Nickel

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Abstract

This page draws on the EFSA CONTAM 2020 update of the nickel risk assessment (EFSA Ni 2020), the ATSDR 2024 Toxicological Profile for Nickel (ATSDR Ni 2024), the NTP 15th Report on Carcinogens nickel chapter (NTP 15th RoC 2021), the EPA Ecological Soil Screening Levels for nickel (EPA Eco-SSL Ni 2007), and the LGC final report on nickel release from piercing post assemblies (LGC 2003).

Chapter-level cross-metal toxicology context for nickel toxicokinetics, contact dermatitis, nickel carbonyl poisoning, carcinogenicity, epigenetic effects, and treatment is connected from Ufelle & Barchowsky 2021.

1 | Overview

The toxicology of nickel splits sharply by route: inhalation exposure to nickel compounds (particularly soluble salts and certain insoluble forms in occupational settings) produces lung and nasal cancer, and the US National Toxicology Program classifies nickel compounds as a class as known human carcinogens (NTP 15th RoC); dietary exposure to nickel produces non-cancer endpoints, primarily reproductive and developmental effects at chronic exposure and systemic contact dermatitis at acute exposure in nickel-sensitized individuals (EFSA 2020). The EFSA CONTAM Panel 2020 update established a chronic TDI of 13 µg Ni/kg b.w./day and an acute reference using a LOAEL of 4.3 µg Ni/kg b.w. for eczematous flare-up reactions in nickel-sensitized humans, applying a margin-of-exposure approach for the acute case (EFSA 2020).

Dietary nickel sources are broadly distributed across the food supply: cocoa products, nuts, legumes (especially beans), oats, whole grains, and certain leafy vegetables carry the highest concentrations (EFSA 2020). Drinking water is a meaningful additional source (EFSA 2020).

2 | At a glance

Three facts that matter most for a consumer trying to interpret nickel exposure.

First, once a person is nickel-sensitized, very low oral nickel doses (4.3 µg/kg body weight, the EFSA acute LOAEL) can produce eczematous flare-up reactions called systemic contact dermatitis (EFSA 2020).

Second, dietary nickel exposure routinely exceeds the EFSA chronic TDI of 13 µg/kg/day in significant fractions of the European population, particularly among consumers of cocoa products, oat products, legume-heavy diets (including chickpea, soy, lentil), and certain leafy vegetables (EFSA 2020). Drinking water can be a meaningful additional contributor in some regions (EFSA 2020). The EFSA finding that exposure routinely exceeds the TDI is a regulatory-level acknowledgment that current dietary nickel exposure is at or above what the agency considers safe for chronic intake (EFSA 2020).

Third, the carcinogenic risk from nickel is dominantly an occupational inhalation concern (refining, electroplating, stainless steel manufacture), not a dietary concern (NTP 15th RoC). NTP and IARC classify nickel compounds as known human carcinogens based on lung and nasal cancer in occupational cohorts inhaling nickel dust and fume (NTP 15th RoC); dietary nickel intake at typical levels has not been associated with cancer outcomes (NTP 15th RoC). Consumers concerned about nickel cancer risk should attend to occupational exposure if relevant; for dietary nickel, the operative concerns are reproductive/developmental and (for sensitized individuals) acute dermatitis flare-ups (EFSA 2020).

3 | Toxicology

The EFSA 2020 chronic TDI of 13 µg Ni/kg b.w./day is anchored on a BMDL10 of 1.3 mg Ni/kg b.w./day for increased post-implantation loss in rats. Reproductive and developmental endpoints (post-implantation loss, decreased fetal weight, reduced viability) are the most sensitive chronic endpoints and dominate the dietary risk assessment. The chronic TDI was raised substantially in the 2020 EFSA update — from the 2.8 µg Ni/kg b.w./day of the 2015 opinion to 13 µg Ni/kg b.w./day — following a revised point of departure.

