

Vitamin D Status and Transplant Outcomes: Which Organs Benefit When Donor or Recipient 25(OH)D Is High?

No transplant type has proof from randomized trials that getting recipients above 30 ng/mL (75 nmol/L) improves graft or patient survival. The most consistent signals, all observational, come from kidney transplant (graft failure, rejection, infections) and from allogeneic HSCT, where a non-randomized trial found less chronic GVHD with cholecalciferol. Liver transplant shows a repeated link between low vitamin D and acute rejection. For heart and lung, the evidence is a few cohorts plus one null lung RCT. I found essentially no clinical data on donor vitamin D status for any organ.

TL;DR

- **Best-supported benefit (recipient side):** In kidney recipients, deficiency (≤ 20 ng/mL) is independently linked to about 43% more overall graft failure, 2.2-fold more death-censored graft failure and 3.3-fold more acute cellular rejection. But the randomized trials of cholecalciferol (VITALE, $n=536$; a Japanese 4,000 IU/day trial; VITA-D) showed no benefit for hard outcomes or eGFR. The one proven, trial-backed benefit is on bone (femoral-neck BMD). Paricalcitol, an active analog rather than nutritional vitamin D, lowers proteinuria.
- **HSCT and liver come next.** In HSCT, giving 1,000–5,000 IU/day of cholecalciferol from day –5 to +100 was followed by less chronic GVHD in a 150-patient sequential-cohort trial, with no effect on acute GVHD or survival. Low pre-transplant levels also predict chronic GVHD and CMV disease. In liver transplant, deficiency roughly doubles to triples the odds of acute cellular rejection across several cohorts. Heart (5-year mortality HR 2.08, mediated partly by CAV) and lung (more pneumonia and deaths) have only single-center observational data, and the only lung RCT (100,000 IU/month) was null for CLAD.
- **Donor vitamin D is an evidence gap.** I found no published study linking deceased or living organ donor 25(OH)D, or donor vitamin D or calcitriol treatment in the ICU, to recipient outcomes. Current ICU donor-management guidance covers thyroid hormone, corticosteroids and vasopressin, not vitamin D. The only donor-side data are genetic: HSCT donor VDR polymorphisms appear to change how well recipient vitamin D prevents GVHD.

Key Findings

Ranking by strength of evidence and effect size (benefit linked to higher vitamin D)

Rank	Organ	Donor evidence	Recipient evidence	Outcomes associated with higher D	Strength of evidence	Key sources
1	Kidney	None found	Large cohorts (n up to 2,504), multiple RCTs of cholecalciferol and paricalcitol, meta-analyses	Graft survival, acute rejection, CMV/BK/UTI, GN recurrence (observational); BMD (RCT/meta-analysis); proteinuria (paricalcitol RCT)	Moderate for association; RCTs null for hard outcomes and eGFR; positive for bone	WisARD (Kidney360 2025), Lee 2014, VITALE, Doi/Tsujita RCT (AJT 2021), Oblak 2018, BMC Nephrology 2026 meta-analysis
2	HSCT (not a solid organ)	Donor VDR genotype modifies effect; no donor 25(OH)D data	Prospective non-randomized dose-cohort trial (n=150); cohorts; small RCT on repletion	Less chronic GVHD; less CMV disease (observational)	Low-moderate; no randomized proof on GVHD or survival	ALOVITA (Caballero-Velazquez 2016), Carrillo-Cruz 2019, von Bahr 2015
3	Liver	None found	Several retrospective cohorts (n=82-528), one prospective controlled calcitriol study, one supplementation cohort	Less acute cellular rejection; less infection	Low-moderate (consistent but observational)	Sci Rep 2023 (n=335), Nutrients 2020 (n=528), Zhou 2019, Xing 2013

