# Vitamin A - Preformed

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|---|---|
| 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

The term “vitamin A” refers to several related compounds known as preformed vitamin A (i.e., retinoids) and precursors to vitamin A (i.e., provitamin carotenoids) (Drake 2024). This chapter focuses on retinoids (referenced herein as “preformed vitamin A”), while select provitamin A compounds (e.g., beta-carotene and other carotenoids) are addressed in other chapters.

Preformed vitamin A has essential actions in areas of health that include vision, cellular differentiation, organ development during embryonic and fetal growth, and membrane structure

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and function (Borel and Desmarchelier 2017). Several other complex physiological processes, including growth, reproduction, and immune system functions, also depend on vitamin A (Ross 1999). Regarding eye health and function, preformed vitamin A is an essential component of the visual cycle within the eye membranes and is required for the normal functioning of the retina, dim-light vision, and color vision. Lower concentrations of preformed vitamin A can result in impaired dark adaptation; in severe deficiency cases, thinning and ulceration of the cornea can occur, leading to blindness (Drake 2024). Vitamin A deficiency is the foremost cause of preventable blindness in the world. Vitamin A deficiency is still common in developing countries due to limited access to animal-based foods, particularly in women and children (NIH 2025).

Preformed vitamin A compounds are fat soluble and naturally occur in select animal-derived foods, including liver, offal, milk, cheese, and eggs (Borel and Desmarchelier 2017; Drake 2024; EFSA 2024). Retinyl palmitate is the predominate precursor of retinol in foods from animals (Drake 2024). The European Commission regulations have approved preformed vitamin A (retinol, retinyl acetate, or retinyl palmitate) for addition to foods and for food supplements (EC 2002; EC 2006; EFSA 2024). Preformed vitamin A is present in U.S. supplements in the forms of retinyl palmitate and retinyl acetate ranging from 900-3,000 µg Retinol Activity Equivalent (RAE) per day (Drake 2024; NIH 2025).

**Bioavailability**

The oral bioavailability of preformed vitamin A ranges from 70-90% (EFSA 2024). Dietary preformed vitamin A is hydrolyzed to free retinol before absorption by intestinal enterocytes (Chungchunlam and Moughan 2024). The free retinol is absorbed by either passive diffusion or by facilitated transport mechanisms utilizing membrane-bound transporter proteins. Subsequently, free retinol undergoes esterification to form retinyl esters, which are incorporated with dietary fat or cholesterol forming chylomicrons. These chylomicrons are transported to the lymphatic system for entry into the bloodstream (Borel and Desmarchelier 2017; Chungchunlam and Moughan 2024; EFSA 2024). Less polar retinoid forms (e.g., all-*trans* retinol and retinoic acid) are secreted in the portal blood (Borel and Desmarchelier 2017). Both pathways (i.e., chylomicrons pathway or portal blood pathway) transport the preformed vitamin A compounds to the liver, which is the

primary site of vitamin A storage (Vašková et al. 2025; Borel and Desmarchelier 2017). Healthy individuals store approximately 70% of the total body concentration of vitamin A in the liver, in the form of retinyl esters; approximately 10-20% of vitamin A is stored in hepatocytes (Borel and Desmarchelier 2017; EFSA 2024). The majority of the retinyl esters are found in the lipid droplets of stellate cells. Retinyl esters can also accumulate in the eye, lung, adipose tissue, testes, skin, and spleen. Once in the liver, retinyl esters are hydrolyzed to retinol by the hepatocytes (Vašková et al. 2025; EFSA, 2024). The retinol can be converted by the body to retinal, which can be in turn oxidized to retinoic acid (Drake 2024; EFSA 2024). The liver will secrete the preformed vitamin A compounds either in the bile (as oxidized and/or conjugated metabolites) or in the blood (as all-*trans* retinol) (Borel and Desmarchelier 2017). Retinol metabolites are primarily excreted in the urine, whereas biliary metabolites generated from unabsorbed preformed vitamin A are excreted in the feces (Chungchunlam and Moughan 2024; EFSA, 2024). Minimal amounts of retinol metabolites are released in breath (EFSA, 2024).

