Palladium

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Abstract

Contact allergy in dental patients, environmental deposition from catalytic converters, and uncharacterized dietary exposure. The literature is dominated by allergy immunology and ecotoxicology; food occurrence data remain sparse.

This page draws on 31 peer-reviewed sources deposited in the HMI-METAL-PALLADIUM-1 evidence unit, plus prior context from Ufelle and Barchowsky 2021. The complete citation list is at the bottom of the page.

1 | Overview

Palladium (Pd, atomic number 46) is a platinum group element (PGE) discovered in 1803 and historically used in jewelry, dental alloys, chemical catalysts, and electrical contacts. Its industrial profile changed substantially in the 1970s when it was incorporated alongside platinum in automotive catalytic converters, making vehicle exhaust catalysts the dominant anthropogenic source of environmental Pd enrichment. This shift prompted the first systematic human biomonitoring and toxicology studies, all published in 1975 as the catalytic converter era began.

The 31 papers in this evidence deposit divide into four clusters: (1) environmental occurrence in air particulate, road dust, sediment, freshwater, urban vegetation, and food matrices; (2) contact allergy and dental exposure; (3) ecotoxicology in freshwater organisms; and (4) pharmaceutical impurity context. Food safety regulators do not currently set maximum levels for palladium in food in any major jurisdiction covered by this corpus.

2 | Sources and physicochemical context

Palladium belongs to the platinum group elements alongside platinum, rhodium, ruthenium, osmium, and iridium. PGEs share exceptional corrosion resistance, high melting points, and strong catalytic activity. Pd and Pt are the most soluble and mobile PGEs in environmental matrices, which explains their preferential transfer from road dust and sediment into aqueous phases and biota. The speciation of Pd as a function of pH shows that Pd(OH)2

predominates from pH 2 to 12, with hydroxyl complexes across the entire environmental pH range (Alaimo and Varrica 2023).

In road dust and air particulates, the Pd/Pt ratio exceeds 1 at most traffic-influenced sites globally (Kolkata, Moscow, Jeddah, Rome, Hong Kong, Beijing, Toronto), reflecting the dominance of Pd-rich catalytic converter formulations in the vehicle fleet (Diong et al. 2016, Ermolin et al. 2022). The nanoparticulate fraction of Pd in Moscow road dust is approximately 1.6-1.8%; the dissolved fraction in road dust leachates is 4-10%, meaning the remaining majority is microparticulate Ermolin et al. 2022.

3 | Environmental occurrence

Air particulate

Traffic-associated PM10 concentrations of Pd reflect the density of catalytic converter-equipped vehicles and local meteorology.

Diong et al. 2016 measured Pd, Pt, and Rh by ICP-MS in PM10 and PM2.5 from Kolkata traffic junctions and industrial sites during winter and monsoon seasons of 2013-2014. Traffic junction PM10 Pd ranged from 2.7 to 111 ng/m3; industrial site PM10 Pd ranged from 3.12 to 32.3 ng/m3. Mean winter traffic junction PM10 Pd was 10.8 ng/m3. At all locations and seasons, Pd exceeded Pt, which exceeded Rh, consistent with Pd-dominant converter catalysts. Most PGE mass was concentrated in the PM2.5 fraction. The strong Pd-Pt correlation at traffic junctions (R = 0.79-0.82) confirms catalytic converters as the common source; weaker correlations at industrial sites indicate mixed emission sources.

Kadi et al. 2020 measured PGEs in fractionated PM10 from three sites in Jeddah, Saudi Arabia, using tellurium and mercury co-precipitation followed by ICP-MS. Pd concentrations at site S3 ranged from 19.6 +/- 24.9 to 195.3 +/- 200.4 pg/m3. Concentrations varied by site and between working days and weekends, consistent with traffic as a primary driver.

Road dust and urban matrices

Ermolin et al. 2022 characterized Pd and Pt size fractionation in Moscow road dust. Pd mean concentration was approximately 50 ng/g dry weight, with a range up to 235 ng/g. Comparable to other major cities: Rome, Hong Kong, Shenzhen, Guangzhou, Beijing, and Toronto all show Pd exceeding Pt in road dust. Nischkauer and Limbeck 2015 found Pd 201-1230 ng/g in road dust from the Vienna area (summer and winter), analyzed by ICP-MS after ion-exchange pre-concentration.

