



ISSN : 2347 - 2243

Indo - American Journal of Life Sciences and Biotechnology



<https://iajlb.org>

Email : editor@iajlb.com or iajlb.editor@gmail.com



Neonatal Hypocalcaemic Seizures Secondary to Maternal Vitamin D Deficiency: A Narrative Review

Michal Kaczmarek¹●, Sofia Theodorou²●, Henrike Oberdorfer³●

¹Associate Professor, Department of Pulmonary Medicine, Kashubian Institute of Medical Sciences, Kartuzy, Poland.

²Consultant Physician, Department of General Medicine, Epirus Institute of Biomedical Sciences, Ioannina, Greece.

³Senior Resident, Department of Pulmonary Medicine, Hunsrueck Institute of Biochemistry, Simmern, Germany.

Email: ¹m.kaczmarek@kims.edu.pl, ²s.theodorou@eibs.gr, ³h.oberdorfer@hib-s.de

ORCID: ¹0000-0001-4110-9107, ²0000-0001-2471-0371, ³0000-0001-4153-7777

Received: April 9, 2022 Accepted: July 21, 2022 Published: December 3, 2022

DOI: [10.62644/iajlb.2022.v19.i4.pp11-18](https://doi.org/10.62644/iajlb.2022.v19.i4.pp11-18)

ABSTRACT

Seizures in the first weeks of life have many causes, and low blood calcium is one that can be corrected quickly once it is recognised. When the mother was severely short of vitamin D during pregnancy, her infant may be born with small vitamin D stores and may become hypocalcaemic after birth, sometimes presenting with jitteriness, seizures or, less often, cardiac failure. This narrative review sets out what the published literature says about that chain of events. It covers the physiology of calcium supply before and after birth, the reasons maternal deficiency arises, the clinical picture and differential diagnosis, and the evidence for prevention through supplementation in pregnancy. It reports no patient data and no new measurements. The literature shows a plausible and well-described mechanism, but most direct evidence comes from case reports, small series and surveillance studies, and several features complicate causal interpretation: the fetus is protected from mild maternal deficiency, other causes of neonatal hypocalcaemia can coexist, and laboratory assays and diagnostic thresholds differ. Trials of supplementation in pregnancy support higher maternal vitamin D status, yet their effect on neonatal calcium outcomes is less clearly established than their effect on blood levels. The review closes with the gaps that matter most for future work.

Keywords: Neonatal hypocalcaemia; Neonatal seizures; Maternal vitamin D deficiency; 25-hydroxyvitamin D; Pregnancy supplementation; Narrative review

Citation: Kaczmarek, Theodorou and Oberdorfer [2022]. Neonatal Hypocalcaemic Seizures Secondary to Maternal Vitamin D Deficiency: A Narrative Review. *Indo-Am. J. Life Sci. Biotechnol.* 19(4):11-18. DOI: [10.62644/iajlb.2022.v19.i4.pp11-18](https://doi.org/10.62644/iajlb.2022.v19.i4.pp11-18)

Article Class: Narrative Review

1. Introduction

A newborn who starts to twitch or seize in the first days or weeks of life sets off a long list of possibilities, among them lack of oxygen at birth, infection, low blood glucose, structural brain disease and

metabolic disturbance (1). Low blood calcium is on that list, and it is one of the few causes that can be put right within hours. When it is missed, the baby may be treated for something else while the real problem continues.



A particular route to neonatal hypocalcaemia runs through the mother. Vitamin D reaches the fetus almost entirely as 25-hydroxyvitamin D carried across the placenta, so a mother who is severely and persistently deficient may give birth to an infant with very small stores (2, 3). Reports from several countries describe such infants presenting with seizures, and sometimes with heart failure, in the early weeks (4, 5).

This article asks what the published literature actually establishes about that chain, from maternal deficiency to neonatal seizure. It is a narrative review. The title reads like a single clinical case, but no patient is described and no local data are reported; we consider the topic in the literature at large, and say plainly where the evidence is thin. The aim is a fair account of what is known, and of what is merely repeated.

