Early Vitamin D Supplementation in Infants Born Extremely Preterm and Fed Human Milk: A Randomized Controlled Trial

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Objective To evaluate the effects of vitamin D supplementation on short-term respiratory outcomes in infants born at 28 weeks of gestation or less and fed human milk.

Study design This masked, randomized controlled trial included infants born extremely preterm and fed human milk in 2023-2024. Infants received either 800 IU/day vitamin D (intervention) or no additional vitamin D (control) for the first 14 days. Masking was maintained by adding 800 IU of vitamin D to daily feedings. The primary outcome was severity of bronchopulmonary dysplasia (BPD) at 36 weeks of postmenstrual age. Secondary outcomes included reactance and resistance ($R_{7\text{-}19}$) measurements, obtained using impulse oscillometry at 36 weeks postmenstrual age, and metabolic bone disease (alkaline phosphatase >500 IU/L and phosphorus <5.5 mg/dL) at postnatal day 28.

Results A total of 126 infants were randomized (mean birthweight: 759 ± 228 g; 52% female). The intervention increased 25-hydroxy vitamin D3 concentrations (mean difference: +29 ng/mL; $P < .0001$). The severity of BPD did not differ between groups ($P = .60$). area under the reactance curve measurements were comparable (674 ± 207 vs 694 ± 240; $P = .64$) and $R_{7\text{-}19}$ measurements were not significantly lower in the intervention group (23 ± 8 vs 25 ± 9; $P = .18$). On postnatal day 28, the lower risk of metabolic bone disease observed in the intervention group did not reach statistical significance (9% vs 20%; relative risk: 0.43; 95% CI: 0.16, 1.15; $P = .08$). No serious adverse events related to the intervention were reported.

Conclusions Administering 800 IU/day of vitamin D during the first 2 weeks did not reduce BPD severity but may contribute to improved bone health. *(J Pediatr 2025;287:114754)*.

Trial registration ClinicalTrials.gov: NCT05615311. https://clinicaltrials.gov/study/NCT05615311.

At birth, infants born extremely preterm (EPT)—at 28 weeks of gestation or earlier—face an abrupt loss of maternal vitamin D supply,[1] leaving nearly all of the 26 000 infants born EPT each year in the United States[2] at risk of vitamin D deficiency.[3-7] Without supplementation,[8] this deficiency persists for weeks,[3,9] particularly in infants born EPT who are critically ill.[10] Despite this risk, recommendations for vitamin D supplementation in infants born EPT vary.[1,11] Some consensus guidelines do not recommend vitamin D doses greater than 400 IU/day before the establishment of full enteral nutrition or before documenting biochemical abnormalities in these infants,[12] which may result in suboptimal supplementation during the first 14 days after birth, a critical window for both bone and lung development.

Although vitamin D is primarily recognized for its role in skeletal health,[13,14] emerging evidence suggests that it also plays a dose-dependent role in lung development.[15-19] During the saccular and alveolar stages of lung growth,[16,17] vitamin D contributes to pneumocyte differentiation,[20] regulates surfactant production,[18,21] and influences alveolar formation.[22] All these findings indicate that early vitamin D supplementation could mitigate adverse respiratory outcomes in preterm infants,[23,24] including in those born EPT.[3]

No prior randomized trials have assessed the impact of vitamin D on bronchopulmonary dysplasia (BPD) and pulmonary function exclusively in infants born EPT.[25] Most trials have systematically excluded this vulnerable population[26] and defined vitamin D adequacy with a limited number of serum markers.[27] To address these gaps, our trial aimed to determine whether early, enteral vitamin D supplementation reduces the severity of BPD and improves lung mechanics at term-equivalent age. By leveraging impulse oscillometry as a bedside tool to quantify lung function, we aim to provide robust evidence on respiratory outcomes and lung mechanics at term-equivalent age in infants born EPT.

