dietary supplements are calcium carbonate and calcium citrate; other forms include calcium sulfate, ascorbate, gluconate, lactate, phosphate, and a microcrystalline hydroxyapatite (NIH 2025).

The current chapter focuses on the derivation of an UL value for supplemental calcium in adults.

Bioavailability

Calcium absorption occurs via both active transport mechanisms and passive diffusion across the intestinal mucosa; both processes are regulated by dietary intake concentrations (EFSA 2012, 2015). The source of calcium, whether consumed from food or dietary supplements, does not influence nor alter the absorption process (EFSA 2012; NIH 2025). Calcium absorption is influenced by vitamin D status, solubility of the calcium salt form, and whether the calcium is

bound to soluble organic molecules (EFSA 2012, 2015).

During active transport, vitamin D facilitates intestinal calcium absorption and maintains adequate calcium levels in the blood (NIH 2025). The entry of calcium into the enterocyte is controlled by a hydroxylated form of vitamin D (25-hydroxy-calciferol). Calcium is transported to the interior of the enterocyte by a calcium-binding protein and extruded from the basolateral membrane, against the concentration gradient, by an intestinal plasma pump. Both transporter proteins are controlled by the presence of vitamin D. The active transport mechanism of calcium can be saturated (EFSA 2012, 2015). Conversely, passive diffusion is a non-saturable, paracellular process taking place throughout the entire length of the intestine where transport occurs at tight junctions and structures within intercellular spaces (EFSA 2012, 2015). Passive diffusion utilizes the electrochemical gradient involving water, sodium, and glucose (SCF 2003).

Active transport is the primary route of calcium absorption and passive diffusion is responsible for 8 to 23% of total calcium absorption in healthy adults (EFSA 2015; McCormick 2002). The fractional absorption of calcium is highest in infants (~60%) and gradually decreases with age in adulthood (Amarnath et al. 2023). Calcium has been shown to be absorbed in the ileum and duodenum, though it can also be absorbed by the colon via passive absorption (EFSA 2015; Amarnath et al. 2023). Calcium is distributed to bone tissue and is stored in the skeleton and teeth (ESFA 2015). Data indicate that 50% of ingested calcium is excreted in feces, 22% in urine, and trace amounts are detected in sweat, skin, and hair (SCF 2003; Amarnath et al. 2023). Unabsorbed calcium is bound to bile acids, free fatty acids, or oxalic acid and excreted in feces (SCF 2003).

Safety Considerations

Calcium from dietary supplement use has been associated with gastrointestinal side effects in some individuals (Delage 2017; NIH 2025). These symptoms — gas, bloating, constipation, diarrhea, and/or cramping — are considered not to be *true hazards* but instead *nuisance effects*, as defined by CRN's methodology (see *Methodology* chapter). In addition, such effects have been shown to be avoided by taking calcium supplements in smaller doses and/or with meals, and by switching to a different form of calcium (NIH 2025).

Several potential adverse effects (i.e., *true hazards*) have been considered regarding supplemental calcium intake, as discussed below.

Cardiovascular Effects

The cardiovascular safety of supplemental calcium has been questioned, primarily based on the findings of a handful of studies. As discussed below, because of significant limitations in design or interpretation, these reports do not provide strong evidence of harmful cardiovascular effects of calcium supplementation. Considering the totality of available evidence, a relationship between calcium intake and cardiovascular outcomes is likely not a concern.

No suggestions of serious cardiovascular effects from calcium supplements or calcium with vitamin D had been reported until Bolland, Reid, and colleagues raised the issue of possible increased risk of adverse cardiovascular events in their respective trials (Bolland et al. 2008; Reid et al. 2008). However, although some of these data suggested a hazard ratio for calcium or calcium plus vitamin D as high as 1.43 (43 percent increase in risk), after adjustment for known cardiovascular risk factors, statistical significance was lost (Bolland et al. 2008).

Subsequently, and prior to the 3rd edition of this chapter, published results from several epidemiological studies and one meta-analysis of select RCTs had prompted concern about possible associations between calcium intake and a small increase in risk of adverse cardiovascular events. In this meta-analysis, a subgroup analysis from a large clinical trial — the Women's Health Initiative (WHI) — played an important role (Bolland et al. 2010, 2011). On the basis of this and a follow-up meta-analysis, study authors concluded that calcium supplementation, with or without vitamin D, "modestly" increased the risk for myocardial infarction or stroke and recommended that the use of such supplements in older people should be reassessed (Bolland et al. 2011). However, several limitations in the design and execution of the WHI trial were previously raised in CRN's 3rd edition that preclude confidence in the analyses and findings. These limitations included (1) inadequate monitoring and assessment of compliance with the treatment protocol, (2) use of non-trial calcium supplements by the majority of subjects in the placebo and calcium treatment groups, and (3) lack of information on and adjustment for

known cardiovascular risk factors. With these limitations, confounding and bias cannot be excluded as explanations for the results of the Bolland et al. (2011) subgroup analysis.

The conclusions and recommendations of Bolland, Reid, and colleagues, based on their own data and interpretations, have also been questioned by experts who have raised concerns about the methodology employed, the potential for bias and confounding, and a lack of biological plausibility (Letters to the Editor 2008, 2010, 2011; Bockman et al. 2011; Nordin et al. 2011; Wallace and Weaver 2020). Importantly, a comprehensive, independent analysis of the original WHI dataset, the observational follow-up, and both datasets combined, found no evidence that calcium supplementation increased the risk of myocardial infarction, coronary heart disease, stroke, or total cardiovascular disease (Prentice et al. 2013). In fact, a reduction in total heart disease risk and cardiovascular disease risk was suggested when considering the observational follow-up study data.