2. Literature Review

2.1 Calcium and Vitamin D Before and After Birth

Vitamin D is obtained mainly from skin synthesis driven by ultraviolet light, with a smaller contribution from food and supplements. It is converted in the liver to 25-hydroxyvitamin D, the form measured in blood, and then in the kidney and other tissues to the active hormone (6, 7).

The fetus lives in a calcium-rich environment. Calcium is moved actively across the placenta, fetal blood calcium runs higher than the mother's, and fetal parathyroid hormone is kept low (8). At birth that supply stops. The newborn must switch quickly to its own regulation, using parathyroid hormone, the kidney, bone and, within days, intestinal absorption from milk. A slow or immature parathyroid response, tuned by the calcium-sensing receptor, leaves the infant exposed for a time (9).

Here the literature contains a complication that is easy to overlook. Work on fetal mineral homeostasis suggests that calcium delivery to the fetus is maintained surprisingly well even when maternal vitamin D is low, so mild or moderate maternal deficiency need not cause neonatal hypocalcaemia on its own (8, 10). The problem appears when deficiency is severe and prolonged, or joined by other pressures on the newborn's calcium balance; it is best seen as a contributor that lowers the margin, not a sufficient cause in every case. Timing matters here as much as the size of any fall.

2.2 Why Mothers Become Deficient

Skin synthesis depends on sunlight, and so on latitude, season, time outdoors, clothing and the amount of pigment in the skin (11, 12). Deficiency has been reported in sunny regions as well as in northern ones (13, 14, 15, 16). Studies of pregnant women and their newborns in northern cities have found low levels in both (17). Such differences look small on paper, but across a population they add up.

Few foods contain much vitamin D, and breast milk from a deficient mother is itself low in it. Surveillance of rickets in Canada found that affected children were typically breastfed, unsupplemented and often darker skinned (18), and an international review noted that infant status follows maternal status closely in the first months (19, 20, 21).

2.3 How Hypocalcaemia Arises in the Newborn

Neonatal hypocalcaemia is commonly divided into an early form, in the first days, and a late form after the first week. The early form is mostly a problem of preterm infants, babies who were stressed at birth and infants of diabetic mothers; the late form is linked to the phosphate load in some milks, low magnesium,



hypoparathyroidism and vitamin D deficiency (8, 22). Table 1 sets out these groups and shows where maternal deficiency fits.

The boundary is blurred in practice. A deficient infant may be hypocalcaemic in the first days if other pressures are present, or only later, once stores are exhausted and the demands of growth rise (3, 24). Genetic forms of low parathyroid function can look the same and need different care (23).

Table 1. Main groups of neonatal hypocalcaemia and where maternal vitamin D deficiency fits.

Group	Typical setting	Mechanism
Early onset	Preterm birth, perinatal stress, maternal diabetes (8)	Abrupt loss of placental calcium; immature hormones
Late onset	Days to weeks after birth, often fed on milk with a high phosphate load (22)	Phosphate load, low magnesium, slow parathyroid
Maternal vitamin D deficiency	Severe, prolonged deficiency in the mother (3, 4)	Low stores; poor calcium uptake and release
Low parathyroid function	Genetic or syndromic, or after maternal hyperparathyroidism (23)	Too little parathyroid hormone action

Note: Qualitative summary of the cited reviews; groups overlap and more than one cause may act in the same infant.

2.4 Clinical Picture and Differential Diagnosis

Low ionised calcium makes nerve and muscle cells more excitable, so the presenting features are varied: some infants are jittery, some seize, and a few present with stridor, a prolonged QT interval or cardiac failure (4, 5, 22). Table 2 lists features by system. This is one reason the same condition can look so different from one report to the next.

Low calcium has to be searched for, not assumed. The current classification of neonatal seizures stresses that the underlying cause must be identified and treated alongside the seizure itself (1). A sensible work-up checks glucose, calcium, magnesium and markers of infection together, and asks about the mother's history and the infant's feeding. A known maternal deficiency is a useful pointer, though seldom decisive on its own. It is a habit worth keeping even when the answer seems obvious. Figure 1 summarises, in schematic form, the checks usually relevant to the common causes.

Table 2. Features that can accompany hypocalcaemia in a young infant.

