during the time course of observation in both patients. Cardiotoxicity as possible side effect of DL sodium β-hydroxybutyrate is thus unlikely. Vomiting or loss of "orality" was preexisting to the administration of DL sodium β-hydroxybutyrate and did not deteriorate upon treatment.

In our observational study, DL sodium β-hydroxybutyrate was administered as an ultima ratio in two infants with PHHI who suffered from recurrent hypoglycemic events despite extensive conventional treatment. Results are preliminary and further experimental and clinical studies are needed prior to general clinical application. In the light of the fact that short chain fatty acid oxidation defects such as SCAD and SCHAD deficiency may be associated with PHHI, we fully agree with Hussain *et al*. that patients who might potentially benefit from this treatment have to be selected carefully. Administration of DL sodium β-hydroxybutyrate in PHHI is complementary and is not a substitute of conventional therapy.

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To the Editor: We read with interest the article by Schultz *et al*. (1), who present data on the neonatal inflammatory response.

As detection of intracellular cytokines may not necessarily correlate with extracellular cytokine concentrations, we do not totally agree with the authors' conclusions on the neonatal immune system extracted from these data.

As well, there may be concern about some methodical issues:

1. The authors stored blood samples for up to 24 hours at room temperature. Reinsberg *et al*. (2) showed that IL-8 concentrations in whole blood lysate increased significantly after 3 hours of storage at room temperature. The authors do not show data on the influences of sample storage.

2. Differences in storage time between the groups may have affected the results.

3. Van Langevelde *et al*. (3) reported a maximum endotoxin concentration of 18 pg/ml in adult febrile patients. In this study a maximal LPS stimulus of 30,000 pg/ml has been used. The effect on intracellular cytokine production may not reflect *in vivo* conditions.

4. It has been shown that labor induces cytokine production in term and preterm infants (4). Modification of cytokine synthesis by labor may differ in neonatal cells from preterm and term infants. The diminished spontaneous production of intracellular cytokines as well as the production after *ex vivo* stimulation in preterm infants <32 weeks gestational age could well have resulted from the 100% cesarean section rate in this group.

5. The authors demonstrate a dose related inhibition of *ex vivo* monocyte cytokine expression by incubation with dexamethasone. They do not comment on the possible effects of the different rates of corticosteroid pretreatment between the study groups (50%, 96%, 100%). There is no information on the number of doses and the type of steroid used for prophylaxis of RDS.

6. The diagnosis of neonatal infection has been based on single parameters. This is not a commonly used definition.

7. Anti-inflammatory activity has not been investigated. Data on IL-10 derived from different study designs are compared in the discussion.

On the background of these questions, the hypothesis of an enhanced neonatal inflammatory response remains at least debatable. Evaluation of imbalances of pro- and antiinflammatory components in neonatates may be more effective by investigation of a larger cytokine panel.

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Response

We would like to thank Dembinski and colleagues for the interest they have taken in our work. They raised concern about the method used and the conclusions drawn from our data. Until recently the neonatal inflammatory response was consid-