$25[OH]D_3$	25-hydroxy vitamin D3
AX	Area under the reactance curve
BPD	Bronchopulmonary dysplasia
EPT	Extremely preterm
PI	Ponderal index
PMA	Postmenstrual age
$R_{7\text{-}19}$	Difference in resistance at 7 and 19 Hz
UAB	University of Alabama at Birmingham

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Methods

Trial Design

This was a parallel-group, masked, randomized controlled trial that included infants born EPT between March 2023 and June 2024 at the University of Alabama at Birmingham (UAB) Hospital. Infants with a gestational age of 28 weeks and 6 days or less were eligible for inclusion. Infants with major congenital anomalies or a terminal illness, where decisions to withhold or limit life support had been made, were excluded from participation. This study was approved by the Institutional Review Board at UAB.

All infants born EPT admitted to the neonatal unit at UAB Hospital were screened for eligibility. Parents or legal guardians of eligible infants were approached for informed consent within the first 72 hours after birth. Written informed consent was obtained before treatment allocation. Randomization was performed using computer-generated, random block sizes of 4-8. Allocation was concealed using numbered, opaque, sealed envelopes, which were opened in sequential order only after informed consent was obtained. Multiples were randomized independently.

Clinicians and primary caregivers were masked. Dietitians and feeding specialists were unmasked and responsible for randomization and preparation of the human milk feedings with the vitamin D supplement (Enfamil D-Vi-Sol Liquid Vitamin D Supplement, Reckitt/Mead Johnson). Double masking ensured that study participants were treated and observed similarly during the trial.

Study Intervention

Participants were randomly assigned to one of two groups in a 1:1 allocation ratio. Infants were randomly assigned to receive either a usual human milk diet with an additional 800 IU/day vitamin D dose (intervention group) or a usual human milk diet without any additional vitamin D supplement (control group) for the first 14 days after birth. After postnatal day 14, vitamin D supplementation continued per routine practice at approximately 400 IU/day, delivered through human milk fortification with bovine-derived human milk fortifiers and enteral multivitamin adminsitration.[12] The 800 IU/day dose was within the maximum limit recommended, ensuring a total vitamin D intake (dietary + supplement) of approximately 1000 IU/day.[13] Infants in the control group received vitamin D only from standard sources, amounting to the minimum limit recommended, approximately 200-300 IU/day for the first 14 days after birth.

Outcome Measures

The primary clinical outcome was BPD at 36 weeks of postmenstrual age (PMA) using the Jensen criteria.[28,29] The five ordinal categories for this outcome included no BPD, grade 1 BPD (nasal cannula or supplemental oxygen requirement with a flow rate of 2 L per minute or less), grade 2 BPD (nasal cannula requirement with a flow rate greater than 2 L per minute or other noninvasive forms of respiratory support including CPAP), grade 3 BPD (invasive mechanical ventilation requirement), and death up to 36 weeks PMA to account for all study participants in the analysis. The primary physiologic outcomes were mean area under the reactance curve (AX) and the difference in resistance from 7 to 19 Hz (R_{7-19}) at 36 weeks PMA or discharge measured using the Tremoflo N-100 Airwave Oscillometry device (Thorasys). AX is a measure of the elasticity of lung tissue and R_{7-19} is a measure of the difference between central and peripheral airway resistance.[30] A higher AX indicates increased lung stiffness and a higher R_{7-19} indicates increased peripheral airway resistance.

Secondary clinical outcomes included any grade of BPD, death, the probability of survival without BPD at postnatal days 14 and 28, the total number of days of mechanical ventilation, length of hospital stay in days, and growth parameters at 36 weeks PMA, including the ponderal index (PI), a surrogate marker of adiposity in infants born EPT.[31]

Safety outcomes included necrotizing enterocolitis or spontaneous intestinal perforation, late-onset sepsis, serum 25-hydroxy vitamin D_3 (25[OH]D_3), metabolic bone disease (alkaline phosphatase >500 IU/L and phosphorus <5.5 mg/dL),[32] alkaline phosphatase, phosphorus, and calcium concentrations by postnatal day 28.