For acute oral nickel exposure, EFSA 2020 identified eczematous flare-up reactions in nickel-sensitized humans (systemic contact dermatitis) as the critical effect. A BMDL could not be derived from the available human-volunteer studies; the LOAEL of 4.3 µg Ni/kg b.w. was selected as the reference point, with a margin-of-exposure approach using MOE ≥ 30 as the threshold for low health concern (EFSA 2020).

Carcinogenic effects of nickel compounds are documented in occupational inhalation cohorts (nickel refinery workers, stainless steel and nickel-alloy production, electroplating) (NTP 15th RoC). Lung and nasal cancer are the primary tumor sites (NTP 15th RoC). The NTP 15th Report on Carcinogens (2021) classifies nickel compounds as a class as known human carcinogens (since the 10th RoC, 2002); metallic nickel as reasonably anticipated to be a human carcinogen (since the 1st RoC, 1980); and explicitly reviewed nickel alloys without recommending listing. Dietary nickel intake at typical levels has not been associated with cancer outcomes in epidemiological studies; the cancer risk from nickel is operationally an inhalation/occupational concern (NTP 15th RoC).

The dermal-contact pathway is separate from dietary nickel. LGC 2003 evaluates nickel sensitisation and allergic contact dermatitis from piercing post assemblies, including release from stainless steel into artificial sweat, urine, and blood plasma. The report is important for nickel sensitisation context, but it is not a food occurrence source and does not supply ppb values for infant formula, baby foods, or other Category 1 food rows.

Ufelle & Barchowsky 2021 supports the same route-gating principle at textbook level: nickel is characterized as a dietary/background exposure for the general population, an occupational inhalation concern for lung and nasal cancers, and a dermal immune concern for sensitized individuals. It does not supply food occurrence concentrations.

4 | Typical exposure routes

Dietary intake is the dominant route for the general non-occupationally-exposed population (EFSA 2020). Inhalation of airborne nickel is occupational (smelting, refining, electroplating, stainless steel manufacture, fluid-flux welding) and produces the cancer endpoints rather than the dietary endpoints (NTP 15th RoC).

Once sensitized, individuals are susceptible to systemic contact dermatitis from oral nickel doses well below the EFSA chronic TDI (EFSA 2020).

5 | Food sources

Primary occurrence data from Flyvholm et al. 1984 (2,221 food samples reviewed from the post-1969 AAS or PIXE literature, plus Danish National Food Institute analyses) establishes the foundational Ni-content rank order for foods. The ten highest-Ni foods by weighted mean concentration are:

RANK	FOOD	MEAN NI (µg/G)	RANGE (µg/G)	N
1	Cocoa	9.8	8.2-12	7
2	Soy beans	5.2	4.7-5.9	3
3	Soy products	5.1	1.08-7.8	7
4	Walnuts	3.6	(single sample)	1
5	Peanuts	2.8	1.6-4.9	2
6	Oats	2.3	0.33-4.8	37
7	Buckwheat	2.0	1.3-2.8	3
8	Bitter (dark) chocolate	1.9	1.3-2.7	7
9	Hazelnuts	1.8	0.66-3.3	12
10	Dried legumes	1.7	0.52-3.3	17

Notable lower-ranked foods include almonds (1.3 µg/g, n=5), pistachios (0.8 µg/g, n=1), milk chocolate (0.7 µg/g, n=11), whole wheat (0.33 µg/g, n=85), and white bread (0.27 µg/g, n=65). Within the modeled Danish average diet (2,099 g/person/day excluding drinking water), Flyvholm estimated 150 µg Ni/day total dietary intake, of which oatmeal alone contributed 14.1 µg/day at a striking load factor F = 24 (the highest single-food load factor in the diet). Wheat flour contributed 14.7 µg/day at F = 1.9, potatoes 24.4 µg/day at F = 2.0, and fats including margarine 4.2 µg/day at F = 5.8. Replacing average-diet items with high-Ni foods can push intake to 900 µg/day or more, well within the 600 to 5,600 µg per oral provocation range that triggers hand-eczema flare in nickel-sensitive patients.