While a comprehensive summary and assessment of all potentially relevant data is outside the scope of this review, other studies have been published with inconsistent findings. For example, Li and colleagues reported that, in a large epidemiological study, higher intakes of total dietary and dairy calcium significantly reduced the risk of myocardial infarction, but users of calcium supplements had significantly increased risk (Li et al. 2012). There was no increased risk of stroke or cardiovascular disease-related mortality after a mean follow-up of 11 years. Similarly, in one RCT — the Calcium Intake Fracture Outcome Study (CAIFOS) — 1,200 mg per day of supplemental calcium for five years was not found to increase the risk of vascular disease or related mortality in elderly women (Lewis et al. 2011). A review and meta-analysis of RCTs and prospective cohort studies by Wang et al. (2010), funded by the American Heart Association and the National Heart, Lung, and Blood Institute of the NIH, showed that the relative risk for cardiovascular disease events was 1.14 (95 percent confidence interval, 0.92 to 1.41) in studies involving calcium supplementation without vitamin D. The authors concluded calcium supplements did not appear to exert cardiovascular effects. Systematic reviews and meta-analyses conducted since CRN's 3rd edition have generally concluded there to be no positive relationship between supplemental calcium intake and cardiovascular risk. Yang et al. (2020) concluded that calcium supplements "might raise" the risk of coronary heart disease, especially myocardial infarction. However, the impact of such findings has been questioned based on

limitations with study design and inherent limitations in these types of meta-analyses. In addition, the analysis of Yang et al. excluded studies with <800 mg calcium per day and was based on low versus high intake levels (discussed in Wallace and Weaver 2020).

Given the widespread use of calcium supplements and the potential of harm from inadequate calcium intake, CRN previously concluded that a thorough examination of the evidence for harm and for benefit from calcium supplementation was warranted. To accomplish this goal, CRN convened a group of academic and industry experts to develop a consensus on the available evidence, with emphasis on five of the Bradford-Hill criteria for causal inference from data: strength, consistency, dose-response, biological plausibility, and results from experimentation. The outcome of this assessment was published by Heaney et al. (2012). Heaney and colleagues (2012) summarized data not only from the papers by Bolland et al. and Li et al. but also results from other pertinent long-term prospective cohort studies and clinical trials. Because of the mixed results across studies, Heaney et al. (2012) determined that the findings from available clinical trials and prospective cohort studies indicate that there is no significant effect of calcium supplements on cardiovascular disease.

Other independent reviews conducted since the original WHI trial (and follow-up analyses) have reached similar conclusions, as summarized below:

- While not discussed in CRN's 3rd edition, the EFSA (2012) reviewed RCTs, prospective cohort studies, and systematic reviews and meta-analyses pertinent to an evaluation of cardiovascular outcomes. The EFSA Panel concluded "that long-term calcium intakes from diet and supplements up to 2,500 – 3,000 mg/day are not associated with an increased risk of cardiovascular disease in adults."
- The National Osteoporosis Foundation (NOF) and American Society for Preventive Cardiology (ASPC) commissioned an independent review by an expert panel to evaluate the effects of calcium intake on cardiovascular disease (Kopesky et al. 2016). Based on its review of four RCTs, one nested case-control study, and 26 prospective cohort studies, the panel concluded "there is moderate-quality evidence (B level) that calcium with or without vitamin D intake from food or supplements has no relationship (beneficial or harmful) to the risk for cardiovascular and cerebrovascular disease, mortality, or all-cause mortality in generally healthy adults at this time." The NOF and

ASPC issued a clinical guideline stating that "intake from food and supplements that does not exceed the tolerable upper level of intake (defined by the National Academy of Medicine as 2000 to 2500 mg/d) should be considered safe from a cardiovascular standpoint."

- The Linus Pauling Institute at Oregon State University reviewed available data from RCTs, prospective cohort studies, reanalysis of previous trial data, and meta-analyses (Delage 2017). The primary conclusion of this review highlighted the need for studies designed to specifically examine the effect of calcium supplements on cardiovascular risk as a primary outcome, citing that because the "clinical trial data are limited to analyses of secondary endpoints, meta-analyses should be interpreted with caution."
- Most recently, an editorial published by Wallace and Weaver (2020) reviewed available human data (RCTs, prospective cohort studies, reanalysis of previous trial data, and meta-analyses) as well as data relevant to a potential mechanism of action (e.g., in animal models). Wallace and Weaver concluded that, "the hypothesis that calcium supplements may have a causal inference on cardiovascular events is founded upon a small portion of secondary analyses of RCTs and observational analyses lacking appropriate primary outcome measures, consideration of kidney function and other potential confounders as covariates, and a small number of events mostly self-reported and non-adjudicated. More importantly, the hypothesis currently lacks biological plausibility and is likely a methodological confound."

Considering the totality of evidence, including the many inconsistencies and uncertainties discussed above, a relationship between calcium intake and cardiovascular outcomes is likely not a concern. However, as new data become available, this area of research may warrant additional consideration.

Of important note, one hypothesized mechanism for potential cardiovascular effects is the development of chronic hypercalcemia, which could in turn cause arterial calcification and increase the risk of cardiovascular events. Any potential adverse effects from such a pathway are not considered to be relevant to the general population but should be taken into account for individuals with chronic kidney disease (Wallace and Weaver 2020). While it is recognized that calcium may be used in the treatment of chronic kidney disease as a phosphate binder, CRN

recommends that patients use calcium supplementation only under medical supervision. As such, patients with chronic kidney disease are excluded from CRN's supplemental UL for the general population.

Other Considerations

The harmful effects of treating peptic ulcers with milk and absorbable antacids were historically called milk-alkali syndrome (MAS). This condition involves metabolic alkalosis and high blood calcium, often accompanied by dehydration, kidney failure, kidney calcification, or kidney stones. Although milk-based ulcer therapy is now obsolete, similar problems have been reported from excessive calcium carbonate supplements. This has led to the term calcium-alkali syndrome (CAS). The IOM (2011) previously concluded that intakes around 3,000 mg per day from supplements could be unsafe with regard to such effects. The EFSA (2012) noted that the potential to develop CAS is increased in individuals with decreased calcium excretion, decreased kidney function, increased calcium resorption (e.g., certain medications), or conditions leading to metabolic alkalosis (e.g., hyperemesis in pregnancy or bulimia). Individuals with these conditions, including age-related decreases in kidney function, should consult with their healthcare providers before taking calcium supplements. Hypercalcemia can be a serious health condition if left untreated (Delage 2017; EFSA 2015).