**Safety Considerations**

The previous edition of this chapter (CRN 3rd edition) discussed the potential serious adverse effects due to the excess consumption of preformed vitamin A identified at that time, including birth defects, bone fragility, and liver abnormalities. The occurrence of birth defects was identified as the critical effect serving as the basis of the UL values previously established by CRN and several authoritative agencies (IOM 2001; EVM 2003; SCF 2002), and more recently by the EFSA (2024). The available human epidemiological data continue to suggest an increased risk of birth defects is associated with intake of preformed vitamin A by pregnant women; as such, this outcome is discussed in detail below (*Birth Defects* section).

Preformed vitamin A is typically listed on food and dietary supplement labels in international units (IU) or in mg or µg RAE. The conversion rate between an IU and RAE is 1 IU retinol = 0.3 µg RAE.[1] This updated chapter will uniformly discuss preformed vitamin A concentrations in terms of RAE.

[1] https://dsid.od.nih.gov/Conversions.php

### *Clinical Studies*

No new data have become available since CRN's 3rd edition that identifies any critical effects in clinical trials with preformed vitamin A. No serious adverse effects were reported in the 20 human intervention studies meeting the inclusion criteria for this review from the updated literature search.[2] Out of these 20 studies, only three studies (two studies within the same cohort) were conducted in pregnant women administered doses of 1,500 µg RAE per day, 3,000 µg RAE once a week, or 3,000 µg RAE three times a week for 15 days to six months (Ahmad et al. 2018; Elom et al. 2017;, 2019). These studies are discussed in the *Birth Defects* section below.

Ten studies (two studies within the same cohort) administered doses of preformed vitamin A ranging from 3,000-7,500 µg RAE per day for durations ranging from 2-6 months in diseased state patients (e.g., post COVID-19, atherosclerotic, or multiple sclerosis patients); however, these studies did not include monitoring for adverse events or side effects in the methodology (Mottaghi et al. 2014; Bitarafan et al. 2015, 2016; Saboor-Yaraghi et al. 2015; Sezavar et al. 2015; Mahmoudi et al. 2016; Mohammadzadeh Honarvar et al. 2016; Ahmadvand et al. 2021; Pfau et al. 2023; Taheri et al. 2024). Six studies administered doses of preformed vitamin A ranging from 6,000-7,500 µg RAE per day for durations ranging from 1-6 months in diseased state patients (e.g., post COVID-19, multiple sclerosis, obese, ulcerative colitis, or post-surgical patients) with no reported serious adverse effects (Farhangi et al. 2016; Masnadi Shirazi et al. 2018; Bitarafan et al. 2019; Uberti et al. 2020; Hanna et al. 2021; Chung et al. 2023; Rahimi-Dehgolan et al. 2023). Similarly, a study administering preformed vitamin A at 45 mg/m$^2$ (minimum 10,000 µg per day) for two weeks reported no serious adverse effects in advanced adenoid cystic carcinoma patients (Hanna et al. 2021). Regarding potential hepatotoxic effects of preformed vitamin A, three studies reported no effects on liver enzyme levels, as discussed in the *Liver Abnormalities* section (Bitarafan et al. 2015, 2016; Uberti et al. 2020).

### *Birth Defects*

[2] Literature search conducted September 2025. Three of these clinical studies were from the same cohort and two other studies were part of another cohort.

Human epidemiological studies indicate an increased risk of birth defects associated with higher intakes of preformed vitamin A. As reviewed previously by CRN, the prospective cohort study conducted by Rothman et al. (1995) concluded that there was a significantly increased risk of neural crest birth defects at maternal daily supplemental levels of more than 3,000 µg RAE per day (total intake levels >3,000 µg RAE per day), but exact exposure levels associated with the increased incidence of birth defects were not reported. A few reports suggested the possibility that there may be some risk of vitamin A toxicity at supplementation levels below 6,000 µg RAE per day. For example, one report to the FDA suggested a characteristic birth defect in association with maternal supplementation at 5,400 µg RAE per day (Rosa et al. 1986). However, this observation has not been confirmed (Stauber et al. 1991), and the same laboratory was unable to repeat this finding in later research (Johnson et al. 1992).