MATRIX	NICKEL CONCERN

Cocoa and chocolate products	Highest-Ni food category at 9.8 µg/g cocoa, 1.9 µg/g bitter chocolate, 0.7 µg/g milk chocolate (Flyvholm 1984; EFSA 2020)
Oats and oat products	Elevated at 2.3 µg/g; oatmeal carries the highest dietary load factor F=24 in the Danish average diet (Flyvholm 1984; EFSA 2020)
Legumes (beans, chickpeas, lentils, soy)	Plant-family-level efficient Ni accumulators; soybeans 5.2 µg/g, soy products 5.1 µg/g, dried legumes 1.7 µg/g (Flyvholm 1984; EFSA 2020)
Nuts (walnuts, peanuts, hazelnuts, almonds, pistachios)	Elevated; walnuts 3.6 µg/g, peanuts 2.8 µg/g, hazelnuts 1.8 µg/g, almonds 1.3 µg/g, pistachios 0.8 µg/g (Flyvholm 1984; EFSA 2020)
Drinking water (in some regions)	Variable; cold tap water mean 9 µg/L (Danish data) but post-8-hour stagnation can reach 490 µg/L; corrosion of Ni-containing plumbing (Flyvholm 1984; EFSA 2020)

EFSA 2020 subsequently confirmed Flyvholm's rank ordering and found that mean dietary nickel exposure across European Member States routinely exceeds the chronic TDI of 13 µg/kg b.w./day, particularly among toddlers and other children, and among adults consuming legume-heavy or cocoa-heavy diets.

What this means for food choice

For consumers without diagnosed nickel sensitivity: dietary nickel exposure routinely exceeds the EFSA TDI, which is a regulatory finding rather than a personal-action signal. The reproductive and developmental endpoints driving the TDI matter most for women planning pregnancy and for child consumers; for adult men outside the reproductive window, the chronic TDI exceedance is less consequential at the individual level than the population level.

For consumers with diagnosed nickel-sensitivity contact dermatitis: the acute LOAEL of 4.3 µg Ni/kg b.w. corresponds to approximately 300 µg total daily nickel for a 70 kg adult (EFSA 2020).

6 | Regulatory limits

JURISDICTION / BODY	TYPE	VALUE	PAGE
EFSA (EU)	Chronic dietary TDI	13 µg Ni/kg b.w./day	efsa-nickel-tdi
EFSA (EU)	Acute oral LOAEL (sensitized)	4.3 µg Ni/kg b.w.; MOE ≥ 30 for low concern	efsa-nickel-tdi

EU	Reg (EU) 2024/1987 — binding food maximum levels (amending 2023/915, Annex entry 3.6)	0.10 mg/kg (liquid infant formula) to 40 mg/kg (wakame); e.g. infant formula powder 0.25, baby food excl. juices 0.50, processed cereal-based baby food 3.0, cereals 0.80; applies from 1 Jul 2025 (cereals 1 Jul 2026)	eu-2024-1987-nickel--maximum-levels
EU	Nickel Directive 94/27/EC (skin-contact items, as described in LGC 2003)	0.5 µg Ni/cm2/week release-rate limit for direct/prolonged skin contact; 0.05% m/m content limit for post assemblies; LGC recommendation: 0.2 µg/cm2/week migration limit for all post assemblies	eu-nickel-directive-94--27-ec
US NTP	15th Report on Carcinogens	Ni compounds: known human carcinogen; metallic Ni: reasonably anticipated	NTP 15th RoC 2021
US EPA	Ecological Soil Screening Levels	Threshold values for ecological-risk screening at hazardous waste sites	EPA Eco-SSL Ni 2007
US ATSDR	MRLs (multiple by route and duration)	See profile	ATSDR Ni 2024

What the reference values mean in practice

The two EFSA reference points operate together as the dietary nickel benchmark: the chronic TDI of 13 µg/kg/day for general-population reproductive/developmental risk, and the acute LOAEL of 4.3 µg/kg b.w. for nickel-sensitized contact dermatitis flare-up. For a 70-kilogram adult, the chronic TDI corresponds to 910 µg Ni/day; the acute LOAEL corresponds to 300 µg per single dose; the MOE of 30 for "low concern" sets the practical acute target at approximately 10 µg Ni in a single sitting for a sensitized individual.