System	Possible feature	Why it matters
Nervous system	Jitteriness, seizures, irritability (1, 4)	Mimics other neonatal seizure causes
Cardiac	Prolonged QT interval, heart failure (5, 22)	Can dominate the picture and delay diagnosis
Skeletal	Rachitic changes on imaging in severe cases (24, 25)	Points to a longer-standing deficiency

Note: Features are drawn from the cited clinical reviews and case reports; none is specific to vitamin D deficiency alone.

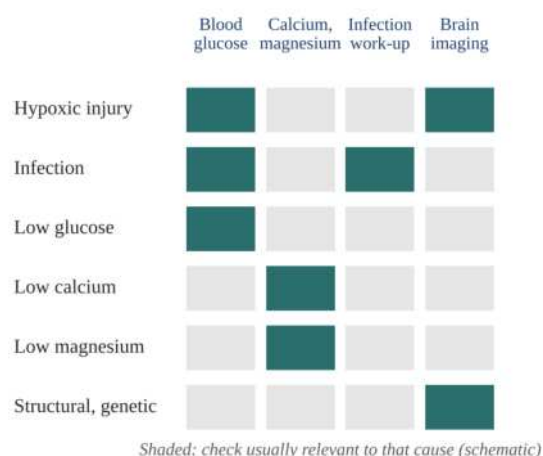


Figure 1. Matrix of common causes of neonatal seizures and the checks usually relevant to each (schematic, not data).



3. Materials and Methods

3.1 Scope and Question

The question was: what does the published literature establish about neonatal hypocalcaemic seizures that follow maternal vitamin D deficiency, and how firmly? The scope covered physiology, presentation, differential diagnosis and prevention through supplementation. Questions of individual treatment were left to clinical guidelines.

3.2 Literature Assembly

This is a narrative synthesis. No formal systematic search, protocol or registered strategy was used. Sources were chosen by the authors from the published literature on vitamin D, mineral metabolism, neonatal medicine and supplementation trials, favouring guidelines, systematic reviews, randomised trials and well-cited clinical reports published up to 2022.

3.3 Appraisal Framework

We asked of each source whether a report described how deficiency was measured, whether other causes of hypocalcaemia were considered, whether the study could support a causal reading or only an association. None of these steps is difficult, but each one is easy to skip. No numerical results were extracted and no data were pooled.

4. Results

4.1 What the Literature Shows About the Link

The mechanism is coherent and well described. Maternal deficiency lowers placental transfer of 25-hydroxyvitamin D, neonatal stores are small, and the infant's adaptation to extra-uterine calcium regulation is impaired (2, 26). Figure 2 sets out the chain and its modifiers. Case reports describe infants with seizures, low calcium and deficient mothers (4), and national surveillance in the United

Kingdom and Ireland identified hypocalcaemic seizures from deficiency as a recognised, if uncommon, presentation (27).

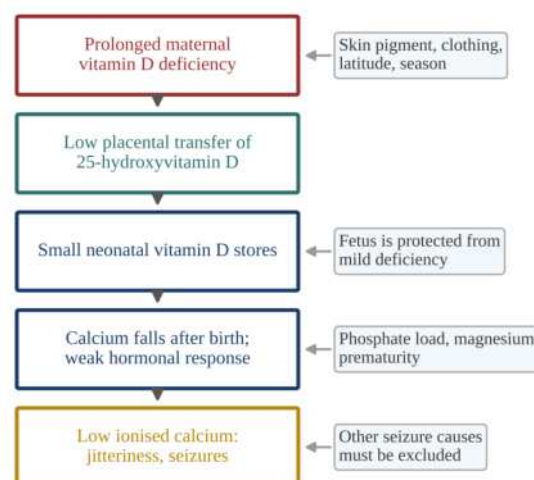


Figure 2. Proposed chain from maternal deficiency to neonatal seizures, with modifying factors (schematic, not data).

Recorded rates of deficiency also depend on how often clinicians test for it, which complicates comparisons over time (28). Read this way, one maternal measurement says little about the risk to the next infant.

4.2 Timing and Course

Calcium supply changes sharply at birth, as Figure 3 shows in schematic form. The infant of a deficient mother has a weaker regulatory response once the placental supply ends, so the dip may be deeper or longer, and the early diet and the infant's own parathyroid function also shape it (22, 24).

