

Response: galactose treatment in focal and segmental glomerulosclerosis

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We want to thank the authors of this letter to the editor for their interest in our manuscript, “Effect of galactose on glomerular permeability and proteinuria in steroid-resistant nephrotic syndrome” [1]. We reported that the mean pre- and post-treatment urine protein:creatinine (UPC) ratios of a prospective study group of 7 children remained unchanged (15.5 ± 18.9 vs 20.8 ± 25.2 g/g respectively) after 16 weeks of oral galactose therapy, and none of the patients achieved partial (UPC 0.2–2 g/g) or complete remission (UPC <0.2 g/g). In response to the author’s suggestions, the UPC ratio data were re-analyzed using STATA 11.1 statistical software (StataCorp LP, College Station, TX, USA). The pre-and post-treatment median and interquartile range were 9.7 (3.0–30.6) vs 6.8 (3.9–53.6) g/g respectively. The difference was not significant, as assessed by the Wilcoxon signed-rank test ($z = -0.338$, $p = 0.73$). Therefore, we stand by our conclusion that no significant reduction in proteinuria was observed in response to galactose therapy in our study population. Furthermore, the clinical significance of reduction in proteinuria without achieving partial or complete remission, as reported by Mishra and Singh, is unclear [2]. In a recent NIH-funded multicenter randomized “clinical trial of focal segmental glomerulosclerosis in children and young

adults,” the only emphasis was placed on the achievement and maintenance of partial/complete remission, noted as the primary and secondary outcomes [3]. We would therefore be interested to learn of prospective data in other populations that may help further elucidate the potential for galactose to induce a clinically significant reduction in proteinuria, as demonstrated by partial or complete remission of disease.

References

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