Monitoring of Cointerventions

This trial was a comparison of a usual human milk diet with or without additional 800 IU of vitamin D during the first 2 postnatal weeks under ordinary clinical circumstances. Therefore, all other intensive care and nutrition aspects were provided at clinicians' discretion. Following current UAB feeding guidelines, infants born EPT received 20 to 25 mL/kg/d of enteral feedings with either maternal or donor milk within the first 72 hours after birth. Then, these enteral feeding volumes were increased daily by 20-25 ml/kg/day, aiming for a target of at least 120 ml/kg/day. Once this target was reached, further increases were not dictated by the trial protocol. Bovine-derived fortifiers and multivitamins were added at approximately postnatal day 14.

Sample Size and Power Estimates

In an ancillary study of our initial vitamin D trial,[33] we demonstrated that grades 1 through 3 BPD at 36 weeks PMA occurred in 30% of infants born EPT and that the risk ratio favoring the early vitamin D supplementation groups was 0.5. To detect a 20% difference between groups, 0.05 level of significance, and 80% power for a chi-square test that compares proportions from two independent samples, we included a total of 63 patients in each group (n = 126).

Statistical Analysis

Data were analyzed using an intention-to-treat approach. Continuous variables were compared using student's *t*-test or the Mann-Whitney *U* test, depending on data distribution. Categorical variables were analyzed using the chi-square test or Fisher exact test as appropriate. For analysis of the primary

outcome, an unadjusted chi-square test to compare proportions between intervention and control groups was performed. The severity of BPD, treated as an ordinal outcome, was also analyzed using ordinal logistic regression. The unadjusted OR and adjusted odd ratio reported represent the change in the odds of being in a higher category of severity, under the assumption that the effect of the intervention is consistent across all outcome categories. A *P* value of <0.05 was considered statistically significant.

Results

A total of 126 infants born EPT were randomized (mean birth weight: 759 ± 228 g) [Figure], with 63 allocated to each group. Of these, 111 infants were eligible for pulmonary function assessment (54 in the intervention group and 57 in the control group). Baseline demographic and clinical characteristics, including birth weight, gestational age, sex, and the probability of survival without BPD at birth were comparable between groups [Table I]. By postnatal day 3, 124 infants had 25[OH]D_3 measurements, and 58 (47%) were found to have vitamin D insufficiency (25[OH]D_3 < 30 ng/mL). Approximately one-quarter (27%) of infants had vitamin D deficiency (25[OH]D_3 < 20 ng/mL).

The median time to full enteral feeding was 10 days (IQR: 7-15) in the intervention group and 10 days (IQR: 7-14) in the control group (P = .96). Human milk fortification with a bovine-derived product was initiated by postnatal day 15 in both groups. In unadjusted repeated-measures mixed models, there were no differences between groups in daily enteral fluid intake (P = .34), enteral caloric intake (P = .34), enteral protein intake (P = .28), or maternal-to-donor milk ratios (P = .50) during the first 14 days after birth.

The distribution of BPD severity did not differ between groups at 36 weeks PMA [Table II]. In an unadjusted ordinal logistic regression model with BPD (none, grade 1, grade 2, grade 3, and death) as outcome, the odds of being in a higher category were not significantly lower in the intervention group (OR: 0.99; 0.53-1.85; P = .98). In an ordinal logistic regression model adjusted for the probability of survival without BPD at birth, the lower odds of being in a higher category did not reach statistical significance (adjusted odds ratio: 0.72; 0.37-1.39; P = .32). AX and $R_{7\text{-}19}$ measurements were obtained in 104 infants. At 36 weeks PMA, AX measurements did not differ between groups (674 ± 207 vs 694 ± 240; P = .64). Lower $R_{7\text{-}19}$ measurements obtained in the intervention group