Rank	Organ	Donor evidence	Recipient evidence	Outcomes associated with higher D	Strength of evidence	Key sources
4	Heart	None found	One prospective cohort (n=327)	Lower 5-year mortality, lower CV death, less CAV	Low (single cohort, no RCT)	Gold et al., Am Heart J 2025
5	Lung	None found	One RCT (n=87, null); one retrospective cohort (n=125)	Less pneumonia, better survival (observational only)	Low; RCT null for CLAD, survival and rejection	Vos 2017 JHLT RCT; Ki 2024 Transpl Int
—	Pancreas/islet, intestine	None found	No human outcome studies found	Animal models only	Very low	Experimental VDR-agonist models
—	Cornea (not a solid organ)	None found	No human outcome studies found	—	None	—

What this means: The ranking reflects how consistent and large the observational associations are, not proven causation. No organ has RCT evidence that pushing recipients from, say, 20 to >30 ng/mL improves graft survival, patient survival, rejection or recovery time. The closest thing to interventional support for an immune benefit is the HSCT chronic-GVHD signal, and that trial was not randomized.

Details

1. Kidney transplant (recipient evidence)

Observational, post-transplant 25(OH)D:

- Wisconsin Allograft Recipient Database (WisARD), Kidney360 2025.** 2,504 recipients with a functioning graft at 13 months, with 25(OH)D measured 5–13 months after transplant. Median follow-up was 6.8 years, with 867 graft failures. (Ovid) Categories were deficiency ≤20 ng/mL, insufficiency 21–29 ng/mL and sufficiency ≥30 ng/mL (75 nmol/L). (PubMed) Compared with sufficiency, deficiency carried:
 - a 43% higher hazard of overall graft failure (95% CI 1.16–1.78); (PubMed Central)

- a 2.24-fold higher hazard of death-censored graft failure (1.60–3.12); (PubMed Central)
- a 2.10-fold higher hazard of infection-related death (1.37–3.21); (PubMed Central)
- faster eGFR decline. (Ovid)

Models were adjusted for donor type and age, delayed graft function, HLA mismatch, immunosuppression, prior rejection, proteinuria, albumin and eGFR. (PubMed Central)
This is the strongest single dataset directly comparing <20 with ≥30 ng/mL.

- **Other WisARD analyses from the same center:**
 - Each 1 ng/mL lower 25(OH)D was linked to 8% higher hazard of recurrent FSGS. Deficiency versus sufficiency gave a subdistribution HR of 5.26 for FSGS recurrence. (PubMed Central)
 - Deficiency was independently associated with late CMV infection. (Lippincott Williams &...)
 - Low 25(OH)D was associated with BK polyomavirus nephropathy. All of these patients were discharged on cholecalciferol 1,000 IU twice daily, so deficiency persisted despite routine supplementation. (nih)
 - Deficiency was associated with late-onset infections, especially UTIs in male recipients. (PubMed Central)
- **Acute rejection (Lee et al., Transplantation 2014).** Among 351 recipients, 61.5% were deficient (≤20 ng/mL). Biopsy-confirmed acute cellular rejection occurred in 10.2% of deficient versus 3.7% of sufficient patients, with an adjusted HR of 3.3. There was no association with CMV disease, BK nephropathy or 1-year graft function. (Lippincott Williams &...)
- **UTIs (Kwon et al.).** Mean 25(OH)D was 12.8 ng/mL and 41.7% of patients were deficient. Deficiency was an independent risk factor for UTI. (PubMed)
- **Early infections (2017 study).** A 2017 renal-transplant study, highlighted on VitaminDWiki's transplant overview page, reported 43% fewer infections within 90 days when 25(OH)D was >30 ng/mL. (vitamindwiki)
- **Berlin cohort (Frontiers in Medicine 2023).** In 600 stable deceased-donor recipients, 25(OH)D predicted graft loss but 1,25(OH)₂D did not. An accompanying meta-analysis agreed. (Frontiers) (Frontiers) This matters because it suggests the nutritional substrate, not the circulating hormone, carries the signal.