Beyond the health-based TDI, the EU has set its first binding nickel maximum levels in food under Regulation (EU) 2024/1987 (amending 2023/915, Annex entry 3.6), with category limits ranging from 0.10 mg/kg for liquid infant formula to 40 mg/kg for wakame seaweed; most entries apply from 1 July 2025, and the cereal limits from 1 July 2026 (eu-2024-1987-nickel-maximum-levels).

Typical European dietary nickel intakes routinely exceed the chronic TDI (EFSA 2020).

For the population question: the EFSA finding that mean dietary nickel exposure exceeds the TDI is a regulatory signal that population-level nickel intake reduction is warranted, not that any individual at typical exposure faces a defined acute health threat (EFSA 2020). The TDI is a health-protective threshold for chronic intake assuming lifetime exposure; brief exceedance is not an acute danger (EFSA 2020).

7 | Testing

Nickel food occurrence values require food-matrix analytical methods and ppb basis matching. By contrast, dermal contact-product nickel release is commonly expressed as µg/cm2/week. The EN 1811 nickel-release method discussed in LGC 2003 is therefore relevant to piercing posts and skin-contact articles, not to food concentration cells.

8 | Microbiome effects

Nickel is an essential cofactor for at least five distinct virulence-associated enzyme systems in human and animal pathogens, documented in Maier and Benoit 2019 (University of Georgia, Center for Metalloenzyme Studies). The pathogen-Ni-virulence systems span gastric, urinary-tract, and central-nervous-system infections, with Helicobacter pylori as the canonical Ni-dependent gastric pathogen: H. pylori uses [NiFe] hydrogenases for H2--utilization-driven host colonization and uses urease for gastric pH neutralization, and both Ni-enzyme systems are required for the translocation of the carcinogenic CagA toxin into host epithelial cells. Salmonella enterica Typhimurium hydrogenases are characterized as host-colonization factors. Proteus mirabilis urease drives urolithiasis pathogenesis. Staphylococcus species ureases participate in soft-tissue infections. Cryptococcus genus urease enables blood-brain-barrier penetration in meningeal cryptococcosis. The pathogen Ni-uptake, Ni-storage, and Ni-maturation enzyme machinery is sophisticated enough to balance Ni availability against Ni toxicity.

Host nutritional immunity (calprotectin-mediated Ni and Zn sequestration) is the principal evolutionary counter to pathogen Ni acquisition (Maier and Benoit 2019). This is the mechanism through which the host gut starves invading pathogens of Ni; dietary Ni loading that overwhelms calprotectin's sequestering capacity is a candidate pathway by which heavy-metal exposure perturbs pathogen virulence beyond its direct host-toxicity endpoints. The EFSA chronic TDI of 13 µg Ni/kg b.w./day is calibrated against direct host endpoints (reproductive/-developmental) and does not address this microbiome-and-pathogenesis axis.

Nickel-microbiome interactions in non-pathogen contexts are documented in Yang et al. 2023 (Environmental Pollution n=109 Chinese cohort): occupational Ni exposure correlates with elevated serum uric acid via diminished uric-acid-lowering bacteria (Lactobacillus and related taxa) in the gut microbiome, with intestinal purine-to-uric-acid degradation impaired by Ni-driven microbiota perturbation. The Yang 2023 cohort-level human evidence complements the Maier and Benoit 2019 mechanistic pathogen-Ni-virulence review.

Cross-cutting metal-microbiome reviews Coryell et al. 2019, Zhu et al. 2024, and Ghosh et al. 2024 contextualize the Ni-microbiome interaction within the broader heavy-metal-and-microbiota literature. See nickel-microbial--pathogenesis for the dedicated synthesis page that crosswalks to WikiBiome.