Varela et al. 2023 demonstrated that physiological variables in urban tree leaves serve as proxies for PGE accumulation, with Pd and Pt measured alongside heavy metals in 50 trees in Logrono, Spain, confirming that vegetation bioaccumulation of PGEs tracks traffic exposure in urban settings.

Freshwater

Bluteau et al. 2025 characterized PGE biodynamics in freshwater systems in Quebec and reviewed environmental concentrations. Dissolved Pd in urban Quebec rivers was 0.085-0.29 ng/L, at or near the analytical detection limit. Urban river sediment Pd in Gothenburg, Sweden reached 155 +/- 73 ng/g. Internalized uptake rate constant for Pd in Chaoborus larvae was 3.5 +/- 0.5 mL/g/day; assimilation efficiency (AE) for Pd was 5.6-32.4%; the elimination rate constant was 0.69 +/- 0.19 per day. These biodynamics parameters position Pd as more rapidly eliminated than classic toxicants: for comparison, nickel ke is 0.19 per day and cadmium ke is approximately 0.011 per day.

Carle et al. 2025 report environmental sediment Pd concentrations of 0.3-90.8 ng/g dry weight (from the literature compilation), well below the toxicity thresholds established in their freshwater invertebrate testing.

Sajkowska and Lesniewska 2024 demonstrated that polyethylene microplastics in lake water (pH 7.63) sorb Pd(II) and Pt(IV), establishing microplastics as a secondary transport vector for PGEs in aquatic environments.

Sub-Saharan Africa and global South

Nkosi et al. 2024 reviewed technology-critical element (TCE) contamination data from Sub-Saharan Africa. PGE-mine-proximate water concentrations of Pd 0.002-0.011 ug/L were reported from South African platinum mine drainage. PGE bioaccumulation in Manta ray muscle and hepatopancreas included Pd at 0.407-0.972 ug/g wet weight (muscle) and 0.236-2.680 ug/g (hepatopancreas) in one study, and Pd nd-0.481 ug/g in fish liver from a separate study. Occupational inhalation exposure near Pd processing was noted as the highest-intensity human Pd exposure pathway; skin exposure mean of 0.008 ug/cm2 was reported from one African platinum processing context.

4 | Dietary and biological matrices

Edible mushrooms

Alaimo and Varrica 2023 measured Pd and Pt in 39 fruiting bodies of Boletus aereus collected from multiple wooded areas in Sicily with sedimentary and volcanic substrates and limited anthropogenic influence. All analyses were by ICP-MS (dry weight basis). Pd ranged from 0.31 to 3.09 ng/g dry weight; Pt ranged from 0.21 to 4.22 ng/g. The distribution was non-normal (Shapiro-Wilk $p < 0.05$). Bioconcentration factors (BCF) versus Sicilian soil were: Pd 2.4, Pt 5.9. BCF versus upper continental crust background was also greater than 1 for both elements, indicating the mushroom is a net accumulator from its substrate. Daily intake rate (DIR) at the worst-case scenario (maximum detected concentration), assuming 300 g fresh mushrooms (approximately 30 g dry), 50 kg adult body weight: 1.85 ug/kg body weight for Pd. The authors concluded that Boletus aereus does not accumulate significant Pd levels and that occasional consumption is safe; content is lower than in other food products reported in the literature. This is currently the only directly measured edible food matrix in this evidence deposit.

The same paper cited a literature estimate that almost half of human PGE exposure occurs through diet, while noting that published concentration data across common food matrices remain scarce.

Human biomonitoring

Johnson et al. 1975 conducted the first systematic human biomonitoring for Pd (and Pt) in populations near a freeway and in a high desert reference area in Southern California, with the explicit goal of establishing pre-catalytic-converter baselines before the 1975 model year. Blood, urine, hair, and feces were collected from three age groups (children, young adults, mature adults). All hair samples were below the detection

limit for Pd (LOD-based reporting value: <3.1 ug/g). All feces samples were below LOD for Pd (<0.001 ug/g). All composite blood samples were below LOD for Pd (<0.1 ug/100 mL). No detectable Pd was found in any individual or composite sample from either population. The same pattern held for Pt. Lead, by contrast, showed significant population-level differences between the freeway-adjacent and remote populations, confirming that the analytical approach would have detected exposure differences had Pd or Pt been measurable. These 1975 baselines predate widespread catalytic converter deployment and represent the floor against which post-deployment monitoring should be compared; a systematic post-deployment biomonitoring dataset equivalent to the Johnson 1975 study was not identified in this evidence deposit.