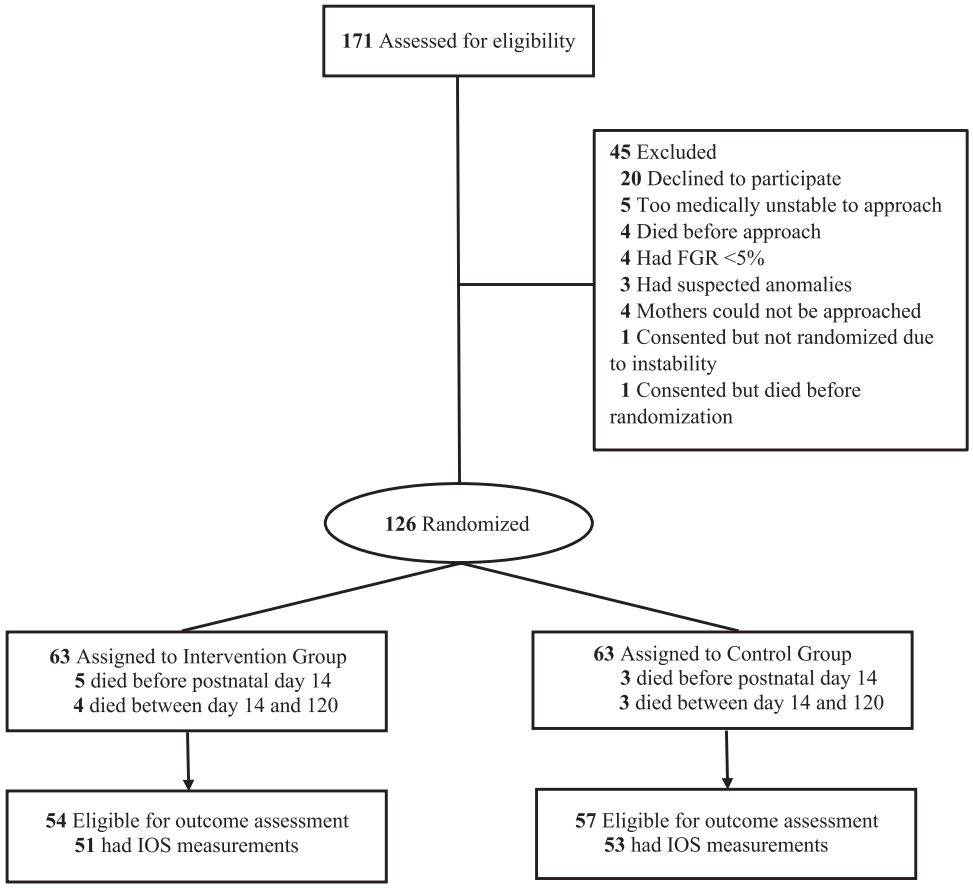

Figure. Flow diagram.

Table I. Baseline Characteristics

Characteristic	Intervention group (n = 63)	Control group (n = 63)
Gestational age in weeks, median (IQR)	25 (24-27)	26 (24-27)
22-23 weeks, n (%)	15 (24)	10 (16)
24-25 weeks, n (%)	15 (24)	23 (36)
26-28 weeks, n (%)	33 (52)	30 (48)
Female, n (%)	31 (49)	35 (56)
Black race, n (%)	34 (54)	28 (44)
Birth weight in grams, mean ± SD	754 ± 204	764 ± 252
Birth weight z-score, mean ± SD	−0.3 ± 1	−0.3 ± 1
Birth length in cm, mean ± SD	32 ± 3	32 ± 4
Birth length z-score, mean ± SD	−0.4 ± 1	−0.7 ± 1
Birth head circumference in cm, mean ± SD	23 ± 2	23 ± 2
Birth head circumference z-score, mean ± SD	−0.3 ± 1	−0.3 ± 1
Small or gestational age, n (%)	11 (17)	14 (22)
Vaginal delivery, n (%)	18 (29)	17 (27)
Multiple gestation, n (%)	8 (13)	14 (22)
Maternal hypertension, n (%)	41 (65)	35 (56)
Maternal diabetes, n (%)	9 (14)	8 (13)
Exposure to prenatal antibiotics, n (%)	39 (62)	44 (70)
Exposure to ≥ 2 doses of antenatal steroids, n (%)	49 (78)	50 (79)
25 (OH) Vitamin D in ng/mL at postnatal day 3, mean ± SD	35 ± 19	35 ± 20
Vitamin D insufficiency at postnatal day 3 (25[OH]D3 < 30 ng/mL)	29 (47)	27 (47)
Probability of survival without BPD at birth, median (IQR)	16 (5-37)	19 (7-49)
Probability of survival without BPD at postnatal day 3, median (IQR)	30 (7-49)	24 (9-51)

compared with the control group (23 ± 8 vs 25 ± 9) were not statistically significant ($P = .18$).