The EFSA (2024) assessment selected birth defects as the critical endpoint for establishing an UL for preformed vitamin A, based on data from Rothman et al. (1995; as previously determined by SCF 2002) and the identification of a dose-response relationship (see *Official Reviews* section). The Panel determined that Rothman et al. (1995) was the only prospective cohort that addressed the dose–response relationship between the intake of preformed vitamin A and birth defects, noting that this study identified a threshold of 3,000 µg RAE per day supplemental retinol consumed during the first trimester of pregnancy, resulting in observations of cranial neural crest defects. The EFSA Panel considered this threshold as the NOAEL for teratogenicity for preformed vitamin A from all sources (i.e., diet and supplementation). Other studies considered by SCF (2002) were concluded by EFSA (2024) to demonstrate no increased risk of birth defects at 3,000 µg RAE per day (Martínez-Frías and Salvador 1990; Mills et al. 1997; Shaw et al. 1997; Mastroiacovo et al. 1999).

The EFSA (2024) also reviewed other prospective cohort studies, case-control, and case-cohort studies, which were determined to support the Panel's conclusions based on Rothman et al. (1999). In a prospective cohort study by Mastroiacovo et al. (1999), consumption of supplemental preformed vitamin A at doses greater than 3,000 µg RAE per day by pregnant women were tracked, and only three cases of major malformations were identified in the exposed group, as compared to four cases in exposed controls (intakes ≥7500 µg RAE per day) and 13 in non-exposed controls. However, the EFSA noted that this study had high risk of bias

for the outcome assessment, confounding, selection bias, and attrition (EFSA 2024). In another prospective cohort by Michikawa et al. (2019), consumption of retinol in the diet was not associated with an increased risk of congenital birth defects (~ 631 to 940 µg RAE per day). The EFSA noted that this study had a moderate risk of bias for exposure assessment and confounding (EFSA 2024). Case-cohort (>800 µg RAE per day) and case-control (3,700 µg RAE per day) studies from Bille (2007) and Johansen et al. (2008), respectively, did not observe any increased risk in defects. Botto et al. (2001) observed an increased risk of outflow tract defects of transposition of great arteries at preformed vitamin A supplementation of greater than 3,000 µg RAE per day.

Three studies identified in CRN's updated literature search did not report any serious adverse effects when administering doses of preformed vitamin A during pregnancy. Two studies, from the same cohort, did not report observing or monitoring for any adverse effects when administering 3,000 µg RAE, three times a week to malaria-infected pregnant women from 6 months pregnancy to delivery (Elom et al. 2017; Elom et al. 2019). Ahmad et al. (2018) administered 3,000 µg RAE per week to pregnant women in their 2$^{nd}$ trimester for over six months with no reported serious adverse effects. These studies did not report any effect on pregnancy outcomes or birth defects and support the currently established UL of 3,000 µg RAE per day set by SCF (2002) and EFSA (2024).

***Other Outcomes Considered***

Liver Abnormalities

Because the liver is the principal storage site for excess vitamin A, a causal relationship between very high intakes and liver toxicity is well established in both animals and humans. The adverse effects can include reversibly elevated liver enzymes as well as other conditions with greater persistence, such as fibrosis, cirrhosis, and even death (IOM 2001). The human data, however, are often confounded by other factors such as alcohol intake, infectious hepatitis, hepatotoxic drugs, and pre-existing liver disease. Consumption of 7,507-15,015 µg RAE per day of preformed vitamin A per day for periods of several months or more has been suggested to cause liver toxicity (Hathcock et al. 1990); but the effects in this intake range may be dependent on compromised liver health or function. Another report found marginal indications of adverse

effects on the liver in elderly subjects with chronic supplementation at levels of 1,501-3,000 µg RAE per day (Krasinski et al. 1989). However, one older study concluded that a supplemental intake of approximately 7,507 µg RAE per day is the lowest dose at which such effect can be confidently attributed to vitamin A in persons with mildly or moderately compromised liver health (Geubel et al. 1991). In addition, in the recent EFSA (2024) scientific opinion, a systematic review was conducted, which identified four human clinical trials reporting on relevant liver parameters (Bitarafan et al. 2015; Farhangi et al. 2013; Alberts et al. 2004; Dougherty et al. 2012). These studies did not report any adverse effects on liver enzyme levels in adults that were administered preformed vitamin A at doses between 7,500- 22,500 µg RAE per day. The EFSA Panel noted that there were limitations to these trials, including short study duration, small sample size, and/or no dose was tested below the LOAEL for liver damage (i.e., 7,500 µg RAE per day). The Panel concluded that the available data do not allow for the determination of a dose-response relationship between the intake of preformed vitamin A and liver damage. In addition to Bitarafan et al. (2015) included in the EFSA systematic review, the updated literature search for this chapter identified two studies that reported no effects on liver enzyme levels at doses of 6,000-7,500 µg RAE per day for 1.5-6 months (Bitarafan et al. 2016; Uberti et al. 2020).