Randomized trials, cholecalciferol (nutritional vitamin D3):

- **VITALE (France, multicentre, double-blind).** 536 non-diabetic recipients with 25(OH)D <30 ng/mL were randomized 12–48 months after transplant to high or low dose: 100,000 IU or 12,000 IU every 2 weeks for 2 months, then monthly for 22 months. There was no difference in the composite of new diabetes, major cardiovascular events, cancer or

death, or in any individual component. (Oxford Academic) (ScienceDirect) Note that the control arm also got vitamin D, so this was a dose comparison, not a comparison against placebo.

- **Japanese incident-KTR RCT (Doi, Tsujita, Hamano et al., Am J Transplant 2021; bone secondary analysis in JBMR 2022).** In this single-centre trial, 193 recipients were randomized at 1 month post-transplant (180 completed) to 4,000 IU/day cholecalciferol or placebo for 11 months, starting from a median baseline 25(OH)D of 10 ng/mL.
 - No benefit on eGFR (between-group difference -0.7 mL/min/ 1.73 m², 95% CI -3.3 to 2.0 , $p=0.63$), histology or urinary kidney-damage biomarkers.
 - Unexpectedly, eGFR declined more with cholecalciferol in patients with eGFR <45 or UACR ≥ 30 mg/g. (ScienceDirect)
 - In a prespecified secondary analysis, it did reduce bone loss. (Oxford Academic) (PubMed)
- **VITA-D (Medical University of Vienna, single-centre, double-blind; Thiem et al., Trials 2009 protocol).** The protocol planned 200 deficient recipients (<50 nmol/L, i.e. <20 ng/mL) to receive 6,800 IU/day or placebo for 1 year, with MDRD-eGFR at 1 year as the primary endpoint. Later reviews report that it did not show renoprotective or immunomodulatory effects, or fewer infections. (EMJ)
- **Meta-analyses:**
 - BMC Nephrology 2026: 8 studies (2005–2025, 79–536 recipients each). Supplementation raised 25(OH)D and improved femoral-neck BMD T-score. PTH trended down but not significantly. (Springer) (PubMed)
 - Systematic Reviews 2025: 14 RCTs with 985 recipients; 8 of the 14 had high risk of bias. (Springer)

Active vitamin D analogs (not the same as raising 25(OH)D):

- **Paricalcitol versus placebo (Oblak, Transplant International 2018).** 168 proteinuric recipients received 2 µg/day for 24 weeks. The protein-to-creatinine ratio fell 49% more than with placebo ($P<0.001$), but mild hypercalcemia was more common. (PubMed)
- **Trillini, JASN 2015.** In a 43-patient crossover trial, paricalcitol lowered PTH and proteinuria and reduced bone loss, but also lowered eGFR. (PubMed) (LWW)
- **Paricalcitol versus calcifediol (93 de novo recipients).** PTH >110 pg/mL at 6 months occurred in 19.6% versus 36.2% ($P=0.07$). BMD did not differ. (PubMed Central)

These findings concern vitamin D receptor activation and PTH control. They should not be read as evidence about 25(OH)D >30 ng/mL.

Interpretation: Kidney transplant has the most robust association data, with consistent dose-response and multiple outcomes. Yet three RCTs of cholecalciferol failed on eGFR or hard

endpoints. Reverse causation and confounding are plausible explanations: sicker, less mobile, proteinuric, sun-avoiding patients with more inflammation have lower 25(OH)D. The trials may also have been underpowered, too short, or started too late (VITALE began 12–48 months after transplant). The only benefit backed by trials is bone.