As described in the *Official Reviews* section, the IOM (2011) identified a LOAEL for increased risk of kidney stone formation in a clinical study (WHI)) of menopausal women consuming a mean of 2,100 mg calcium per day (including 1,000 mg supplemental) together with vitamin D supplementation (Jackson et al. 2006). However, as discussed in the *Cardiovascular Effects* section, the WHI trial had significant limitations and follow-up analyses have concluded there to have been no significant association when only subjects compliant with the study protocol were included (Wallace et al. 2011). The EFSA (2012) concluded that Jackson et al. (2006) "does not provide evidence for an increased risk of kidney stones which could be attributed to high calcium intakes." In addition, findings in other clinical and prospective cohort studies have demonstrated an inverse association between total calcium intake and kidney stone formation (reviewed in Delage 2017; EFSA 2012). The EFSA (2012) concluded that intakes up to 3,000 mg per day were not associated with an increased risk of kidney stones in the general

population.

The potential relationship between intake of calcium and increased risk of prostate cancer has also been studied in several prospective cohort and clinical studies, as reviewed by Delage (2017) and EFSA (2012). While the impetus for these assessments may have been originally based on concerns regarding high intakes of dairy and increased risk of effects, the available data are inconsistent with regards to calcium intake. Following its review, the EFSA (2012) concluded that "long-term calcium intakes from diet and supplements above 2,000 mg/day are not associated with an increased risk of prostate cancer." While the available data are insufficient to conclude a potential relationship between calcium supplementation and prostate cancer risk, as new data become available, this area of research may warrant additional consideration.

Updated Clinical Data

A large volume of human intervention studies published since the 3rd edition were identified with potential relevance to this update.[1] Following full text screening, 54 four peer-reviewed publications available since 2006 were identified that met all inclusion criteria for supplemental calcium (described below).[2,3] In the 38 unique trials identified, supplemental calcium was tested at doses ranging from 200 to 2,000 mg per day with no serious adverse effects reported.

Six studies[4] were identified in the updated literature that administered up to 1,200 mg per day calcium. Subih et al. (2018) randomized 45 obese females to receive vitamin D3, calcium (1,200 mg per day), both, or neither for 12 weeks and concluded that no subjects showed any symptoms of vitamin D or calcium toxicity. Two additional new studies reported a lack of any safety-related findings or side effects other than constipation following supplemental calcium of 1,200

[1] Literature search conducted December 2025.
[2] 38 unique studies were identified; 16 publications were determined to be secondary analyses of study cohorts already represented
[3] Studies in which calcium was use a phosphate binder in chronic kidney disease patients and/or in which calcium was used as a negative control/comparator were excluded.
[4] Reported across eleven total publications

mg per day (Ettinger et al. 2014; Ring Madsen et al. 2018). Three of the publications identified were additional analyses from an older study published by Prince et al. (2006). In this large, randomized, double-blind, placebo-controlled trial, 1,460 elderly women received 1,200 mg calcium as calcium carbonate per day (600 mg twice per day) or placebo for five years. Of the 92,000 adverse events recorded, only constipation was found to be higher in the calcium group when compared to the placebo group. No other adverse events related to calcium were reported in the three follow-up studies to Prince et al. (2006) identified in this update (Lewis et al. 2014, 2016; Ghasemifard et al. 2025). In one large, randomized, double-blind, placebo-controlled trial, 2,259 patients with recently diagnosed adenomas (without known colorectal polyps remaining) were given vitamin D3, calcium as calcium carbonate (1,200 mg per day), both, or neither for 3 to 5 years (Baron et al. 2015). The authors stated that "important" adverse events were generally uncommon. In addition, no adverse effects were reported in the three follow-up or add-on studies to the original Baron et al. (2015) publication also identified as part of this update (Rees 2016a,b; Barry et al. 2017; Hodge et al. 2018). The final study identified that included up to 1,200 mg calcium per day was an additional follow-up analysis of the cohort from Reid et al. (2008) (see *Cardiovascular Effects* section above) (Kalluru et al. 2015). Adverse events data from this follow-up showed no differences in cardiovascular effects between groups. In addition to these studies at 1,200 mg per day, one small study in 13 physically active males did not identify adverse events following administration of 400 mg per day or 1,400 mg calcium per day for two weeks (Gonzalez et al. 2014).

Ten studies across ten publications were identified that administered 1,500 mg calcium per day; all studies were conducted in women during pregnancy, and no adverse effects were reported across these studies. One study specifically reported a lack of side effects (Hofmeyr et al. 2019),[5] while another publication reported no differences in safety outcomes between the 500 mg per day and 1,500 mg per day groups in two studies (Dwarkanath et al. 2024). Cormick et al. (2020) stated that, "none of the participants required to interrupt calcium or placebo supplementation due to adverse events." Two additional studies reported no effects on safety-related parameters such as preeclampsia, blood pressure, pregnancy loss, and/or renal function

[5] Subjects received 0 or 500 mg per day prior to pregnancy and up to gestation day 20, then all subjects were given 1,500 mg per day.

(Chaudhary and Manju 2024a,b; Singh et al. 2023). The remaining four studies of 1,500 mg per day calcium did not specifically address side effects or adverse events; however, none were reported (Omotayo et al. 2017; de Brito Pitilin et al. 2024; Prentice et al. 2024; Perumal et al. 2025).

Newer studies administering supplemental calcium at higher levels were limited to a large, single-center study in which thyroidectomy patients with symptoms of hypocalcemia (regardless of serum calcium level) were given up to 2,000 mg per day (500 – 1,000 mg of calcium twice daily) for a median of 3.5 months (Järhult and Landerholm 2016). Asymptomatic patients did not receive calcium supplementation in this study. Aside from monitoring for symptoms of hypocalcemia, no other safety-related data were reported. Two additional publications were identified that reported ad hoc analyses of an older RCT (Bostick et al. 1995). While no adverse effects with calcium supplementation up to 2,000 mg per day in patients with colorectal cancer were reported, an assessment of side effects was not presented (Um et al. 2017; Yang et al. 2016).