Bone Fragility

In January 2001, the IOM released its review of vitamin A and other micronutrients. In assessing the safety of vitamin A, the IOM considered possible adverse effects in relation to bone mineral density and hip fracture, concluding that the studies are "provocative but conflicting, and therefore they are not useful for setting a UL for vitamin A." The evidence that the IOM reviewed relating vitamin A to potential adverse effects on bone included animal studies, human mechanistic studies, and epidemiological evidence (IOM 2001). In the recent EFSA (2024) opinion, the Panel determined that the causality of the relationship between the intake of preformed vitamin A at levels that are below the EFSA's current UL of 3,000 µg RAE per day (~ 10,000 IU per day and an increased risk of bone fractures cannot be established. The EFSA Panel also commented that studies published after the SCF (2002) review do not support an association between preformed vitamin A at intakes less than 3,000 µg RAE per day and impaired bone health (EFSA 2024). The EFSA's evaluation included prospective observational studies; no RCTs were

identified meeting EFSA's inclusion criteria. No studies assessing endpoints related to bone fragility were identified in the updated literature search for this chapter.

**Official Reviews**

**IOM (2001).** Based on the available data, the IOM selected teratogenicity as the critical adverse effect for women of childbearing age, while liver abnormalities were selected as the critical adverse effect for all other adults. The IOM identified an NOAEL of 4,500 µg RAE per day (~15,000 IU; from consumption of food and supplements) based on case-control studies that reported intakes greater than 4,500 µg RAE per day (Brent et al. 1996; Watkins et al. 1996; Werler et al. 1996; Mastroiacovo et al. 1999; Rothman et al. 1995). An UF of 1.5 was applied to the NOAEL of 4,500 µg RAE per day to derive an UL of 3,000 µg RAE per day (~10,000 IU per day) for women of childbearing age. The Committee noted that were "numerous studies showing no adverse effects" at intake levels below 3,000 µg RAE per day of vitamin A from supplements only (Czeizel and Rockenbauer 1998; Dudas and Czeizel 1992; Khoury et al. 1996; Rothman et al. 1995) or 4,500 µg RAE per day of preformed vitamin A from food and supplements (Rothman et al. 1995).

Regarding hepatotoxicity, the IOM stated that studies have demonstrated vitamin A-induced hepatotoxicity at intakes less than 14,000 µg RAE per day (Eaton 1978; Hatoff et al., 1982; Kowalski et al., 1994; Oren and Ilan, 1992). In addition, hepatoxicity effects were observed in numerous studies at doses at and above 14,000 µg per day. The IOM derived an UL of 3,000 µg RAE per day for adults, other than women of reproductive age, from the LOAEL of 14,000 µg RAE per day for hepatotoxicity, after a UF of 5 was applied.

**Expert Group on Vitamins and Minerals (EVM 2003).** The UK's EVM concluded that retinol intakes of 3,000 µg RAE per day were not teratogenic. This conclusion was based on the study by Rothman et al. (1995) which demonstrated that supplemental doses greater than 3,000 µg RAE per day, in addition to the diet, "may be teratogenic." The EVM noted that an "excess of cranial defects" were observed in the offspring of women consuming 5,000 µg RAE/day[3] from food and supplements compared to those taking 1,660 µg RAE per day (Rothman et al.

[3] This value appears to be based on the value of >4,500 µg RAE per day, as reported by the study.

1995), while other studies indicated that the "critical effect threshold may be higher" (Martínez-Frías and Salvador 1990; Mills et al. 1997; Shaw et al. 1997; Mastroiacovo et al. 1999). The EVM also reviewed long-term studies with dietary intake of vitamin A that had been associated with "decreased bone density and increased risk of hip fracture." The EVM discussed that epidemiological data suggested that long-term high intakes of vitamin A are associated with an increased risk of hip fracture in men and women, but this effect was also observed at average dietary intakes. The EVM concluded that "it is not possible to identify an intake that is without some degree of risk of hip fracture." The EVM identified a supplemental guidance level of 1,500 µg RAE per day based on the available data.