Growth outcomes did not differ significantly between groups [Table III]. The difference in PI at 36 weeks PMA favoring the intervention group did not reach statistical significance (30.1 ± 4.8 vs 31.7 ± 4.3; $P = .07$).

The difference in 25[OH]D3 concentrations from postnatal day 3 to postnatal day 28 was significantly higher in the intervention group (mean difference favoring the intervention group: +29 ng/mL; 95% CI: 20-40; $P < .0001$). On postnatal day 28, the risk reduction of metabolic bone disease (alkaline phosphatase >500 IU/L and phosphorus <5.5 mg/dL) observed in the intervention group did not reach statistical significance (9% vs 20%; relative risk: 0.43; 95% CI: 0.16, 1.15; $P = .08$). By postnatal day 28, none of the infants in the intervention group had calcium values exceeding 12 mg/dL. Three infants had 25[OH]D3 concentrations > 120 ng/mL without concurrent hypercalcemia indicative of toxicity (two in the intervention group and one in the control group). No serious adverse events related to vitamin D supplementation were reported. No significant differences in rates of late-onset sepsis, spontaneous intestinal perforation or necrotizing enterocolitis between groups were observed. Incidental fractures were observed in approximately 5% of infants, with no significant differences between the intervention and control groups.

Discussion

In this masked, randomized controlled trial, we sought to determine whether early enteral vitamin D supplementation

Table II. Respiratory Outcomes

Outcome	Intervention (n = 63)	Control (n = 63)	*P*
BPD at 36 weeks PMA			
None, n (%)	18 (29)	18 (29)	.60
Grade 1 BPD, n (%)	21 (33)	19 (30)	
Grade 2 BPD, n (%)	11 (18)	13 (21)	
Grade 3 BPD, n (%)	4 (6)	8 (13)	
Death before 36 weeks PMA	9 (14)	5 (8)	
Pulmonary function at 36 weeks PMA			
IOS measurement, n (%)	51 (81.0)	53 (84.1)	.64
Postnatal age at the time of IOS measurement in days, mean ± SD	81 ± 31	81 ± 28	.97
AX, mean ± SD	674 ± 207	694 ± 240	.64
R7-19, mean ± SD	23 ± 8	25 ± 9	.18
Other respiratory outcomes			
Mechanical ventilation during the first 21 days after birth in days, mean ± SD	9.90 ± 9.03	9.17 ± 9.16	.65
Postnatal steroids, n (%)	29 (46.0)	34 (54.0)	.48
Probability of survival without BPD at postnatal day 14, median (IQR)	24 (8-74)	34 (4-81)	.86
Probability of survival without BPD at postnatal day 28, median (IQR)	27 (6-55)	19 (2-67)	.58

IOS, impulse oscillometry. IOS was performed in infants receiving up to 2 L/min nasal cannula flow. A larger AX indicates increased lung stiffness. A larger R7-19 indicates increased peripheral airway resistance.