2. Liver transplant (recipient evidence)

- **Iran (Scientific Reports 2023).** 335 cirrhotic recipients with a mean pre-transplant 25(OH)D of 21.5 ng/mL. Pre-transplant deficiency was independently associated with acute cellular rejection (adjusted OR 2.69, 95% CI 1.50–4.68). ([Nature](#))
- **Nutrients 2020.** 528 recipients (2014–2019), 55% deficient (<20 ng/mL) before transplant. 25(OH)D correlated inversely with MELD-Na, ([nih](#)) and survival and ACR were analyzed by pre- and post-transplant supplementation status. ([DOI](#)) This inverse correlation with MELD is the core confounding problem: sicker livers make less 25(OH)D.
- **Zhou et al., International Immunopharmacology 2019.** 141 adult recipients. Deficiency was an independent risk factor for T-cell-mediated rejection. Supplemented deficient recipients had less rejection and infection, along with higher Treg and memory T-cell counts. ([DOI](#)) ([DOI](#))
- **Other studies:**
 - Zhou et al., as cited in the 2023 Scientific Reports cohort, found pre-transplant 25(OH)D >25 ng/mL associated with less ACR within 30 days.
 - Bitetto et al. (Liver International 2010; 133 recipients, median pre-transplant 25(OH)D 12.5 ng/mL) linked very low levels (<5 ng/mL) to moderate or severe ACR within two months of transplant.
 - Xing 2013 was a prospective controlled study of calcitriol (active form) that reported less ACR. ([DOI](#))
- **Pediatric (Children 2021).** Of 82 children, 65% were insufficient before transplant and 32% developed T-cell-mediated rejection. Insufficiency was associated with rejection in the first year, especially in cholestatic disease. ([DOI](#))
- **Trials:** A trial of 25-OH-vitamin D3 (calcifediol) in liver recipients is registered as NCT04111146. ([clinicaltrials](#)) I found no published results.

Interpretation: The link between low vitamin D and acute rejection is reproducible, and the effect size is moderate (OR/HR about 2–3). But most thresholds are <20 or <25 ng/mL, not >30. No RCT of cholecalciferol with clinical endpoints has been published.

3. Heart transplant (recipient evidence)

- **Gold et al., American Heart Journal 2025 (Emory).** 327 prospectively followed heart recipients, 29.1% deficient. ([PubMed Central](#))

- Deficiency doubled 5-year all-cause mortality (HR 2.08, 95% CI 1.20–3.60), driven mostly by cardiovascular death (SHR 2.21). ([PubMed Central](#))
- After full adjustment, deficiency was associated with a 3.1-fold higher likelihood of cardiac allograft vasculopathy in a 248-patient subgroup, and each 10 ng/mL increase in 25(OH)D with a 27% lower likelihood of CAV.
- Causal mediation analysis attributed 26.5% of the vitamin D–cardiovascular-death link to CAV grade. ([PubMed Central](#))
- **Mechanistic caveat:** In mice, immune-cell VDR deficiency did not change cardiac allograft survival. ([PubMed Central](#)) A tolerance effect in the heart is therefore not established.
- **VitaminDWiki:** The site's transplant overview shows a heart-transplant survival-versus-calcitriol figure. ([vitamindwiki](#)) It concerns the active hormone and should be read as hypothesis-generating.

4. Lung transplant (recipient evidence)

- **RCT, null (Vos et al., JHLT 2017, Leuven).** 87 bilateral lung recipients received cholecalciferol 100,000 IU monthly (n=44) or placebo (n=43) for 2 years. 25(OH)D rose significantly, but there was no difference in: ([PubMed](#))
 - CLAD prevalence at 3 years (p=0.7); ([PubMed](#))
 - CLAD-free survival (p=0.7); ([PubMed](#))
 - survival, acute rejection, lymphocytic bronchiolitis, infection, lung function, inflammation or BMD (all p>0.05). ([PubMed](#))
- **Cohort (Ki et al., Transplant International 2024, Seoul).** 125 recipients, 40.8% deficient (<20 ng/mL) at 3–9 months after transplant. ([nih](#))
 - Deficiency was associated with more pneumonia and higher mortality, and the gradient held across tertiles (cutoffs 18.2 and 24.5 ng/mL). ([nih](#))
 - Patients with higher cumulative supplementation had better prognosis. Estimated daily doses were about 871, 1,685 and 1,884 IU across the supplementation tertiles, and the recorded supplements included both cholecalciferol and calcitriol. ([nih](#)) ([PubMed Central](#))
 - VitaminDWiki summarized this study as "Lung transplant survival 4X more likely if take 2,000 IU of vitamin D daily". ([VitaminDWiki](#)) That is observational and confounded by indication, so the headline overstates what the design can show.
- Even the top tertile was below 30 ng/mL (median only around the mid-20s), so the lung data say little directly about the >30 ng/mL threshold.