Calcium and Vitamin D

Consistent with CRN's methodology (see *Methodology* chapter), clinical trials that did not include at least one group receiving calcium alone were excluded. However, CRN recognizes that supplemental calcium and vitamin D are often used together, and many clinical trials are designed to investigate the effects of their combined exposure. Concurrent use of supplemental calcium and vitamin D is not expected to have any substantial impact on the safety assessment of calcium. In fact, the EFSA (2012) Panel noted, "that the additional data considered (supplementation with calcium plus vitamin D against placebo) does not provide information about the risk associated with supplemental calcium intakes per se." However, CRN considers that concomitant supplemental intakes of these two nutrients may be relevant to the safety assessment of vitamin D. Therefore, studies with combined administration of calcium and vitamin D as the only intervention were considered in CRN's assessment of vitamin D, which can be found in the chapter titled, *Vitamin D*, also available on CRN's website.

Official Reviews

IOM (2011). The IOM evaluated the various potential adverse effects of excess calcium intake (hypercalcemia, hypercalciuria, vascular and soft tissue calcification, kidney stones, prostate cancer, interactions with iron and zinc, and constipation) and concluded that kidney stone formation was the only adverse effect with appropriate data to support a risk assessment (IOM 2011). The IOM determined that a UL of 2,000 mg per day (with no UF) for adults age 51 years and older was justified based on "unknowns surrounding the precision of the LOAEL" and given that the LOAEL was close to the recommended intake for calcium (1,000 mg to 1,200 mg in adults). Due to a lack of relevant data in young adults and given that the risk of kidney stones is less in this population due to better kidney function, 2,500 mg per day — the midpoint between the UL for adolescents (3,000 mg per day) and the UL for older adults (2,000 mg per day) — was selected as the UL for adults 19-50 years of age.

EVM (2003). The UK's EVM concluded that the available data were insufficient to set a safe UL and instead determined a supplemental guidance level. The report recognized that few gastrointestinal side effects have occurred in clinical trials with 1,600 or 2,000 mg of supplemental calcium (Levine et al. 1997; Hofstad et al. 1998; Bonithon-Kopp et al. 2000). Based on a mean dietary calcium intake of 830 mg per day in the UK, the EVM set the guidance level for supplemental calcium at 1,500 mg per day, stating that such a supplemental level "would not be expected to result in any adverse effect."

EFSA (2012). The EC SCF (2003) reviewed the available human clinical studies, including those that looked at hypercalcemia and renal insufficiency (milk-alkali syndrome), kidney stones, cardiovascular disease risk, and interactions with select minerals. For adults, the EC SCF established an UL of 2,500 mg based on a NOAEL of 2,500 mg (total intake) and an UF of 1 due to the "abundance of data" in the overall clinical trial dataset, which demonstrated a lack of any adverse effects at this level. Subsequently, the EFSA (2012) considered available human intervention studies in adults published since the EC SCF's review and concluded that these

studies showed a lack of adverse effects associated with daily calcium intakes of 2,500 mg from both diet and supplements in adults. The EFSA determined that no new data had become available that would require revision to the previous UL and maintained an UL of 2,500 mg per day of calcium from all sources for adults.

Other potential adverse health effects were evaluated in EFSA (2012) and conclusions include:

- Calcium intakes up to 3,000 mg per day (total) had not been associated with an increased risk of kidney stones in the general population.
- Calcium intakes up to ~2,000 mg per day (total) had not been "generally associated" with an increase in cardiovascular events.
- Long-term calcium intakes up to 2,500 to 3,000 mg per day (total) were not associated with an increased risk of cardiovascular disease in adults.
- Long-term calcium intakes above 2,000 mg per day (total) were not associated with an increased risk of prostate cancer.

Chinese Nutrition Society (CNS 2023). The CNS derived an UL of 2,000 mg per day for adults, including pregnant and lactating individuals.

Indian Council of Medical Research - National Institute of Nutrition (ICMR-NIN 2020). The ICMR-NIN reviewed "various studies in India and observed that the values recommended by IOM are similar." An exception was noted by the ICMR-NIN, in which one study reported hypercalciuria and hypercalcemia in 9% and 11% of participants, respectively. Nevertheless, the Council noted that "only few studies on calcium supplementation have reported adverse events" and derived an UL of 2,500 mg per day for adults, including pregnant and lactating individuals.

Korean Nutrition Society (KNS 2020). The KNS published its general approach to evaluating data for setting DRI values. The KNS derived multiple UL values for calcium depending on age: 3,000 mg per day for individuals 15-18 years of age; 2,500 mg per day for adults 19-49 years of age (including pregnant and lactating individuals); and 2,000 mg per day for adults 50 years and older. The KNS also concluded that no relationship had been established between long-term

calcium intakes (from diet and supplements) and increased risk of kidney stones, cardiovascular disease, or prostate cancer.

CRN Recommendations

CRN previously derived an UL for calcium of 1,500 mg per day for adults. The previous edition of this chapter did not review clinical studies in detail but instead relied on the totality of evidence previously reviewed by regulatory agencies to identify a NOAEL, and therefore an UL, as described above. The goal of the current chapter was to determine whether more recent human clinical data are available that might impact the conclusions published in the 3rd edition. While not all human clinical trials are specifically designed to evaluate adverse effects, no new trials were identified following CRN's updated methodology that reported any serious adverse effects with calcium supplementation. As with any assessment in which not all available data are reviewed, inherent uncertainties with the risk assessment and selection of the UL are recognized. The table below summarizes new human clinical studies considered in reviewing the supplemental UL by CRN according to its principal points of departure for risk assessment (as described in the *Methodology* chapter).[6]

[6] Where numerous relevant studies were identified with similar design, dose, and safety-related data, those most pertinent to the UL derivation are included in the table as representative studies. Prioritization was given to studies at dose levels informing the UL and studies with higher weighting based on CRN's methodology (e.g., duration, number of participants, placebo control, randomization, etc.).