Table III. Other Clinical Outcomes

Outcome	Intervention group (n = 63)	Control group (n = 63)	*P*
Growth outcomes at 36 weeks PMA			
Weight (grams), mean ± SD	2063 ± 459	2078 ± 453	.86
Weight z-score, mean ± SD	−1.34 ± 0.91	−1.38 ± 1.16	.82
Length (cm), mean ± SD	41.2 ± 3.2	40.2 ± 3.7	.13
Length z-score, mean ± SD	−2.19 ± 1.23	−2.52 ± 1.38	.19
Head circumference (cm), mean ± SD	30.0 ± 1.7	29.9 ± 1.8	.88
Head circumference z-score, mean ± SD	−1.66 ± 1.12	−1.64 ± 1.12	.90
Growth rate (2-point method) in g/kg/day, mean ± SD	12.65 ± 4.93	13.21 ± 1.91	.44
Growth rate (exponential method) in g/kg/day, mean ± SD	13.74 ± 5.59	14.37 ± 2.33	.45
Decline in weight z-score from birth, mean ± SD	−1.02 ± 0.87	−1.07 ± 0.85	.79
Decline in length z-score from birth, mean ± SD	−1.69 ± 0.98	−1.85 ± 1.09	.42
Decline in head circumference z-score from birth, mean ± SD	−1.35 ± 1.12	−1.35 ± 0.85	.98
Postnatal growth faltering, n (%)	32 (60)	38 (65)	.58
Body mass index, mean ± SD	12.33 ± 1.53	12.68 ± 1.48	.22
Ponderal index, mean ± SD	30.13 ± 4.81	31.71 ± 4.33	.07
Bone metabolism markers at postnatal day 28	n = 57	n = 59	
25(OH) D_3 in ng/mL, mean ± SD	63 ± 24	33 ± 21	<.0001
Vitamin D insufficiency (25[OH]D3 < 30 ng/mL)	3 (5)	34 (58)	<.0001
Calcium in mg/dL, mean ± SD	9.56 ± 0.62	9.11 ± 0.93	.003
Phosphorus in mg/dL, mean ± SD	5.25 ± 1.14	5.19 ± 1.22	.75
Alkaline phosphatase in IU/L, mean ± SD	390 ± 143	422 ± 185	.30
Alkaline phosphatase > 500 IU/L, n (%)	11 (19)	19 (32)	.12
Phosphorus < 5.5 mg/dL, n (%)	33 (58)	39 (66)	.36
Metabolic bone disease, n (%)	5 (9)	12 (20)	.08
Safety outcomes			
Late-onset sepsis up to 120 days, n (%)	14 (22)	16 (25)	
NEC up to 120 days, n (%)	2 (3)	3 (5)	.68
SIP up to 120 days, n (%)	2 (3)	5 (8)	.27
Death up to 120 days, n (%)	9 (13)	6 (10)	.41
Duration of hospital stay in days, mean ± SD	129 ± 62	124 ± 52	.62

NEC, necrotizing enterocolitis; *SIP*, spontaneous intestinal perforation.

in infants born EPT could improve respiratory outcomes. Despite growing evidence suggesting a potential role of vitamin D in lung development, our findings indicate that supplementation with 800 IU/day mixed in human milk feedings during the first 14 days after birth did not significantly reduce the severity of BPD at 36 weeks PMA. Although pulmonary function measurements (AX and $R_{7\text{-}19}$ values) favored the intervention group, the differences did not reach statistical significance. Importantly, our results confirm that this supplementation approach is both safe and effective in preventing vitamin D insufficiency by postnatal day 28. Although the *P* value did not reach statistical significance, the effect size and direction suggest a potential reduction in the risk of metabolic bone disease among infants randomized to the intervention group.

The variability in reported outcomes across clinical studies may stem from differences in vitamin D dosing strategies,[34] the absence of an international consensus on optimal supplementation for infants born EPT,[11] ongoing controversies regarding the different methods used to measure vitamin D status and define deficiency, and the common practice of reporting BPD as a binary outcome.[25,35] Furthermore, evolving prenatal supplementation practices have led to a shift in the prevalence of vitamin D deficiency among infants born EPT, potentially influencing study findings.

This trial has several limitations. First, it was conducted in a single neonatal unit, which may limit the generalizability of findings to other settings with different risk factors for vitamin D deficiency or BPD. In neonatal intensive care units with different mixes of risk factors (or endotypes) for BPD, the impact of vitamin D supplementation on the complex pathophysiology of BPD may differ.[36] Second, the intervention was limited to the first 14 days after birth, with routine 400 IU/day vitamin D supplementation thereafter. Although this approach could minimize the risk of potential toxicity reported in previous dose-response trials,[33] it may not fully capture the potential dose-dependent effects of early high-dose vitamin D on respiratory and skeletal outcomes. The lack of a significant difference in respiratory outcomes may reflect the fact that, even though early supplementation improved vitamin D concentrations more rapidly, the cumulative exposure over time in both groups due to current supplementation practices might have been sufficient to mitigate major differences in lung development. Third, with a sample size of 126 infants, the study may have been underpowered to detect smaller but clinically meaningful differences, particularly given the declining prevalence of vitamin D deficiency at birth. In our prior trial,[3] infants born EPT and randomized to 800 IU/day achieved sufficient 25[OH]D3 concentrations without increased toxicity risk by 14 days.[33] However, the prevalence of vitamin D deficiency before treatment allocation was approximately 70% in our previous trial (vs 27% in the current trial), potentially contributing to greater differences in clinical outcomes. The observed response in 25[OH]D3 concentrations suggests that the supplementation method used in this trial to ensure masking does not