**European Food Safety Authority (EFSA 2024).** The EC SCF (2002) identified a UL of 3,000 µg RAE per day (~10,000 IU per day; from foods and supplements). Similar to IOM's conclusion, the EC SCF based its conclusion on the risk of birth defects due to "the severe and irreversible nature of this form of toxicity." In 2024, the EFSA delivered a scientific opinion after reviewing the safety of preformed vitamin A (including retinol, retinyl acetate, and retinyl palmitate). The EFSA Panel upheld the UL of 3,000 µg RAE per day (~ 10,000 IU per day) set in 2002 by SCF. This UL is based on teratogenicity as the critical effect; however, the EFSA noted that "a UL based on this effect would also cover for other adverse effects of excess preformed vitamin" and it therefore applies to all adults, including men and women, including those of child- bearing age, pregnant, lactating, and post-menopausal. As reviewed by EFSA (2024), a NOAEL of 3000 µg per day was derived from a prospective cohort study (Rothman et al. 1995) that demonstrated a dose-response between the intake of preformed vitamin A and birth defects. The EFSA Panel did not apply uncertainty factors to this NOAEL based on data from another prospective cohort study (Mastroiacvo et al. 1999) and three case-control studies (Martínez-Frías and Salvador 1990; Mills et al. 1997; Shaw et al. 1997) which suggested that the "true threshold for this adverse effect could be higher" than 3,000 µg RAE per day. The EFSA (2024) also described two newer case-control studies that supported its conclusions (Botto et al. 2001; Johanson et al. 2008).

Other potential adverse health effects were evaluated by the EFSA (2024) Panel, with the following conclusions:

- The causality of the relationship between the intake of preformed vitamin A at levels that are below the current UL (i.e., in the range 1,000–3,000 µg RAE per day) and an increased

risk of bone fractures could not be established. The Panel also considered that the "evidence that has become available since the assessment of the SCF (2002) does not support the association between preformed vitamin A at intakes ≤ 3,000 µg RAE per day and impaired bone health."

- The available evidence for hepatotoxicity did not allow for characterizing the dose–response relationship between the intake of preformed vitamin A and liver damage or to explore whether high intakes of β- carotene could potentiate preformed vitamin A-induced hepatotoxicity. The EFSA Panel commented that "the currently available randomized controlled trial, conducted with doses of retinyl palmitate up to 22,500 µg RAE per day for up to 12 months, does not address the question raised by the SCF (2002) of whether intake levels of preformed vitamin A < 7,500 µg RAE per day consumed for six years are hepatotoxic."

**Chinese Nutrition Society (CNS 2023).** The CNS developed an UL of 3,000 µg RAE per day for adults, including pregnant and lactating individuals.

**Indian Council of Medical Research - National Institute of Nutrition (ICMR-NIN 2020).** The ICMR-NIN derived an UL of 3,000 µg RAE 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 an UL of 3,000 µg RAE per day for adults (age 19 years and older), including pregnant and lactating individuals. An UL of 2,800 µg RAE per day was derived for males and females 15-18 years of age.

**CRN Recommendations**

The goal of this chapter was to determine whether more recent human clinical data are available that might impact the conclusions published in the 3rd edition, which derived two UL values for preformed vitamin A of 3,000 µg RAE per day for low consumers of fortified foods and liver and 1,500 µg RAE per day for high consumers of fortified foods and liver.

CRN's safety methodology for deriving supplemental UL values prioritizes data from human

studies, when available. Approximately 20 human clinical trials published since 2014 were identified and subsequently reviewed that met the inclusion criteria for the current chapter. While not all human clinical trials are specifically designed to evaluate adverse effects, no new trials were identified that reported any serious adverse effects with supplemental preformed vitamin A. Therefore, risk of birth defects based on human epidemiological data was carried forward as the critical effect for updating CRN's supplemental UL value. An updated search for epidemiological data published since the EFSA (2023) assessment was not conducted as part of this review and is noted as a limitation. The table below summarizes the key human studies considered in deriving the supplemental UL value by CRN according to its principal points of departure for risk assessment (as described in the Methods). Only studies identified in the review that are most pertinent[4] based on CRN's methodology are summarized below.