New Studies Considered for the CRN UL for Calcium in Adults

Reference	Study Design	Participant Description	No. of Subjects	Dose(s) (mg/day)	Duration	NOAEL (mg/day)
Järhult et al. 2016	Clinical, single-center	Thyroidectomy patients[a]	640	0, 1,000, 2,000	3.5 months (median)	2,000
Hofmeyr et al. 2019	Randomized, double-blind, placebo-controlled	Women prior to and during pregnancy	1,355	0, 500, 1,500	>9 months[b]	1,500
Perumal et al. 2025	Randomized, double-blind, placebo-controlled	Pregnant women	11,000	0, 500, 1,500	From 20 weeks of gestation until delivery	1,500
Dwarkanath et al. 2024	Randomized, double-blind	Pregnant women	11,000[c]	500, 1,500	From 20 weeks of gestation until delivery	1,500
Dwarkanath et al. 2024	Randomized, double-blind	Pregnant women	11,000[c]	500, 1,500	From 20 weeks of gestation until delivery	1,500
Cormick et al. 2020	Randomized, double-blind, placebo-controlled	Pregnant women	630	0, 1,500	From 20 weeks of gestation until delivery	1,500

[a] Only those with symptoms of hypocalcemia were treated, regardless of serum calcium level.
[b] Subjects received 0 or 500 mg per day prior to pregnancy and up to gestation day 20, then all subjects were given 1,500 mg per day.
[c] 11,000 in each of two studies in the same publication

The previous CRN UL value was based on a wide range of clinical and epidemiological studies previously discussed by the IOM (2011), the EC SCF (2003), the EFSA (2012), the EVM (2003), and several published reviews and meta-analyses that had shown no adverse effects with total calcium intakes (from diet and supplements) of 2,000 mg to 2,500 mg per day. In its previous assessment, CRN supported the UL for total intake of 2,000 mg per day or less in adults ages 51 years or older derived by the IOM (2011) and supported the judgment of the IOM (2011) that the calcium UL for persons ages 19-50 years should be 2,500 mg per day, which is the midpoint between the value for individuals ages 51 years and older and the 3,000 mg UL for

adolescents. Based on these levels and considering the variable calcium intake from foods, dairy products, and fortified foods, CRN agreed in its 3rd edition with the EVM that a maximum supplement level for adults should be 1,500 mg. It should be noted the IOM UL was based on the increased risk of kidney stone formation reported in one elderly population supplemented with calcium (Jackson et al. 2006). This finding has not been duplicated in other studies with calcium supplementation, and an association has not been established for the general population.

In addition, no new data were identified that would change conclusions regarding potential adverse effects previously determined not to be associated with calcium intake (CAS, cardiovascular effects, and prostate cancer). Concerns related to the development of CAS have not been fully elucidated, but this effect may be potentially relevant at higher total intakes (e.g., 3,000 mg per day) and associated with specific kidney-related heath limitations (see below).

As summarized in this review, 38 unique trials published since the 3rd edition (and the key regulatory reviews) were identified in which calcium was tested at doses ranging from 200 to 2,000 mg per day with no serious adverse effects reported. Ten of these clinical trials were conducted in pregnant women who were administered up to 1,500 mg calcium per day (Hofmeyr et al. 2019; Dwarkanath et al. 2024; Cormick et al. 2020; Chaudhary and Manju 2024a,b; Singh et al. 2023; Omotayo et al. 2017; de Brito Pitilin et al. 2024; Prentice et al. 2024; Perumal et al. 2025). Studies in pregnant individuals are considered relevant to the development of an UL for calcium for the general population given that intestinal calcium absorption can be increased during pregnancy, thus potentially making this population more vulnerable to adverse effects (IOM 2011). One new study was identified that administered calcium at levels greater than 1,500 mg per day. In this study, thyroidectomy patients with symptoms of hypocalcemia were given up to 2,000 mg per day for a median of 3.5 months with no serious side effects reported (Järhult and Landerholm (2016)). However, this study was not randomized and, other than monitoring for symptoms of hypocalcemia, no other safety-related data were reported. Taken together, the newly available clinical trial data support the previous CRN UL for calcium of 1,500 mg per day.

No new data were identified in this update that would impact the conclusions published in the 3rd

edition of this chapter. Therefore, based on the overall dataset, 1,500 mg per day is maintained as the supplemental NOAEL for calcium for adults following the CRN process. Consistent with CRN's methodology, an UF of 1 is applied to yield an UL of 1,500 mg per day for supplemental calcium in adults.

This UL does not apply to patients with chronic kidney disease. In addition, individuals with otherwise decreased kidney function, decreased calcium excretion or increased reabsorption, or conditions leading to metabolic alkalosis (e.g., hyperemesis in pregnancy or bulimia) should consult with their healthcare providers before taking calcium supplements.

Due to the many inconsistencies and uncertainties in the data discussed herein, a relationship between calcium intake and cardiovascular outcomes is unlikely and not suitable for UL development. However, as this area of research continues to grow, additional data may become available to warrant revision of this assessment.

Quantitative Summary for Calcium in Adults

CRN (2026) UL, supplemental intake	1,500 mg/day for most adults[a]
IOM (2011) UL, total intake	2,500 mg/day (19-50 years) 2,000 mg/day (51 years and older)
EFSA (2012) UL, total intake	2,500 mg/day
EVM (2003), guidance level, supplemental intake	1,500 mg/day
CNS (2023), total intake	2,000 mg/day
ICMR-NIN (2020), total intake	2,500 mg/day
KNS (2020), total intake	3,000 mg/day (15-18 years) 2,500 mg/day (19-49 years) 2,000 mg/day (50 years and older)

[a] Excludes patients with chronic kidney disease. In addition, individuals with otherwise decreased kidney function, decreased calcium excretion or increased reabsorption, or conditions leading to metabolic alkalosis (e.g., hyperemesis in pregnancy or bulimia) should consult with their healthcare providers before taking calcium supplements.