compromise bioavailability. This approach may serve as an alternative to the current common practice of separate administration of vitamin D supplements, particularly when full enteral nutrition is established within the first 10 days after birth. Although detailed nutritional data were not collected across the full first 28 days after birth, we found no significant differences in enteral nutrition practices between groups during the first 14 days. This suggests that meaningful differences in parenteral nutrition during the first 14 days were unlikely.

Another limitation is the lack of an ideal gold standard and the potential need for predefined adjustments for pulmonary function measures (AX and $R_{7\text{-}19}$ values). These metrics may require correction for sex and anthropometric variables, such as length, weight or PI,[31] which accounts for weight and length at the time of measurement. The lower PI observed at 36 weeks PMA in the intervention group likely reflects improved linear growth rather than reduced adiposity, possibly mediated by the prevention of impaired bone homeostasis, a useful surrogate marker for metabolic bone disease that has not been validated against definitive diagnostic studies.

This study has several strengths. It employed a parallel-group, masked randomized clinical trial design, which minimized selection bias, balanced baseline characteristics, ensured allocation concealment, and enhanced the reliability of findings. The innovative method of vitamin D supplementation—administering 800 IU/day mixed with breast milk within the first 72 hours after birth—targeted a critical window for lung development and appeared safe and effective in regulating calcium, phosphorus, and bone metabolism in infants born EPT.

A major strength is the incorporation of pulmonary function tests in the assessment of respiratory outcomes. The trial evaluated not only the severity of BPD at 36 weeks PMA, a clinically meaningful endpoint, but also lung mechanics with impulse oscillometry, an objective and quantifiable measure. Although the expected AX and $R_{7\text{-}19}$ effect sizes of an effective intervention have not yet been established, and the clinical significance of a mean 2-point difference remains uncertain, these measurements provide deeper insight into pulmonary function beyond traditional binary outcomes. Unlike previous trials, which have inconsistently reported respiratory outcomes across different vitamin D dosing regimens, our trial provides robust data on both pulmonary and skeletal effects in infants born EPT.[37]

It is unclear whether a sustained higher dose beyond 2 weeks would have led to greater improvements in lung function. Ongoing trials are expected to provide further insight into whether a longer duration of supplementation may yields greater pulmonary benefits.[38] Moreover, future meta-analyses using individual patient data and combining results from this and ongoing trials in this high-risk population may help clarify the clinical significance of some potentially meaningful, but statistically non-significant, findings.

In conclusion, although administering 800 IU/day of vitamin D mixed with human milk during the first 2 weeks after birth does not reduce the severity of BPD at 36 weeks PMA in infants born EPT with a relatively low prevalence of vitamin D deficiency, it appears to be a safe and effective method for achieving vitamin D sufficiency. Secondary analyses suggest that this approach may help prevent metabolic bone disease by postnatal day 28. Further research is warranted to evaluate long-term outcomes of sustained supplementation beyond the neonatal period.

CRediT authorship contribution statement

Ariel A. Salas: Writing – review & editing, Writing – original draft, Supervision, Project administration, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Tori Argent:** Supervision, Resources, Methodology, Investigation. **Seabrook Jeffcoat:** Writing – review & editing, Project administration, Formal analysis, Data curation. **Marley Tucker:** Investigation, Data curation. **Ambika P. Ashraf:** Writing – review & editing, Investigation, Conceptualization. **Colm P. Travers:** Writing – review & editing, Methodology, Investigation, Conceptualization. ■

Declaration of Competing Interest

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Data Statement

Data sharing statement available at www.jpeds.com.

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