**Key Studies Considered for the CRN UL for Vitamin A in Adults: Increased Risk of Brith Defects Following Maternal Consumption**

| | | Food and Supplemental Intake Combined (µg RAE per day) | | Supplemental Intake Only (µg RAE per day) | |
|---|---|---|---|---|---|
| **Reference** | **Study Design** | **NOAEL** | **LOAEL** | **NOAEL** | **LOAEL** |
| Rothman et al. 1995 | Prospective cohort | 4,500 | >4,500 | 3,000 | >3,000 |
| Johansen et al. 2008 | Population-based, case-control | N/A | N/A | >3,763 | N/A |
| Botto et al. 2001 | Case-control | ≥3,000[a] | N/A | <3,000 | ≥3,000[b] |
| Mastroiacovo et al. 1999 | Prospective, controlled cohort | N/A | N/A | 3,000 | 7,500 |
| Martínez-Frías and Salvador 1990 | Hospital-based, case-control | N/A | N/A | 3,000 | 12,000 |
| Mills et al. 1997 | Geographically-based, case-control | N/A | N/A | >3,000[c] | N/A |
| | | N/A | N/A | >2,400[d] | N/A |
| Shaw et al. 1997 | Case-control | N/A | N/A | 3,000 | N/A |

N/A, not applicable
[a] Food only (no supplements)
[b] Only retinol intakes from supplements ≥3,000 µg RAE per day were associated with an increased risk and only of one defect type (outflow tract defects with transposition of great arteries).
[c] Fortified cereals plus supplements
[d] Supplements only

[4] Where numerous relevant studies were identified, 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 Methods (e.g., duration, number of participants, etc.) and the outcome of the risk of bias assessment conducted by EFSA (2024).

Since the establishment of the most recent CRN supplemental UL for preformed vitamin A in 2014, no new data have demonstrated adverse effects at intake levels less than 3,000 µg RAE per day. Previous CRN recommendations were based on evidence of birth defects risks at higher levels of preformed vitamin A intakes from human epidemiological studies. This critical effect is carried forward as the basis for CRN's updated supplemental UL value, where a supplemental NOAEL of 3,000 µg RAE per day of preformed vitamin A is identified following the CRN process (Rothman et al. 1995; Mastroiacovo et al. 1999; Martínez-Frías and Salvador 1990). This supplemental NOAEL assumes intake of vitamin A from foods alone is approximately 1,500 µg RAE per day or less, corresponding to the NOAEL for total intake of 4,500 µg RAE per day from the Rothman et al. (1995) study. As described in CRN's Methods, if the supplemental intake dose-response relationship is identified from the strongest data and assessed conservatively, no additional uncertainty factor is needed (that is, the implicit UF is 1.0). Consistent with CRN's methodology, an UF of 1 is applied to the supplemental NOAEL of 3,000 µg RAE per day to yield an UL of 3,000 µg RAE per day for preformed vitamin A in adults.

This UL value does not apply to nutritionally deprived populations requiring higher supplemental levels of vitamin A under medical supervision.

CRN considers supplemental intakes of 3,000 µg RAE per day of preformed vitamin A to be safe for most people. However, some uncertainty regarding effects in populations with higher dietary intakes is noted. For example, the mean daily intake of preformed vitamin A has been reported to range from less than ~1,200 µg RAE per day to 1,500 µg RAE per day from food sources only (EFSA 2024; Feskanich et al. 2002; Rothman et al. 1995). Comparatively, the EFSA (2024) estimated very high consumer intake (95th percentile) can range from 4,048-6,850 µg RAE per day. Therefore, individuals with a higher-than-average intake of vitamin A in the diet, such as from vitamin A-fortified foods, liver, or other organ meats, should consult their healthcare provider before taking preformed vitamin A-containing supplements.

## Quantitative Summary for Preformed Vitamin A

| | |
|---|---|
| CRN UL (2025), supplemental intake | 3,000 µg RAE per day[a] |
| IOM UL (2001), total intake | 3,000 µg RAE per day |
| EFSA UL (2024), total intake | 3,000 µg RAE per day |
| EVM (2003), guidance level, total intake | 1,500 µg RAE per day |
| CNS (2023) | 3,000 µg RAE per day |
| ICMR-NIN (2020) | 3,000 µg RAE per day |
| KNS (2020) | 3,000 µg RAE per day (19 years and older);<br>2,800 µg RAE per day (15-18 years of age) |

[a] Individuals with a higher-than-average intake of vitamin A in the diet, such as from vitamin A-fortified foods, liver, or other organ meats, should consult their healthcare provider before taking preformed vitamin A-containing supplements.

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