5 | Toxicology

Oral absorption and tissue distribution

Moore et al. 1975 conducted the foundational kinetics study using radiolabeled 103Pd given to rats by oral, intravenous, intratracheal, and inhalation routes. After a single oral dose, almost all 103Pd was excreted in the feces due to nonabsorption; oral bioavailability was substantially lower than for all parenteral routes. After IV dosing, Pd distributed primarily to kidney, spleen, and liver. The highest tissue retention was achieved by IV dosing; the lowest by oral dosing. After IV dosing in pregnant rats, a small amount of 103Pd was found in fetuses, establishing trans-placental transfer but only under conditions of complete systemic availability. Rats given Pd or Pt in drinking water (pH 3.0) were monitored for appearance, body weight, and water consumption. These 1975 data remain the foundational reference for Pd pharmacokinetics and are consistent with the general pattern for platinum group elements: poorly absorbed from the gut, efficiently cleared once systemically absorbed, with kidney as a primary accumulation organ.

Cardiovascular toxicity (parenteral)

Wiester 1975 administered soluble Pd compounds intravenously to surgically prepared unanesthetized rats (n = 41) and monitored ECG, aortic blood pressure, cardiac contractility (dp/dt), and respiration. The minimum effective IV dose was 0.4 mg/kg Pd as PdSO4, administered over 40 seconds via the femoral vein, which produced cardiac arrhythmias with consequential blood pressure reduction. Pd(NO3)2 and PdCl2 showed similar cardiovascular activity. To achieve an equivalent response, three times as much Pd was needed when given as (NH4)2PdCl4 or K2PdCl4, suggesting that the counter-ion affects acute bioavailability. Severe effects were observed at higher doses; the mild-effect mean was 0.4 +/- 0.2 mg/kg as PdSO4. A historical observation was also noted: Pd(OH)2 injected subcutaneously at 5-7 mg/day for 3 months (a mid-20th century obesity treatment) caused weight loss and fever, constituting an uncontrolled human parenteral exposure record. All dose-response data in this study are from parenteral routes; they establish a hazard from systemic Pd exposure but are not directly applicable to dietary risk assessment.

6 | Contact allergy: the principal clinical concern

Palladium allergy is primarily a contact hypersensitivity disorder and represents the most extensively characterized human health outcome in the current evidence base. The mechanism is a type IV (delayed-type) T-cell-mediated hypersensitivity; a type IVb (CD8+ T cell-driven) subclass is established for intraoral exposure in murine models. The principal route of human sensitization is contact with Pd-containing dental alloys (crowns, bridges, partial dentures) and, to a lesser extent, pierced jewelry with Pd-containing metalwork.

Prevalence

Sadrolvaezin et al. 2023 synthesized epidemiological data from multiple European and Israeli patch-testing series. Pd sensitivity as measured by patch test: 9% in mainland Britain, 9% in Turkey, 8.3% in unselected eczema patients in Austria, 7.4% in Germany, 13% in Israel, and 2% in Northern Ireland. The mean prevalence of allergy to Pd was 7.8% (range less than 1-19%) in dermatitis patients and 7.4% (range 1.3-13.9%) in dental patients. Monosensitivity to Pd (positive for Pd but not nickel): 0.2% in dermatitis patients and 0.5% in dental patients. Among young women, Pd allergy prevalence was 16.0% in 1995-1999, declining to 9.1% in 2012-2016; among older women, it increased from 7.4% to 13.6% over the same period. The review identifies the CD8+ T cell as the effector cell driving Pd sensitization, with stimulation of NKG2D receptors on CD8+ increasing IFN-gamma production and sensitization.

Tajima et al. 2025 conducted a 15-year retrospective analysis of 1085 patients at the Dental Metal Allergy Clinic, Tokushima University Hospital, Japan. 65.4% of patients tested positive for at least one metal allergen; palladium chloride was identified as one of the most common allergens alongside nickel sulfate, potassium dichromate, and cobalt chloride. Female patients comprised 78.4% of the study population. Patients with a history of earring-related inflammation had 81.3% metal allergy prevalence, significantly higher than the 60.4% in patients without that history (chi-square, $p < 0.001$). Among earring-history patients, 40.2% tested positive for both Pd and nickel, 6.5% only for Pd, and 16.1% only for nickel. Among non-earring-history patients, 9.5% tested positive for Pd and nickel together, 12.8% only for Pd, and 9.5% only for nickel. Earring-related inflammation reports at the clinic increased from 5.0% of patients in 2005 to 43.2% in 2020.