5. Pancreas/islet and intestine

I found no human clinical studies relating recipient or donor 25(OH)D, or vitamin D supplementation, to pancreas, islet or intestinal graft outcomes. Reviews cite experimental work in which VDR agonists, usually combined with low-dose cyclosporine, delayed rejection of islet and small-bowel allografts. (Ovid) That is animal evidence only.

6. HSCT / bone marrow transplant (labeled separately; not a solid organ)

- **ALOVITA trial (Caballero-Velazquez, Clinical Cancer Research 2016; NCT02600988).** 150 allo-HSCT patients in three consecutive, non-randomized cohorts of 50: no vitamin D, 1,000 IU/day or 5,000 IU/day, given from day -5 to day +100. (American Association...)
 - No differences in acute GVHD (overall or grade 2-4), relapse, non-relapse mortality or overall survival. (American Association...)
 - Chronic GVHD at 1 year was significantly lower with vitamin D: 37.5% overall and 19.5% moderate-to-severe in the low-dose group, and 42.4% overall in the high-dose group, versus controls. (American Association...)
 - 1,000 IU was not worse than 5,000 IU.
- **Donor genetics (Carrillo-Cruz, Clinical Cancer Research 2019).** Patient and donor VDR polymorphisms (FokI, BsmI, ApaI, TaqI) modified the GVHD benefit. (PubMed) This is the only "donor" vitamin D evidence I found for any transplant type.
- **Cohorts:**
 - Karolinska (von Bahr, Bone Marrow Transplantation 2015): 166 patients with a median pre-transplant 25(OH)D of 42 nmol/L (about 17 ng/mL). Low vitamin D independently predicted chronic GVHD, and no CMV disease occurred in patients who were sufficient before transplant. (PubMed)
 - Glotzbecker et al. (Bone Marrow Transplantation 2013) found 2-year chronic GVHD in 63.8% of 53 myeloablative HSCT patients with pre-transplant 25(OH)D <25 ng/mL versus 23.8% of those at ≥25 ng/mL (P=0.009).
 - A Nordic nutritional RCT found no link between vitamin D and acute GVHD, but less chronic GVHD with supplementation. (PubMed Central)
- **Repletion trials:**
 - A British Columbia RCT (n=74) tested a 100,000 IU loading dose before transplant plus 2,000 IU/day. It raised the share of patients at ≥75 nmol/L by day 30 (89.7% versus 74.3%, p=0.08) but did not test GVHD. (nih)
 - An oral thin-film cholecalciferol pilot (n=20) targeted >35 ng/mL in patients who had not responded to standard repletion. (DOI)
- **Guidance:** A 2026 clinical-guidance review recommends screening and repletion to at least 30 ng/mL by day 30 as low-risk supportive care "pending definitive evidence".

7. Corneal transplant

I found no clinical studies relating donor or recipient vitamin D status to corneal graft rejection or survival.

8. Donor vitamin D status: what exists and what doesn't

- **Deceased donors:** I found no study measuring brain-dead or DCD donor 25(OH)D and relating it to delayed graft function, primary graft dysfunction, rejection or survival in any organ. I also found no trial giving vitamin D or calcitriol to donors. ICU donor-management reviews (Intensive Care Medicine 2019 and 2024) address hemodynamics, thyroid hormone (a large RCT in 838 potential heart donors found no increase in hearts procured), corticosteroids and vasopressin. (Springer) (Springer) None mentions vitamin D.
- **Living donors:** No outcome studies found.
- **HSCT donors:** Only VDR genotype data (ALOVITA/Carrillo-Cruz); no donor 25(OH)D outcome data found.
- **Limitation:** A planned deep-dive into donor-side literature could not be completed. This section reflects the searches above, and small or non-English reports may exist.