9 | Vulnerable populations

POPULATION	BASIS
Nickel-sensitized individuals	Acute systemic contact dermatitis flare-up at very low oral nickel doses (4.3 µg/kg b.w. LOAEL) (EFSA 2020)
Frequent consumers of cocoa, oats, legumes, nuts	Chronic dietary exposure routinely exceeds the 13 µg/kg/day TDI (EFSA 2020)

Pregnant women	Reproductive/developmental endpoint anchors the TDI; pregnancy is the most sensitive life stage (EFSA 2020)
Children and toddlers	Higher per-kg dietary intake; routine TDI exceedance documented in EFSA 2020
Workers in nickel-related occupations	Inhalation exposure to nickel compounds (refining, electroplating, stainless steel manufacture) (NTP 15th RoC)

If you are in one of these groups

For pregnant women: the EFSA reproductive/developmental endpoint anchoring the chronic TDI applies most directly to this population (EFSA 2020). The TDI itself incorporates uncertainty factors for inter-individual variability; brief exceedance is not an acute danger, but sustained exceedance during pregnancy is the regulatory concern the reproductive/developmental TDI is anchored on (EFSA 2020).

10 | App-layer integration

Machine-readable takeaways from this synthesis for the Heavy Metal Index consumer app pipeline.

The reference-value structure for nickel is bimodal: a chronic TDI (13 µg Ni/kg b.w./day for general-population reproductive risk) and an acute LOAEL with MOE approach (4.3 µg Ni/kg b.w. for sensitized-individual dermatitis). The app should benchmark against the chronic TDI for non-sensitized users and against the acute LOAEL with MOE ≥ 30 for users flagged as nickel-sensitized.

Pediatric multipliers for nickel are through body-weight scaling. EFSA 2020 documents routine TDI exceedance in toddlers and children at the population level.

Structured outputs:

- Acute LOAEL (sensitized, oral): 4.3 µg Ni/kg b.w. (EFSA 2020).
- Acute MOE threshold for low concern: ≥ 30 (EFSA 2020).
- Chronic TDI: 13 µg Ni/kg b.w./day (EFSA 2020).
- High-Ni food categories for app dietary calculation: cocoa, chocolate, nuts (cashews, hazelnuts), oats, legumes (soy, chickpea, lentil, bean), leafy vegetables (spinach, lettuce), shellfish (EFSA 2020).

For nickel-sensitized users, the app should provide a "low-nickel diet" mode flagging high-nickel foods and offering substitutions. For non-sensitized users, the chronic TDI benchmark is the primary signal.

11 | Open questions

Two load-bearing open questions for nickel:

First, the population-level TDI exceedance reported by EFSA 2020 is a regulatory finding without a clear individual-level intervention pathway. Reducing dietary nickel below the TDI through ordinary food choice is difficult given nickel's broad distribution across plant foods and beverages. Whether this constitutes a population-health concern warranting policy intervention beyond the existing EU Nickel Directive on skin-contact items is an open question the wiki tracks as the regulatory landscape develops.

Second, the relationship between dermal sensitization (the gateway exposure for many sensitized individuals) and dietary flare-up tolerance is mechanistically established but quantitatively variable across sensitized populations. The individual-level oral nickel threshold at which a sensitized person experiences a flare-up is not fully characterized in the literature, so the dietary nickel concentration that would reliably benefit sensitized consumers cannot be specified from current evidence.

Third, LGC 2003 shows why dermal/contact-material evidence must be route-gated inside the wiki: a robust release-rate page for piercing post assemblies can coexist with Category 1 food data gaps without letting a skin-contact migration limit masquerade as a food occurrence value.

12 | Systemic Nickel Allergy Syndrome (SNAS) and the low-nickel diet

Systemic Nickel Allergy Syndrome (SNAS) is the clinical bridge between contact-allergy (the most-recognized nickel endpoint) and a much broader spectrum of systemic disease triggered by dietary nickel. SNAS affects approximately 20 percent of nickel allergic-contact-dermatitis patients and is defined by three diagnostic criteria per Braga et al. 2013: positive patch test to nickel, symptom improvement on a low-nickel diet, and positive oral nickel challenge (gold standard: double-blind placebo-controlled). The Braga 2013 BraMa-Ni structured low-nickel diet (~50 µg Ni/day, nutritionally balanced) achieves 94.4 percent sensitivity and 93.3 percent specificity for SNAS diagnosis, dramatically outperforming conventional forbidden-foods lists (51.1 percent / 44.2 percent).