4.3 Prevention in Pregnancy and Infancy

Prevention rests on maternal vitamin D status. Guidelines recommend supplementation for pregnant women at risk and a daily supplement for infants, especially breastfed ones (29, 30, 31). Population intakes in the Institute of Medicine report may fall short for



higher-risk groups (32, 33). Table 3 summarises how the measures compare.

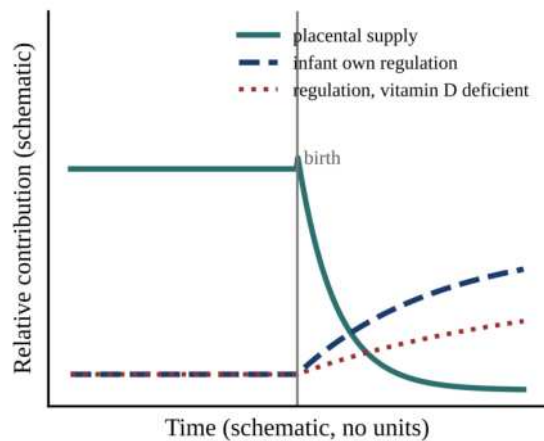


Figure 3. Qualitative shape of calcium supply across the transition from fetus to newborn (schematic, not data).

Early trials in Britain reported better calcium status in supplemented mothers and infants (34, 35). A later randomised trial found that higher daily doses brought more mothers and infants to sufficiency without evident harm (36), and a Cochrane review judged that supplementation raises maternal vitamin D concentrations (37). A large trial of high-dose supplementation in Bangladesh did not change infant growth, a reminder that raising 25-hydroxyvitamin D does not automatically improve every downstream outcome (38). Nothing in this reading argues against supplementation; it argues for measuring what supplementation achieves.

4.4 Quality of the Evidence

Several difficulties limit what can be said. Case reports and small series dominate the direct evidence, and in many the maternal level was measured late or not at all. A clinician reading a single case report needs a simple answer: was anything else going on? Laboratory methods for 25-hydroxyvitamin D have differed in what they report, and early work showed that assay variation can change which patients are called deficient (39).

Thresholds for deficiency and insufficiency also differ between guidelines and are still debated (40, 41, 42). A single case report seldom separates the acute seizure from the deficiency behind it, which are managed differently (43, 44). The point is not that a report is poor, only that its conclusion travels less far than its title suggests.

Table 3. Prevention measures and how firmly the evidence supports them.

Measure	What the evidence shows	Main uncertainty
Maternal supplementation	Raises maternal and cord vitamin D status (36, 37)	Effect on neonatal calcium outcomes is less clear
Early-trial observations	Supplemented mothers had better calcium status (34, 35)	Older, small studies; methods dated
Infant supplementation	Recommended for breastfed infants (30, 31)	Does not help a seizure in the first days

Note: A qualitative reading of the cited trials, guidelines and reviews, not a formal grading of evidence.

5. Discussion

5.1 Interpretation

The literature supports a plausible, mechanistically sound link between severe maternal vitamin D deficiency and neonatal hypocalcaemic seizures, mainly through low neonatal stores and a weaker postnatal calcium response. It does not support a claim that deficiency is the whole explanation in every infant, and it cannot say how often seizures follow a given level of maternal deficiency. That is a modest claim, and it is the one the evidence will bear.

5.2 Limitations

The most important limitation sits in the evidence itself, not in this review: direct



data are mostly descriptive, and diagnostic thresholds and assays vary. The review adds its own limits: sources were chosen by judgement without a systematic search, so relevant studies may have been missed, and readers who need effect sizes must go to the original studies. Reviews are more useful when they say where the evidence stops than when they smooth it over.

5.3 Future Work

Prospective studies that measure maternal and cord vitamin D, calcium, magnesium and parathyroid hormone together would clarify how often, and at what level of deficiency, neonatal hypocalcaemia occurs. Trials of supplementation should report neonatal calcium outcomes, not only blood levels, and standardised assays would make studies comparable. These points apply to many nutritional deficiencies, not only to this one.