Linauskiene et al. 2017 reported patch test results from Lithuania: PdCl2 positive in 18.0% with the dental screening series and 16.7% with the metal series; sodium tetrachloropalladate positive in 7.0% (dental) and 8.5% (metal). 76.9% (10/13) of Pd-positive patients were also sensitized to nickel.

Cross-reactivity with nickel

The co-occurrence of nickel and palladium sensitization is a consistent finding across the literature. Kapp and Summer 2020 analyzed the DKG (German Contact Dermatitis Group) registry data from 2006-2016: of 1017 Pd patch-test-positive individuals, 782 (76.9%) also reacted to nickel, leaving approximately 235 monosensitized to Pd. Patients with concomitant nickel patch test reactions showed lymphocyte proliferation responses at lower Pd concentrations (below 3.13 x 10^-5 M) compared to Pd-monosensitized patients, suggesting that prior nickel sensitization lowers the threshold for Pd T-cell activation. The lymphocyte transformation test (LTT) and in vitro cytokine release assay were used to differentiate true Pd monosensitization from cross-reactive nickel/palladium allergy, an important distinction for clinical counseling about dental material selection.

Shigematsu et al. 2020 confirmed nickel-palladium cross-reactivity in a murine model of metal-induced allergic contact dermatitis. Mice sensitized with nickel and challenged with PdCl2 (nickel-palladium group) or sensitized with chromium and challenged with PdCl2 (chromium-palladium group) were compared with saline-sensitized Pd-challenged controls ($n = 5$ per group). Histological and immunohistochemical analysis of footpad T cells showed that nickel sensitization induces cross-reactivity with Pd challenge, with significant differences at 1, 3, and 7 days after the third challenge ($p < 0.05$, $p < 0.01$).

Immunological mechanism

Bordignon et al. 2008 characterized the cytokine profile of Pd-sensitized patients using ELISpot analysis of IFN-gamma and IL-10 producing peripheral blood mononuclear cells. Optimal stimulation concentrations for ELISpot were 2.5 ug/mL PdCl2 (versus 20 ug/mL nickel sulfate). Cytotoxic thresholds: Pd at concentrations above 25 ug/mL inhibited PHA-induced cytokine production by more than 25%. Five patients were confirmed as Pd

allergy cases (n = 2 Pd-ACD, n = 3 nickel-ACD for comparison). IL-10 co-production modulates Th1 development; the Pd response was characterized as a mixed Th1/Th2 cytokine pattern.

Nasu et al. 2023 developed a murine model of Pd-induced intraoral allergic contact mucositis (ACM) and established that the dominant effector mechanism is type IVb (CD8+ T cell) hypersensitivity. Oral mucosa swelling was measured from day 1 to day 7 after Pd challenge in ACM and ICM mice (n = 10 per group), with statistically significant swelling at multiple timepoints ($p < 0.05$, $p < 0.01$).

Matsubara et al. 2017 demonstrated that fexofenadine (a second-generation antihistamine) suppresses Pd-induced delayed-type hypersensitivity in a murine model at 5 and 10 mg/kg, with efficacy equal to 50 mg/kg. High-dose prednisolone (30, 50 mg/kg) caused weight loss and poor fur condition; fexofenadine did not, suggesting a better tolerability profile. This finding has clinical implications for managing Pd allergy symptoms in patients who cannot immediately undergo dental alloy replacement.

Zemelka-Wiacek 2022 reviewed state-of-the-art mechanisms of metal allergy including palladium, noting environmental exposure routes (jewelry, everyday metal items, mobile phones, leather, metal-rich food, implants) and the relevance of implant-related hypersensitivity.

Clinical cases: dental alloys and systemic dermatitis

Adachi et al. 2024 reported a case of Pd-induced systemic contact dermatitis from dental alloys and characterized the pH-dependent ionization mechanism that may drive sensitization. In vitro dissolution experiments showed that nearly all palladium was ionized from a Pd alloy specimen at pH 1.68 (gastric acid level), approximately 90% at pH 4.01 (phthalate buffer), and less than 10% at pH 7.20 (physiological). The complete-dissolution concentration was approximately 40 mg/L. This pH dependence means that dental alloys are exposed to acidic conditions during food digestion that substantially exceed the ionization fraction at neutral oral pH, potentially driving systemic Pd exposure in sensitized individuals.