The low-nickel diet has substantial clinical-trial evidence across a remarkable range of conditions. In dermatitis, Kaaber et al. 1978 (the foundational paper) found 9 of 17 oral-Ni-challenge-positive patients improved during 6-week low-Ni diet, with 7 of 9 flaring on return to normal diet. Veien et al. 1993 enrolled 90 nickel-sensitive patients and documented 64.4 percent short-term benefit, with 72.7 percent of responders sustaining improvement at 1-2 years follow-up. In IBS, Rizzi et al. 2017 found that nickel-sensitive IBS patients had intestinal permeability 2.7-fold elevated versus controls (5.91 percent vs 2.20 percent 51Cr-EDTA excretion) and that all GI symptoms except vomiting improved on a low-Ni diet. In GERD, Yousaf et al. 2021 documented 95 percent symptom-severity reduction (19 of 20 refractory GERD patients) at 8 weeks, with response independent of patch-test status. In recurrent aphthous stomatitis, Pacor et al. 2003 showed 45.7 percent of nickel-sensitive RAS patients had positive DBPC oral Ni challenge, with 21 of 32 improving on nickel-free diet (and 92.6 percent of the 380-patient RAS cohort reported chocolate/cocoa-aggravated symptoms, reinforcing the cocoa-as-high--nickel matrix). In endometriosis, Borghini et al. 2020 found 90.3 percent Ni allergic contact mucositis prevalence in endometriosis patients with GI symptoms, and a 3-month low-Ni diet significantly improved all 15 GI symptoms, all 7 extra-intestinal symptoms, and gynecological symptoms (dysmenorrhea, dyspareunia, pelvic pain). In H. pylori eradication, Campanale et al. 2014 showed a nickel-free diet added to standard triple therapy nearly doubled eradication rate (84.6 percent vs 46.2 percent, $p<0.01$) — direct in-vivo confirmation that disabling H. pylori's nickel-dependent urease and hydrogenase translates to clinical pathogen-clearance, consistent with the Maier and Benoit 2019 mechanism review.

Oral hyposensitization is an emerging alternative to lifelong dietary adherence. Minelli et al. 2010 enrolled 36 SNAS patients in a graduated oral nickel sulphate protocol (sub-nanogram to microgram doses over ~6 months) and documented both clinical improvement and significant cytokine reductions: IFN-gamma -55.3 percent, IL-13 -58.6 percent, IL-5 -31.2 percent. The dual-pathway downregulation (Th1 plus Th2) is consistent with classical oral-tolerance induction mechanisms.

13 | Clinical population implications

The clinical-trial cluster above expands the population for whom dietary nickel matters substantially beyond the EFSA TDI's reproductive/developmental endpoint and the EFSA acute LOAEL's contact-allergy framing:

CLINICAL CONDITION	POPULATION SCALE	LOW-NI-DIET EVIDENCE
Nickel allergic contact dermatitis	8-19 percent of adults; 13-18 percent of women	Kaaber 1978, Veien 1993
SNAS (subset of ACD)	~20 percent of ACD patients	Braga 2013, Minelli 2010
Refractory GERD	Subset of the ~20 percent adult prevalence; response not patch-test-gated	Yousaf 2021
IBS with Ni sensitization	Subset of the ~10 percent adult IBS prevalence	Rizzi 2017
Recurrent aphthous stomatitis with Ni reactivity	Subset of the 5-25 percent lifetime RAS prevalence	Pacor 2003
Endometriosis with GI / extra-intestinal symptoms	90.3 percent Ni ACM prevalence in symptomatic subset	Borghini 2020
H. pylori infection on triple therapy	Global; H. pylori colonizes ~50 percent of humans	Campanale 2014

The EFSA TDI framework calibrates against direct host endpoints in healthy populations. The clinical-trial cluster documents that dietary nickel modulates clinically meaningful endpoints across a population substantially larger than the EFSA framework recognizes, and that intervention via reduced dietary intake produces measurable response in most of these populations. The literature therefore identifies a population for whom dietary nickel matters that is wider than the reproductive/developmental and contact-allergy endpoints anchoring the EFSA reference values capture.