6. Conclusion

Severe maternal vitamin D deficiency is a recognised and preventable contributor to neonatal hypocalcaemic seizures, acting by shrinking neonatal stores and weakening the postnatal calcium response. The published evidence for that link is mechanistic and descriptive, with limited controlled data on outcomes, and mild deficiency does not reliably produce it. Supplementation raises maternal status; its effect on neonatal calcium still needs direct study.

7. Acknowledgements

The authors thank colleagues who commented on earlier drafts of this review.

8. Funding

This work received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

9. Conflict of Interest

The authors declare no conflict of interest.

10. Author Contributions

M.K. conceived the review and wrote the sections on physiology. S.T. wrote the sections on clinical presentation and differential diagnosis. H.O. wrote the sections on prevention and appraisal of the evidence. All authors revised the manuscript and approved the final version.

11. Ethics Statement

This is a review of published literature. No patients were studied and no ethical approval was required.

12. Data Availability

No new data were generated. All sources are cited in the reference list.

13. References

1. Pressler, R. M., Cilio, M. R., Mizrahi, E. M., Moshé, S. L., Nunes, M. L., Plouin, P., ... Zuberi, S. M. (2021). The ILAE classification of seizures and the epilepsies: Modification for seizures in the neonate. *Epilepsia*, 62(3), 615–628. DOI: [10.1111/epi.16815](https://doi.org/10.1111/epi.16815)
2. Salle, B. L., Delvin, E. E., Lapillonne, A., Bishop, N. J., & Glorieux, F. H. (2000). Perinatal metabolism of vitamin D. *American Journal of Clinical Nutrition*, 71(5), 1317S–1324S. DOI: [10.1093/ajcn/71.5.1317s](https://doi.org/10.1093/ajcn/71.5.1317s)
3. Thandrayen, K., & Pettifor, J. M. (2010). Maternal vitamin D status: Implications for the development of infantile nutritional rickets. *Endocrinology and Metabolism Clinics of North America*, 39(2), 303–320. DOI: [10.1016/j.ecl.2010.02.004](https://doi.org/10.1016/j.ecl.2010.02.004)
4. Camadool, L., Tibbott, R., & Isaza, F. (2007). Maternal vitamin D deficiency associated with neonatal hypocalcaemic convulsions. *Nutrition Journal*, 6, 23. DOI: [10.1186/1475-2891-6-23](https://doi.org/10.1186/1475-2891-6-23)
5. Maiya, S., Sullivan, I., Allgrove, J., Yates, R., Malone, M., Brain, C., ... Bhattacharya, K. (2008). Hypocalcaemia and vitamin D deficiency: An important, but preventable, cause of life-threatening infant heart failure. *Heart*, 94(5), 581–584. DOI: [10.1136/hrt.2007.119792](https://doi.org/10.1136/hrt.2007.119792)
6. Jones, G., Prosser, D. E., & Kaufmann, M. (2014). Cytochrome P450-mediated metabolism of vitamin D. *Journal of Lipid Research*, 55(1), 13–31. DOI: [10.1194/jlr.R031534](https://doi.org/10.1194/jlr.R031534)