Fujii 2017 reported a case of severe dermatitis attributed to nickel-palladium cross-reactivity from dental restorations, resolved after replacement of all Pd-containing alloys with Pd-free gold alloys (Aurofluid CPF, Au 71.5%). The case documents complete clinical resolution after dental alloy substitution.

Dental alloy corrosion and ion release

Milheiro et al. 2015 measured Pd ion release from two Pd-based dental alloys (Orion Argos, Pd-Ag; Orion Vesta, Pd-Cu) cast as crowns and disks, with and without polishing, in ISO 10271 lactic acid solution at 37 degrees C for 7 days. Release rates: 0.06-0.69 ug/cm2/week for cast and polished alloys. Unpolished Pd-Ag (Orion Argos) as-received: 0.69 ug/cm2/week; polished: 0.11 ug/cm2/week. For comparison, the EU Nickel Directive 94/27/EC limit for nickel in items inserted in pierced body parts is 0.4 ug/cm2/week (the paper applies a molecular mass correction to compare palladium and nickel releases on an equivalent basis). Some Pd-alloy formulations at some surface preparation states release Pd at rates approaching or exceeding this nickel threshold, which the authors identify as clinically significant given established nickel-palladium cross-reactivity. Polishing is thus an important mitigating factor in dental alloy Pd release.

Ellakany et al. 2022 and Ellakany et al. 2026 characterized cytotoxicity of Ag-Pd, nickel-chromium alloy, and commercially pure titanium (cpTi) alloy discs to human gingival fibroblasts (HGF) by MTT assay. Ag-Pd alloy MTT absorbance in DMEM: 0.316 (SD 0.006); in artificial saliva: 0.294 (SD 0.002). Cell viability: Ag-Pd in DMEM 77.6% (range 75.8-79.4%); in artificial saliva 65.3% (range 63.5-66.5%). cpTi showed the least cytotoxicity (DMEM 91.8%); nickel-chromium alloy the most (DMEM 67.5%). Ag-Pd demonstrated intermediate cytotoxicity

between cpTi and nickel-chromium alloy. The reduction in viability with artificial saliva versus DMEM for Ag-Pd is consistent with enhanced Pd dissolution under the lower pH conditions of simulated oral fluid.

Zhang et al. 2021 demonstrated that palladium chloride (2.5% PdCl2) and sodium tetrachloropalladate at standard patch-test concentrations caused localized cytotoxicity including apoptosis in skin ex vivo. Similar but less extreme findings were observed for Pd compared to some other metal salts. This finding complicates interpretation of patch-test positivity because cytotoxicity at the application site can mimic sensitization-driven responses.

7 | Ecotoxicology

Daphnia magna

Boukhari et al. 2025 characterized acute and chronic Pd toxicity to Daphnia magna. Acute 48-hour LC50: 52 +/- 2 ug/L (measured concentrations 2-110 ug/L); LC10: 33 +/- 3 ug/L. The protective effect of dissolved organic carbon (DOC) was substantial: at 0.8 mg C/L DOC, LC50 rose to 74 +/- 1 ug/L; at DOC concentrations of 1, 2, 5, and 8 mg C/L, no mortality occurred. Pd bioaccumulation in D. magna decreased by 30-40% with increasing DOC, with up to 50% reduction at the highest tested DOC concentration compared to controls. Chronic 15-day exposure: EC50 for offspring viability (most sensitive endpoint) was 1 +/- 0.1 ug/L; EC50 for total broods per female was 7 +/- 2 ug/L; parental LC50 was 14 +/- 2 ug/L. These values are orders of magnitude above ambient dissolved Pd concentrations in uncontaminated freshwater (0.085-0.29 ng/L in Quebec urban rivers), but above-background contamination near Pd-emitting point sources would narrow the margin.