References

Amarnath SS, Kumar V, Barik S. 2023. Vitamin D and Calcium and Bioavailability of Calcium in

Various Calcium Salts. *Indian J Orthop*. 57(Suppl 1):62-69.

Baron JA, Barry EL, Mott LA, Rees JR, Sandler RS, Snover DC, Bostick RM, Ivanova A, Cole BF, Ahnen DJ, et al. 2015. A trial of calcium and vitamin D for the prevention of colorectal adenomas. *N Engl J Med*. 373:1519–30.

Barry EL, Peacock JL, Rees JR, Bostick RM, Robertson DJ, Bresalier RS, Baron JA. 2017. Vitamin D Receptor Genotype, Vitamin D3 Supplementation, and Risk of Colorectal Adenomas: A Randomized Clinical Trial. *JAMA Oncol*. 3(5):628-635.

Bockman RS, Zapalowski C, Kiel DP, Adler RA. 2011. The Challenges of the Single Micronutrient Study: Commentary on Calcium Supplements and Cardiovascular Events. Position paper of the ASBMR Professional Practice Committee. Washington, DC: American Society for Bone and Mineral Research.

Bolland MJ, Avenell A, Baron JA, et al. 2010. Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis. *BMJ*. 341:c3691.

Bolland MJ, Barber PA, Doughty RN, et al. 2008. Vascular events in healthy older women receiving calcium supplementation: randomized controlled trial. *BMJ*. 336:262–266.

Bolland MJ, Grey A, Avenell A, Gamble GD, Reid IR. 2011. Calcium supplements with or without vitamin D and risk of cardiovascular events: reanalysis of the Women's Health Initiative limited access dataset and meta-analysis. *BMJ*. 342:d2040.

Bonithon-Kopp C, Kronborg O, Giacosa A, Räthe U, Faivre J. 2000. Calcium and fibre supplementation in prevention of colorectal adenoma recurrence: a randomised intervention trial. *Lancet*. 356:1300–1306.

Bostick RM, Fosdick L, Wood JR, et al. 1995. Calcium and colorectal epithelial cell proliferation in sporadic adenoma patients: a randomized, double-blinded, placebo-controlled clinical trial. *J. Natl. Cancer. Inst*. 87(17):1307–1315.

Chaudhary PK, Manju KK. 2024a. Maternal and Fetal Outcomes Following Calcium Supplementation in High-Risk Pregnancies: Focus on Preeclampsia Prevention Int. *J. Pharm. Clin. Res.* 16(12); 1419-1424.

Chaudhary PK, Manju KK. 2024b. Calcium Supplementation in High-Risk Pregnancies: Impact on the Prevention of Preeclampsia. *Int. J. Pharm. Clin. Res*. 16(12); 1425-1430.

Chinese Nutrition Society (CNS). 2023. Dietary Reference Intakes for China, A summary Report. People's Medical Publishing House.

Chung M, Tang AM, Fu Z, Wang DD, Newberry SJ. 2016. Calcium intake and cardiovascular disease risk: An updated systematic review and meta-analysis. *Ann Intern Med*. 165(12):856.

Cormick G, Betrán AP, Harbron J, Seuc A, White C, Roberts JM, Belizán JM, Hofmeyr GJ. 2020. The Effect of Calcium Supplementation on Body Weight Before and During Pregnancy in Women Enrolled in the WHO Calcium and Preeclampsia Trial. *Food Nutr Bull*. 41(3):332-342.

de Brito Pitilin E, Marafon F, da Silva Rosa Bonadiman B, Pelazza BB, Pillat MM, de Lara JD, de Oliveira PP, Bagatini MD, Schirmer J. 2024. Effects of calcium supplementation on changes in the IL2, IL4, IL6, IL10 axes and oxidative stress in pregnant women at risk for pre-eclampsia. *BMC Pregnancy Childbirth*. 24(1):71.

Delage B. 2017. Calcium webpage. Linus Pauling Institute website. https://lpi.oregonstate.edu/mic/minerals/calcium. (Updated September 2017; Reviewed September 2017).

Dwarkanath P, Muhihi A, Sudfeld CR, Wylie BJ, Wang M, Perumal N, Thomas T, Kinyogoli SM, Bakari M, Fernandez R, Raj JM, Swai NO, Buggi N, Shobha R, Sando MM, Duggan CP, Masanja HM, Kurpad AV, Pembe AB, Fawzi WW. 2024. Two Randomized Trials of Low-Dose Calcium Supplementation in Pregnancy. *N Engl J Med*. 390(2):143-153.

Ettinger AS, Lamadrid-Figueroa H, Mercado-García A, Kordas K, Wood RJ, Peterson KE, Hu H, Hernández-Avila M, Téllez-Rojo MM. 2014. Effect of calcium supplementation on bone resorption in pregnancy and the early postpartum: a randomized controlled trial in Mexican women. *Nutr J*. 13(1):116.

European Commission, Scientific Committee on Food (EC SCF). 2003. Opinion of the Scientific Committee on Food on the Tolerable Upper Intake Level of Calcium. SCF/CS/NUT/UPPLEV/64 Final Report. Brussels.

European Food Safety Authority (EFSA). 2012. EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA); Scientific Opinion on the Tolerable Upper Intake Level of Calcium. *EFSA J*. 10(7):2814–2844.

European Food Safety Authority (EFSA). 2015. EFSA Panel on Dietetic Products, Nutrition and Allergies. Scientific Opinion on Dietary Reference Values for Calcium. *EFSA J*. 13(5):4101.

Expert Group on Vitamins and Minerals (EVM), Committee on Toxicity. 2003. *Safe Upper Levels for Vitamins and Minerals*. London: Food Standards Agency Publications.

Food and Drug Administration (FDA). 1994. Food labeling: health claims; calcium and osteoporosis. *Federal Register*. 58:2665–2681.

Ghasemifard N, Lewis JR, Radavelli-Bagatini S, Laws SM, Stephan BCM, Hodgson JM, Zhu K, Prince RL, Sim M. 2025. Calcium supplementation and the risk of dementia in the Perth Longitudinal Study of Aging Women: a post-hoc analysis of a randomised clinical trial for fracture prevention. *Lancet Reg Health West Pac*. 63:101694.