14 | Frequently asked questions

Which foods are highest in nickel?

Cocoa and chocolate products, soybeans and soy products, nuts (walnuts, peanuts, hazelnuts, almonds), oats, whole grains, buckwheat, and legumes (beans, chickpeas, lentils) carry the highest nickel concentrations (Flyvholm 1984; EFSA 2020). Cocoa is the single highest-nickel food at about 9.8 µg/g, and oatmeal carries the highest dietary load factor in the modeled Danish average diet. Drinking water can be a meaningful additional source in some regions, especially after water sits in nickel-containing plumbing (EFSA 2020).

Does dietary nickel cause cancer?

No. Nickel's carcinogenic risk is dominantly an occupational inhalation concern (refining, electroplating, stainless steel manufacture), where breathing nickel dust and fume causes lung and nasal cancer (NTP 15th RoC). The US National Toxicology Program classifies nickel compounds as known human carcinogens on that basis, but dietary nickel intake at typical levels has not been associated with cancer outcomes (NTP

15th RoC). For diet, the operative concerns are reproductive/developmental effects and, for sensitized people, contact-dermatitis flare-ups (EFSA 2020).

How much dietary nickel is considered safe?

EFSA set a chronic tolerable daily intake (TDI) of 13 µg nickel/kg body weight/day, anchored on reproductive and developmental effects, which works out to roughly 910 µg/day for a 70-kg adult (EFSA 2020). For nickel-sensitized individuals there is a separate acute reference point, a LOAEL of 4.3 µg/kg body weight (about 300 µg in a single dose for a 70-kg adult), with a margin of exposure of at least 30 for low concern. EFSA also found that average dietary nickel exposure across Europe routinely exceeds the chronic TDI, particularly among toddlers, children, and people eating legume- or cocoa-heavy diets.

Who is most vulnerable to nickel in food?

Nickel-sensitized individuals are most vulnerable because very low oral doses (the 4.3 µg/kg acute LOAEL) can trigger eczematous flare-up reactions called systemic contact dermatitis (EFSA 2020). Pregnant women are also a key group because the reproductive/developmental endpoint anchors the chronic TDI, and children and toddlers because their higher per-kg intake means routine TDI exceedance (EFSA 2020). Frequent consumers of cocoa, oats, legumes, and nuts, plus workers in nickel-related occupations exposed by inhalation, round out the vulnerable populations (NTP 15th RoC).

Are there legal limits on nickel in food?

Yes. The EU set its first binding nickel maximum levels in food under Regulation (EU) 2024/1987 (amending 2023/915), with category limits ranging from 0.10 mg/kg for liquid infant formula up to 40 mg/kg for wakame seaweed (eu-2024-1987-nickel-maximum-levels). Examples include infant formula powder at 0.25 mg/kg, baby food (excluding juices) at 0.50 mg/kg, and cereals at 0.80 mg/kg. Most entries apply from 1 July 2025, and the cereal limits from 1 July 2026. Separately, the EU Nickel Directive 94/27/EC limits nickel release from skin-contact items like piercing posts (eu-nickel-directive-94-27-ec).

Can a low-nickel diet help medical conditions beyond a skin rash?

Clinical-trial evidence links a low-nickel diet to improvement across several conditions in nickel-sensitive patients, framed under Systemic Nickel Allergy Syndrome (SNAS), which affects about 20 percent of nickel contact-dermatitis patients (Braga 2013). Reported benefits include reduced IBS symptoms (Rizzi 2017), refractory GERD symptom reduction (Yousaf 2021), improvement in recurrent aphthous stomatitis (Pacor 2003), and better outcomes in endometriosis patients with GI symptoms (Borghini 2020). A nickel-free diet added to standard therapy also nearly doubled H. pylori eradication rates (Campanale 2014).

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DATA AVAILABILITY

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