Mechanistic rationale (brief, where it supports clinical data)

Immune cells express the vitamin D receptor and CYP27B1, so circulating 25(OH)D can be activated locally. (PubMed Central) (PubMed) 1,25(OH)₂D inhibits T-cell proliferation and IL-2 and IFN-γ production, shifts Th1 and Th17 responses toward Th2, keeps dendritic cells tolerogenic and induces Tregs through IL-10. (PubMed Central +2) This fits the clinical pattern: the signals are strongest for T-cell-mediated outcomes (acute cellular rejection in kidney and liver, chronic GVHD) and for infections (antimicrobial peptides). In rats, 1,25(OH)₂D₃ prevented acute kidney rejection and chronic allograft nephropathy. (nih) Vitamin D's suppression of the renin-angiotensin system and its anti-fibrotic effects may explain the eGFR and CAV associations, (nih) but no trial has confirmed a clinical anti-fibrotic benefit.

Recommendations

1. **Recipients: aim for sufficiency, but for bone and infection risk rather than proven graft survival.** In kidney, liver and HSCT recipients, measuring and correcting deficiency (<20 ng/mL) with daily cholecalciferol is low-risk. It is supported by bone RCTs and consistent observational signals. A target of ≥30 ng/mL is reasonable, and it is the threshold used by WisARD, VITALE and the 2026 HSCT guidance. Be honest that no RCT shows better graft survival above 30 ng/mL.
2. **Prefer daily or weekly cholecalciferol over large monthly boluses.** Both null immune trials (VITALE and the lung RCT) used 100,000 IU boluses. The positive HSCT signal

came from daily dosing of 1,000–5,000 IU started before transplant. Monthly bolus dosing is a plausible reason trials underperform, though this has not been proven in transplant patients.

3. **Start early.** Signals cluster around peri-transplant levels (pre-transplant in liver and HSCT, the first year in kidney). VITALE started 12–48 months after transplant.
4. **Don't conflate forms.** Paricalcitol and calcitriol act on PTH, proteinuria and bone, and they carry hypercalcemia risk. They are not a way to raise 25(OH)D. In the Japanese RCT, cholecalciferol lowered eGFR in recipients with low eGFR or albuminuria, so monitor eGFR and calcium.
5. **For VitaminDWiki editors:** Present kidney and HSCT as the best-supported organs, liver as moderate, and heart and lung as preliminary. Present donor vitamin D as essentially unstudied, and therefore a research opportunity: a cheap donor 25(OH)D assay at procurement, linked to delayed graft function and primary graft dysfunction in registries. Headline claims such as "4X lung survival" or "vitamin D helps organ transplant" should carry observational caveats.

Caveats

- **Confounding and reverse causation:** 25(OH)D tracks liver synthetic function (MELD), proteinuria (loss of vitamin D-binding protein), inflammation, frailty, sun avoidance (advised to prevent skin cancer), race and season. (Ovid) (nih) Adjustment is incomplete.
- **Threshold heterogeneity:** Cutoffs of <5, <10, ≤20, <25, <50 nmol/L and ≥30 ng/mL are used interchangeably across studies. Few studies test >30 versus 20–30 ng/mL directly. WisARD is the main exception.
- **Null RCTs:** VITALE (n=536), the Japanese 4,000 IU/day RCT, VITA-D and the lung RCT (n=87) all failed on their primary immune or graft endpoints. The HSCT chronic-GVHD benefit comes from a non-randomized sequential-cohort design.
- **Outcomes with no usable data:** I found no transplant-specific data on skin cancer, post-transplant diabetes (VITALE's composite, which included diabetes, was null), length of stay or recovery time. Delayed graft function appears only as a covariate.
- **Secondary sources:** VitaminDWiki summaries and some review-level statements (e.g., the VITA-D results) were used where primary full texts could not be retrieved.