Gonzalez JT, Green BP, Campbell MD, Rumbold PL, Stevenson EJ. 2014. The influence of calcium supplementation on substrate metabolism during exercise in humans: a randomized controlled trial. *Eur J Clin Nutr*. 68(6):712-8.

Heaney RP, Abrams S, Dawson-Hughes B, et al. 2000. Peak bone mass. *Osteoporosis Int*. 11:985–1009.

Heaney RP, Kopecky S, Maki KC, Hathcock J, MacKay D, Wallace TC. 2012. Review of *Calcium Supplements and Cardiovascular Disease Risk*. *Adv Nutr*. 3:763–771.

Hodge R, Mandle HB, Ray S, Tandon S, Peterson M, Henry A, Jahan FA, Bostick RM, Baron JA, Barry EL, Yacoub R, Rutherford RE, Seabrook ME, Fedirko V. 2018. Effects of Supplemental Calcium and Vitamin D on Expression of Toll-Like Receptors and Phospho-IKKα/β in the Normal Rectal Mucosa of Colorectal Adenoma Patients. *Cancer Prev Res (Phila)*. 11(11):707-716.

Hofmeyr GJ, Betrán AP, Singata-Madliki M, Cormick G, Munjanja SP, Fawcus S, Mose S, Hall D, Ciganda A, Seuc AH, Lawrie TA, Bergel E, Roberts JM, von Dadelszen P, Belizán JM. 2019. Calcium and Pre-eclampsia Study Group. Prepregnancy and early pregnancy calcium supplementation among women at high risk of pre-eclampsia: a multicentre, double-blind, randomised, placebo-controlled trial. *Lancet*. 393(10169):330-339.

Hofstad B, Vant MH, Anderson SN, Owen RW, Larsen S, Osnes M. 1998. The relationship between faecal bile acid profile with or without supplementation with calcium and antioxidants on recurrence and growth of colorectal polyps. *Eur J Cancer Prev*. 7:287–294.

Indian Council of Medical Research - National Institute of Nutrition (ICMR-NIN). 2020. *ICMR-NIN Expert Group on Nutrient Requirement for Indians, Recommended Dietary Allowances (RDA) and Estimated Average Requirements (EAR).*

Institute of Medicine (IOM). 2011. *Dietary Reference Intakes for Calcium and Vitamin D*. Washington, DC: National Academy Press.

Jackson RD, LaCroix AZ, Gass M, Wallace RB, Robbins J, Lewis CE, Bassford T, Beresford SA, Black HR, Blanchette P, Bonds DE, Brunner RL, Brzyski RG, Caan B, Cauley JA, Chlebowski RT, Cummings SR, Granek I, Hays J, Heiss G, Hendrix SL, Howard BV, Hsia J, Hubbell FA, Johnson KC, Judd H, Kotchen JM, Kuller LH, Langer RD, Lasser NL, Limacher MC, Ludlam S, Manson JE, Margolis KL, McGowan J, Ockene JK, O'Sullivan

MJ, Phillips L, Prentice RL, Sarto GE, Stefanick ML, Van Horn L, Wactawski-Wende J, Whitlock E, Anderson GL, Assaf AR, Barad D; Women's Health Initiative Investigators. 2006. Calcium plus vitamin D supplementation and the risk of fractures. *N Engl J Med.* 354(7):669-83.

Järhult J, Landerholm K. 2016. Outcome of hypocalcaemia after thyroidectomy treated only in symptomatic patients. *Br J Surg*. 103(6):676-683.

Kopecky SL, Bauer DC, Gulati M, Nieves JW, Singer AJ, Toth PP, Underberg JA, Wallace TC, Weaver CM. 2016. Lack of evidence linking calcium with or without Vitamin D supplementation to cardiovascular disease in generally healthy adults: A clinical guideline from the National Osteoporosis Foundation and the American Society for Preventive Cardiology. *Ann Intern Med*. 165(12):867.

Korean Nutrition Society (KNS). 2020. Ministry of Health and Welfare (KR). The Korean Nutrition Society. *Dietary Reference Intakes for Koreans*. Sejong: Ministry of Health and Welfare.

Letters to the Editor. 2008. *BMJ*. 336:403–406.

Letters to the Editor. 2010. *BMJ*. 341:569–572.

Letters to the Editor. 2011. *BMJ*. 342:1273–1276.

Levine RJ, Hauth JC, Curet LB, et al. 1997. Trial of calcium to prevent eclampsia. *N Engl J Med*. 337:69–76.

Lewis JR, Calver J, Zhu K, Flicker L, Prince RL. 2011. Calcium supplementation and the risksof atherosclerotic vascular disease in older women: results of a 5-year RCT and a 4.5-year follow-up. *J Bone Miner Res*. 26(1):35-41.

Lewis JR, Zhu K, Thompson PL, Prince RL. 2014. The effects of 3 years of calcium

supplementation on common carotid artery intimal medial thickness and carotid atherosclerosis in older women: an ancillary study of the CAIFOS randomized controlled trial. *J Bone Miner Res*. 29(3):534-4.

Lewis JR, Brennan-Speranza TC, Levinger I, Byrnes E, Lim EM, Blekkenhorst LC, Sim M, Hodgson JM, Zhu K, Lim WH, Adams LA, Prince RL. 2019. Effects of calcium supplementation on circulating osteocalcin and glycated haemoglobin in older women. *Osteoporos Int.* 30(10):2065-2072.

Li K, Kaaks R, Linseisen J, Rohrmann S. 2012. Associations of dietary calcium intake and calcium supplementation with myocardial infarction and stroke risk and overall cardiovascular mortality in the Heidelberg cohort of the European Prospective Investigation into Cancer and Nutrition study (EPIC-Heidelberg). *Heart.* 98:920–925.

McCormick CC. 2002. Passive diffusion does not play a major role in the absorption of dietary calcium in normal adults. *J nutr.* 132(11):3428-3430.