7. Holick, M. F. (2007). Vitamin D deficiency. *New England Journal of Medicine*, 357(3), 266–281. DOI: [10.1056/NEJMra070553](https://doi.org/10.1056/NEJMra070553)
8. Kovacs, C. S. (2014). Bone development and mineral homeostasis in the fetus and neonate: Roles of the calciotropic and phosphotropic hormones. *Physiological Reviews*, 94(4), 1143–1218. DOI: [10.1152/physrev.00014.2014](https://doi.org/10.1152/physrev.00014.2014)
9. Hannan, F. M., Kallay, E., Chang, W., Brandi, M. L., & Thakker, R. V. (2019). The calcium-sensing receptor in physiology and in calcitropic and noncalcitropic diseases. *Nature Reviews Endocrinology*, 15(1), 33–51. DOI: [10.1038/s41574-018-0115-0](https://doi.org/10.1038/s41574-018-0115-0)
10. Kovacs, C. S. (2016). Maternal mineral and bone metabolism during pregnancy, lactation, and post-weaning recovery. *Physiological Reviews*, 96(2), 449–547. DOI: [10.1152/physrev.00027.2015](https://doi.org/10.1152/physrev.00027.2015)
11. Webb, A. R., Kline, L., & Holick, M. F. (1988). Influence of season and latitude on the cutaneous synthesis of vitamin D3: Exposure to winter sunlight in Boston and Edmonton will not promote vitamin D3 synthesis in human skin. *Journal of Clinical Endocrinology & Metabolism*, 67(2), 373–378. DOI: [10.1210/jcem-67-2-373](https://doi.org/10.1210/jcem-67-2-373)
12. Clemens, T. L., Adams, J. S., Henderson, S. L., & Holick, M. F. (1982). Increased skin pigment reduces the capacity of skin to synthesise vitamin D3. *The Lancet*, 319(8263), 74–76. DOI: [10.1016/S0140-6736\(82\)90214-8](https://doi.org/10.1016/S0140-6736(82)90214-8)
13. Wacker, M., & Holick, M. F. (2013). Sunlight and vitamin D: A global perspective for health. *Dermato-Endocrinology*, 5(1), 51–108. DOI: [10.4161/derm.24494](https://doi.org/10.4161/derm.24494)
14. Palacios, C., & Gonzalez, L. (2014). Is vitamin D deficiency a major global public health problem? *Journal of Steroid Biochemistry and Molecular Biology*, 144(Pt A), 138–145. DOI: [10.1016/j.jsbmb.2013.11.003](https://doi.org/10.1016/j.jsbmb.2013.11.003)
15. Cashman, K. D., Dowling, K. G., Škrabáková, Z., Gonzalez-Gross, M., Valtueña, J., De Henauw, S., ... Kiely, M. (2016). Vitamin D deficiency in Europe: Pandemic? *American Journal of Clinical Nutrition*, 103(4), 1033–1044. DOI: [10.3945/ajcn.115.120873](https://doi.org/10.3945/ajcn.115.120873)
16. Saraf, R., Morton, S. M. B., Camargo, C. A., Jr., & Grant, C. C. (2016). Global summary of maternal and newborn vitamin D status: A systematic review. *Maternal & Child Nutrition*, 12(4), 647–668. DOI: [10.1111/mcn.12210](https://doi.org/10.1111/mcn.12210)
17. Bodnar, L. M., Simhan, H. N., Powers, R. W., Frank, M. P., Cooperstein, E., & Roberts, J. M. (2007). High prevalence of vitamin D insufficiency in black and white pregnant women residing in the northern United States and their neonates. *Journal of Nutrition*, 137(2), 447–452. DOI: [10.1093/jn/137.2.447](https://doi.org/10.1093/jn/137.2.447)
