used over a period of several weeks, they enable to find out personal best and diurnal variation.

2. Study was done over a period of 6 months (It should be from April 2014 to September 2014).

3. Although authors' acknowledge language barrier being one of the limitations of the study, I would like to know if C-ACT Questionnaire was translated in Hindi/ Kannada or English version was used? Getting it translated and answering in regional/ local language would have better validated results.

4. Whether clinicians taking patient history and performing clinical evaluation to assess asthma control were blinded to patient responses on the questionnaires to reduce the subjective nature of the questionnaire [3]?

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**AUTHOR'S REPLY**

1. Due to the limitations of FEV1 in the age group of below 6 years, both FEV1 and PEFR were used in this study, and their values were clinically interpreted and analyzed with GINA criteria (as GINA criteria allows the use of either).
2. We regret a typographical error in mentioning the duration of the study. The study was conducted from April 2014 to September 2014.
3. Indeed, the questionnaires were verbally translated by the person administering the questionnaire. Photographic representation of the responses provided as a feature in the questionnaire, aided in helping the children understand the questions and to optimally answer it. A written translated version was not used because the study sample was heterogeneous with respect to education level and the language spoken at home (varying from Hindi, Bengali, Tamil, Telugu and Kannada). Hence, to maintain uniformity, a standard English questionnaire was used, and was verbally translated appropriately according to the participant's needs. Further, translated versions would itself need a validation process before being used for the study.
4. One author was solely responsible for clinical evaluation and the other author was involved in administering the questionnaire.

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# Two Novel Missense Mutations in Very Long Chain Acyl-CoA Dehydrogenase Deficiency

Very long chain Acyl-CoA dehydrogenase deficiency (VLCADD) is an inherited metabolic disease caused by deleterious mutations in the *ACADVL* gene [1]. In this communication, we describe two novel mutations in a patient with VLCADD and his parents, and suggest a phenotype-genotype correlation.

The full term male infant had uneventful antenatal and perinatal history. His newborn screening was performed at 7 days of age in our facility. Analysis of blood acylcarnitine profile indicated markedly increased levels of long chain acylcarnitines, with significantly elevated C14:1 level of 2.17 µM. Blood samples from the infant and his parents were collected for genetic studies and mutation analysis, after informed consent. Genetic analysis of the infant's sample showed two heterozygous mutations in the *ACADVL* gene. Two novel missense mutations *ACADVL* NW_001270447: c.1768C>T (p.Arg590Trp) and *ACADV*L NW_001270447: c.1865A>G (p.Glu622Gly) were detected. The father was heterozygous for c.1768C>T (p.Arg590Trp) and the mother was heterozygous for c.1865A>G (p.Glu622Gly). Blood glucose, creatine kinase and alanine aminotransferase levels in the infant were normal, and the baby had no symptom or sign associated with VLCADD. As recommended [1], the parents were instructed to feed the baby boy every 3 h, to avoid fasting,