National Institutes of Health (NIH). 2025. Fact Sheet for Health Professionals. https://ods.od.nih.gov/factsheets/Calcium-HealthProfessional/.

Nordin BE, Lewis JR, Daly RM, et al. 2011. The calcium scare: what would Austin Bradford Hill have thought? *Osteoporosis Int*. 22:3073–3077.

Omotayo MO, Dickin KL, Chapleau GM, Martin SL, Chang C, Mwanga EO, Kung'u JK, Stoltzfus RJ. 2015. Cluster-Randomized Non-Inferiority Trial to Compare Supplement Consumption and Adherence to Different Dosing Regimens for Antenatal Calcium and Iron-Folic Acid Supplementation to Prevent Preeclampsia and Anaemia: Rationale and Design of the Micronutrient Initiative Study. *J Public Health Res*. 4(3):582.

Perumal N, Muhihi A, Sudfeld C, Zhong K, Kain KC, Belizán JM, Cormick G, Aboud S, Wylie BJ, Duggan CP, Wang M, Masanja H, Mwanyika-Sando M, Dwarkanath P, Raj JM, Thomas T, Kurpad AV, Pembe AB, Fawzi W. 2025. Calcium Supplementation and the Pathogenesis of

Preeclampsia and Preterm Birth in Tanzania. Available at *SSRN*: https://ssrn.com/abstract=5487875 or http://dx.doi.org/10.2139/ssrn.5487875.

Prentice RL, Pettinger MB, Jackson RD, Wactawski-Wende J, LaCroix AZ, Anderson GL, Chlebowski RT, Manson JE, Van Horn L, Vitolins MZ, et al. 2013. Health risks and benefits from calcium and vitamin D supplementation: Women's Health Initiative clinical trial and cohort study. *Osteoporos Int*. 24(2): 567–580.

Prentice A, Jarjou LM, Goldberg GR, Schoenbuchner SM, Moore SE, Ward KA, Cole TJ. 2024. Effects of maternal calcium supplementation on offspring blood pressure and growth in childhood and adolescence in a population with a low-calcium intake: follow-up study of a randomized controlled trial. *Am J Clin Nutr*. 119(6):1443-1454.

Prince RL, Devine A, Dhaliwal SS, Dick IM. 2006. Effects of calcium supplementation on clinical fracture and bone structure: results of a 5-year, double-blind, placebo-controlled trial in elderly women. *Arch Intern Med*. 166(8):869-7.

Rees JR, Mott LA, Barry EL, Baron JA, Bostick RM, Figueiredo JC, Bresalier RS, Robertson DJ, Peacock JL. 2016a. Lifestyle and Other Factors Explain One-Half of the Variability in the Serum 25-Hydroxyvitamin D Response to Cholecalciferol Supplementation in Healthy Adults. *J Nutr*. 146(11):2312-2324.

Rees JR, Mott LA, Barry EL, Baron JA, Figueiredo JC, Robertson DJ, Bresalier RS, Peacock JL. 2016b. Randomized controlled trials: who fails run-in? *Trials*. 17:374.

Reid IR, Ames R, Mason B, et al. 2008. Randomized controlled trial of calcium supplementation in healthy nonosteoporotic, older men. *Arch Intern Med.* 168:2276–2282.

Ring Madsen L, Espersen R, Rejnmark L, Richelsen B. 2018. Effect of calcium citrate vs calcium carbonate on elevated parathyroid hormone after Roux-en-Y gastric bypass. A double-blinded, randomized trial. *Clin Endocrinol (Oxf)*. 89(6):734-741.

Sing V, Panwar S. 2023. Study on Prevention of Preeclampsia using Calcium Supplements in Early Pregnancy. *Int. J. Pharm. Clin. Res*. 15(6); 988-996.

Subih HS, Zueter Z, Obeidat BM, Al-Qudah MA, Janakat S, Hammoh F, Sharkas G, Bawadi HA. 2018. A high weekly dose of cholecalciferol and calcium supplement enhances weight loss and improves health biomarkers in obese women. *Nutr Res*. 59:53-64.

Um CY, Fedirko V, Flanders WD, Höflich C, Wirthgen E, Bostick RM. 2017. Circulating insulin-like growth factor-related biomarkers: Correlates and responses to calcium supplementation in colorectal adenoma patients. *Mol Carcinog*. 56(9):2127-2134.

Wallace RB, Wactawski-Wende J, O'Sullivan MJ, Larson JC, Cochrane B, Gass M and Masaki K. 2011. Urinary tract stone occurrence in the Women's Health Initiative (WHI) randomized clinical trial of calcium and vitamin D supplements. *Am. J. Clin. Nutr*. 94, 270-277.

Wallace TC, Weaver CM. 2020. Calcium Supplementation and Coronary Artery Disease: A Methodological Confound? *J Am Coll Nutr*. 39(5):383-387. Erratum in: *J Am Coll Nutr.* 2021 Nov-Dec;40(8):735.

Wang L, Manson JE, Song Y, Sesso HD. 2010. Systematic review: vitamin D and calcium supplementation in prevention of cardiovascular events. *Ann Intern Med*. 152:315–323.

Yang B, Bostick RM, Tran HQ, Gewirtz AT, Campbell PT, Fedirko V. 2016. Circulating Biomarkers of Gut Barrier Function: Correlates and Nonresponse to Calcium Supplementation among Colon Adenoma Patients. *Cancer Epidemiol Biomarkers Prev*. 25(2):318-26.

Yang C, Shi X, Xia H, Yang X, Liu H, Pan D, Sun G. 2020. The Evidence and Controversy Between Dietary Calcium Intake and Calcium Supplementation and the Risk of Cardiovascular Disease: A Systematic Review and Meta-Analysis of Cohort Studies and Randomized Controlled Trials. *J Am Coll Nutr*. 39(4):352-370.

Yu E, Sharma S. Physiology, Calcium. In: StatPearls [Internet]. Treasure Island (FL): StatPearls

Publishing. https://www.ncbi.nlm.nih.gov/books/NBK482128/ (Updated August 14, 2023).

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