18. Ward, L. M., Gaboury, I., Ladhani, M., & Zlotkin, S. (2007). Vitamin D–deficiency rickets among children in Canada. *Canadian Medical Association Journal*, 177(2), 161–166. DOI: [10.1503/cmaj.061377](https://doi.org/10.1503/cmaj.061377)
19. Dawodu, A., & Wagner, C. L. (2007). Mother–child vitamin D deficiency: An international perspective. *Archives of Disease in Childhood*, 92(9), 737–740. DOI: [10.1136/adc.2007.122689](https://doi.org/10.1136/adc.2007.122689)
20. Hollis, B. W., & Wagner, C. L. (2004). Assessment of dietary vitamin D requirements during pregnancy and lactation. *American Journal of Clinical Nutrition*, 79(5), 717–726. DOI: [10.1093/ajcn/79.5.717](https://doi.org/10.1093/ajcn/79.5.717)
21. Mulligan, M. L., Felton, S. K., Riek, A. E., & Bernal-Mizrachi, C. (2010). Implications of vitamin D deficiency in pregnancy and lactation. *American Journal of Obstetrics and Gynecology*, 202(5), 429.e1–429.e9. DOI: [10.1016/j.ajog.2009.09.002](https://doi.org/10.1016/j.ajog.2009.09.002)
22. Cooper, M. S., & Gittoes, N. J. L. (2008). Diagnosis and management of hypocalcaemia. *BMJ*, 336(7656), 1298–1302. DOI: [10.1136/bmj.39582.589433.BE](https://doi.org/10.1136/bmj.39582.589433.BE)
23. Mannstadt, M., Bilezikian, J. P., Thakker, R. V., Hannan, F. M., Clarke, B. L., Rejnmark, L., ... Brandi, M. L. (2017). Hypoparathyroidism. *Nature Reviews Disease Primers*, 3, 17055. DOI: [10.1038/nrdp.2017.55](https://doi.org/10.1038/nrdp.2017.55)
24. Pettifor, J. M. (2004). Nutritional rickets: Deficiency of vitamin D, calcium, or both? *American Journal of Clinical Nutrition*, 80(6), 1725S–1729S. DOI: [10.1093/ajcn/80.6.1725S](https://doi.org/10.1093/ajcn/80.6.1725S)
25. Carpenter, T. O., Shaw, N. J., Portale, A. A., Ward, L. M., Abrams, S. A., & Pettifor, J. M. (2017). Rickets. *Nature Reviews Disease Primers*, 3, 17101. DOI: [10.1038/nrdp.2017.101](https://doi.org/10.1038/nrdp.2017.101)
26. Hollis, B. W., & Wagner, C. L. (2013). Vitamin D and pregnancy: Skeletal effects, nonskeletal effects, and birth outcomes. *Calcified Tissue International*, 92(2), 128–139. DOI: [10.1007/s00223-012-9607-4](https://doi.org/10.1007/s00223-012-9607-4)
27. Basatemur, E., & Sutcliffe, A. (2015). Incidence of hypocalcemic seizures due to vitamin D deficiency in children in the United Kingdom and Ireland. *Journal of Clinical Endocrinology & Metabolism*, 100(1), E91–E95. DOI: [10.1210/jc.2014-2773](https://doi.org/10.1210/jc.2014-2773)
28. Basatemur, E., Horsfall, L., Marston, L., Rait, G., & Sutherland, A. (2017). Trends in the diagnosis of vitamin D deficiency. *Pediatrics*, 139(3), e20162748. DOI: [10.1542/peds.2016-2748](https://doi.org/10.1542/peds.2016-2748)
29. Holick, M. F., Binkley, N. C., Bischoff-Ferrari, H. A., Gordon, C. M., Hanley, D. A., Heaney, R. P., ... Weaver, C. M. (2011). Evaluation, treatment, and prevention of vitamin D deficiency: An Endocrine Society clinical practice guideline.