Freshwater benthic organisms

Carle et al. 2025 tested acute and chronic Pd and Pt sediment toxicity in Chironomus riparius and Hyalella azteca. Acute sediment LC50 values (ug/g dry weight): Pd in H. azteca 1192 +/- 356; Pd in C. riparius 209 +/- 44. Platinum was more toxic: Pt H. azteca LC50 289 +/- 28; Pt C. riparius LC50 84 +/- 7. In chronic C. riparius exposure, Pd impacted survival, emergence, and female adult weight; emergence time was significantly elevated versus controls at all tested concentrations (29-705 ug/g dw). Environmental sediment Pd concentrations from the literature (0.3-90.8 ng/g dw) remain well below the acute LC50 values by four to six orders of magnitude. However, historical sediment data suggest that Pd in some European river sediments (Rhine) has increased with traffic growth, and continued catalytic converter use raises long-term enrichment concerns.

Hydra vulgaris and multiple species

Auclair et al. 2025 exposed Hydra vulgaris to 0.3, 1, 10, 32, and 100 ug/L PGE for 96 hours and characterized gene expression responses. At 10 ug/L, measured Pd concentration was 60% of nominal, indicating some sorption or precipitation. Recovery at 100 ug/L after 48 hours was 87% for Pd (versus 43% for Pt and 100% for Rh). The study established multi-species ecotoxicity benchmarks across Pd, Pt, Rh, Ru, and Ir for a freshwater invertebrate model organism.

8 | Pharmaceutical impurity context

Palladium is widely used as a catalyst in Suzuki-Miyaura cross-coupling, Buchwald-Hartwig amination, and other synthetic reactions used to manufacture active pharmaceutical ingredients (APIs). ICH guidelines set a Pd limit in oral drugs of 100 ppm (10 ug/day at the threshold). Chatzopoulou et al. 2022 conducted a pilot study quantifying residual Pd in lead-like pharmaceutical compounds after three reaction types and multiple purification steps. Residual Pd ranged from below the LOQ to approximately 5000 ppm depending on Pd source

and purification. 5 of 39 products exceeded 50 ppm; all five were below 100 ppm. The nitro reduction reaction with one Pd source produced below-LOQ residuals. This context is relevant to understanding pharmaceutical dietary exposure routes for Pd, which are entirely distinct from dietary exposure through food matrices.

9 | What remains uncertain

No food occurrence data are available in this deposit for high-consumption staple food matrices such as grain, vegetable, meat, dairy, fish, or infant formula. The edible mushroom data from Alaimo and Varrica 2023 represent the only directly measured food matrix, and their finding of low concentrations in Sicilian mushrooms from unpolluted soils cannot be generalized to mushrooms grown near traffic corridors or Pd-mining regions, nor to other food categories.

The 1975 Johnson et al. baseline established that Pd was undetectable in blood, urine, hair, and feces of Californians before catalytic converter adoption. No equivalent post-deployment biomonitoring dataset covering a general population with modern analytical methods was identified in this deposit, leaving the actual change in human body burden from automotive Pd emissions unquantified.

The oral and dermal allergy threshold for Pd in food is uncharacterized. The dental alloy dissolution data from Milheiro et al. 2015 and the pH-dependent ionization data from Adachi et al. 2024 establish that Pd ions are released from oral dental alloys and that gastric acidity substantially amplifies ionization; this exposure pathway is clinically meaningful for sensitized individuals but not quantified on a population basis.

No government jurisdiction in this evidence deposit has set a maximum level for Pd in food. Palladium is not among the HMT&C analyte panel (lead, total arsenic, cadmium, methylmercury, total mercury, inorganic arsenic, nickel, aluminum, hexavalent chromium, tin) and is not currently a certification analyte.

10 | Frequently asked questions

Is there detailed safety information about palladium in food or consumer products on this page?

No. This page is a source-map stub and does not yet contain substantive, palladium-specific findings. It notes only that palladium is listed among minor toxic metals for which toxicity has been described, per Ufelle & Barchowsky 2021.

What does the literature currently cited here say about palladium?

The single cited source, Ufelle & Barchowsky 2021, lists palladium among minor toxic metals for which toxicity has been described. That toxicology chapter does not provide a detailed palladium subsection, so the page carries this as source-map context only.

Why is this palladium page so short?

The page was created as a source-map stub during the re-ingest of Ufelle & Barchowsky 2021, which mentions palladium but does not devote a dedicated section to it. Substantive palladium-specific expansion awaits a dedicated ingest, so the page is marked with status "stub."

Which source is palladium information on this page based on?

It is based on a single source: Ufelle & Barchowsky 2021, the chapter "Toxic Effects of Metals" (Ch 23) in Casarett & Doull's Essentials of Toxicology, 4th ed. That chapter references palladium among minor toxic metals but does not detail it further.

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