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CORRESPONDENCE

Letter to the Editor RE: Singh AJ *et al.* Pediatr Res 67:619–623

To the Editor: We read with great interest the article by Singh *et al.* (1) which provided a new idea on the effect of vitamin A in neonatal respiratory disease. Further clinical research may be needed, but some wonders exist which are as follows:

1. Three groups were set in the article: I = control; II = surfactant; and III = surfactant + vitamin A. Maybe groups II and III should be set, II = surfactant (100% activity) and III = surfactant (60% activity) + vitamin A, because the surface activity was 40% lower in group III than that in group II, and there was no significant difference of gas exchange between groups II and III. Did vitamin A have the efficacy equal to surfactant with 40% activity or what?
2. The study should have included both preterm and term infants. Although vitamin A supplementation could be more effective on premature infants with chronic lung disease (CLD), recent research suggested that 25% of infants remain vitamin A deficient despite vitamin A supplementation (2). The persistence of biochemical vitamin A deficiency might be due to impaired vitamin A transportation. Transthyretin, a major vitamin A transport protein, has been suggested to be reduced by inflammation (3).
3. Intramuscular administration of 5000 IU vitamin A every other day for 4 wk could decrease the incidence of CLD. How much and how often should vitamin A be supplemented to premature infants by intratracheal administration to ensure the effect?
4. How about vitamin A supplemented together with retinoic acid? Recent study has shown that a combination of vitamin A (the nutrient) and retinoic acid (the metabolite) improved more tissue retinoid stores than either vitamin A or retinoic acid alone in infant rats (4).

In conclusion, although intramuscular administration of vitamin A has been suggested to reduce the incidence of CLD, intratracheal administration of vitamin A may provide a new way with more absorb dosage and less pain.

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Letter to the Editor RE: Okogbule-Wonodi *et al.* Pediatr Res 69:442–447

To the Editor: We read with great interest the recent article by Okogbule-Wonodi *et al.* (1) about whether *Ureaplasma* respiratory tract colonization of preterm infants <33 wk gestation is associated with an increased risk for necrotizing enterocolitis (NEC). The authors evaluated 368 infants <33 wk gestation with one or more tracheal or nasopharyngeal aspirates for *Ureaplasma* culture and PCR obtained during the first week of life. They stated that NEC was confirmed in 29 of 368 (7.9%) of the combined cohorts, and the incidence of NEC was 2.2-fold higher in *Ureaplasma*-positive (12.3%) than *Ureaplasma*-negative (5.5%) infants <33 wk and 3.3-fold higher in *Ureaplasma*-positive (14.6%) than *Ureaplasma*-negative (4.4%) infants ≤28 wk. In addition, cord serum IL-6 and IL-1β concentrations were significantly higher in *Ureaplasma*-positive than in *Ureaplasma*-negative NEC-affected infants, and so, the authors suggested that *Ureaplasma* may be a factor in NEC pathogenesis in preterm infants by contributing to intestinal mucosal injury and/or altering systemic or local immune responses.

We have recently completed a study concerning effects of *Ureaplasma* respiratory tract colonization and its management on development of chronic lung disease (unpublished data). When we evaluated 224 enrolled infants who were <32 wk and seeked for *Ureaplasma* respiratory colonization with one or more tracheal or nasopharyngeal aspirates for *Ureaplasma* culture, we could not demonstrate any relationship between *Ureaplasma* colonization and NEC development (Table 1), which is similar to previous published study by Perzigian *et al.* (2). Two important factors, which were associated with NEC stage ≥2 development, were late-onset proven sepsis, volume of red blood cell transfusion, and cord serum IL-6 (Table 1). Multivariate logistic regression analysis revealed that there was only significant association between the red blood cell transfusion and NEC development (OR, 1.4; 95% CI, 1.18–1.74; $p = 0.01$). Although our results shows similarity with study by Okogbule-Wonodi *et al.* (1) in high cord serum IL-6 levels and we agree with authors on its possible association with NEC development, it is obvious that