- Journal of Clinical Endocrinology & Metabolism*, 96(7), 1911–1930. DOI: [10.1210/jc.2011-0385](https://doi.org/10.1210/jc.2011-0385)
30. Wagner, C. L., Greer, F. R., & American Academy of Pediatrics Section on Breastfeeding and Committee on Nutrition. (2008). Prevention of rickets and vitamin D deficiency in infants, children, and adolescents. *Pediatrics*, 122(5), 1142–1152. DOI: [10.1542/peds.2008-1862](https://doi.org/10.1542/peds.2008-1862)
 31. Munns, C. F., Shaw, N., Kiely, M., Specker, B. L., Thacher, T. D., Ozono, K., ... Högl, W. (2016). Global consensus recommendations on prevention and management of nutritional rickets. *Journal of Clinical Endocrinology & Metabolism*, 101(2), 394–415. DOI: [10.1210/jc.2015-2175](https://doi.org/10.1210/jc.2015-2175)
 32. Ross, A. C., Manson, J. E., Abrams, S. A., Aloia, J. F., Brannon, P. M., Clinton, S. K., ... Shapses, S. A. (2011). The 2011 report on dietary reference intakes for calcium and vitamin D from the Institute of Medicine: What clinicians need to know. *Journal of Clinical Endocrinology & Metabolism*, 96(1), 53–58. DOI: [10.1210/jc.2010-2704](https://doi.org/10.1210/jc.2010-2704)
 33. Specker, B. (2004). Vitamin D requirements during pregnancy. *American Journal of Clinical Nutrition*, 80(6), 1740S–1747S. DOI: [10.1093/ajcn/80.6.1740S](https://doi.org/10.1093/ajcn/80.6.1740S)
 34. Brooke, O. G., Brown, I. R. F., Bone, C. D. M., Carter, N. D., Cleeve, H. J. W., Maxwell, J. D., ... Winder, S. M. (1980). Vitamin D supplements in pregnant Asian women: Effects on calcium status and fetal growth. *British Medical Journal*, 280(6216), 751–754. DOI: [10.1136/bmj.280.6216.751](https://doi.org/10.1136/bmj.280.6216.751)
 35. Cockburn, F., Belton, N. R., Purvis, R. J., Giles, M. M., Brown, J. K., Turner, T. L., ... Pocock, S. J. (1980). Maternal vitamin D intake and mineral metabolism in mothers and their newborn infants. *British Medical Journal*, 281(6232), 11–14. DOI: [10.1136/bmj.281.6232.11](https://doi.org/10.1136/bmj.281.6232.11)
 36. Hollis, B. W., Johnson, D., Hulsey, T. C., Ebeling, M., & Wagner, C. L. (2011). Vitamin D supplementation during pregnancy: Double-blind, randomized clinical trial of safety and effectiveness. *Journal of Bone and Mineral Research*, 26(10), 2341–2357. DOI: [10.1002/jbmr.463](https://doi.org/10.1002/jbmr.463)
 37. De-Regil, L. M., Palacios, C., Lombardo, L. K., & Peña-Rosas, J. P. (2016). Vitamin D supplementation for women during pregnancy. *Cochrane Database of Systematic Reviews*, 2016(1), CD008873. DOI: [10.1002/14651858.CD008873.pub3](https://doi.org/10.1002/14651858.CD008873.pub3)
 38. Roth, D. E., Morris, S. K., Zlotkin, S., Gernand, A. D., Ahmed, T., Shanta, S. S., ... Al Mahmud, A. (2018). Vitamin D supplementation in pregnancy and lactation and infant growth. *New England Journal of Medicine*, 379(6), 535–546. DOI: [10.1056/NEJMoa1800927](https://doi.org/10.1056/NEJMoa1800927)
 39. Binkley, N., Krueger, D., Cowgill, C. S., Plum, L., Lake, E., Hansen, K. E., ... Drezner, M. K. (2004). Assay variation confounds the diagnosis of hypovitaminosis D: A call for standardization. *Journal of Clinical Endocrinology & Metabolism*, 89(7), 3152–3157. DOI: [10.1210/jc.2003-031979](https://doi.org/10.1210/jc.2003-031979)
 40. Thacher, T. D., & Clarke, B. L. (2011). Vitamin D insufficiency. *Mayo Clinic Proceedings*, 86(1), 50–60. DOI: [10.4065/mcp.2010.0567](https://doi.org/10.4065/mcp.2010.0567)
 41. Hollis, B. W. (2005). Circulating 25-hydroxyvitamin D levels indicative of vitamin D sufficiency: Implications for establishing a new effective dietary intake recommendation for vitamin D. *Journal of Nutrition*, 135(2), 317–322. DOI: [10.1093/jn/135.2.317](https://doi.org/10.1093/jn/135.2.317)
 42. Misra, M., Pacaud, D., Petryk, A., Collett-Solberg, P. F., & Kappy, M. (2008). Vitamin D deficiency in children and its management: Review of current knowledge and recommendations. *Pediatrics*, 122(2), 398–417. DOI: [10.1542/peds.2007-1894](https://doi.org/10.1542/peds.2007-1894)
 43. Pearce, S. H. S., & Cheetham, T. D. (2010). Diagnosis and management of vitamin D deficiency. *BMJ*, 340, b5664. DOI: [10.1136/bmj.b5664](https://doi.org/10.1136/bmj.b5664)
 44. Pilz, S., Zittermann, A., Trummer, C., Theiler-Schwetz, V., Lerchbaum, E., Keppel, M. H., ... Pandis, M. (2019). Vitamin D testing and treatment: A narrative review of current evidence. *Endocrine Connections*, 8(2), R27–R43. DOI: [10.1530/EC-18-0432](https://doi.org/10.1530/EC-18-0432)