

The GLOW Study does not light up the true Pediatric Endocrine Society recommendations for management of hypoglycemia in newborns



To the Editor:

Using intermittent plasma glucose testing combined with continuous interstitial glucose monitoring, Harris et al¹ demonstrate with unprecedented clarity, patterns of glucose concentrations in the first days of life in healthy term newborns. After a transitory decline following separation from the placenta, glucose concentrations increase in the first 18 hours, remaining stable at 59 ± 11 mg/dL for 48 hours, then rising to a new plateau of 83 ± 13 mg/dL by day 4. The authors report that by 72 hours a significant proportion of normal babies had at least one glucose concentration below the Pediatric Endocrine Society (PES) thresholds for managing at-risk babies.² We emphasize that the PES recommendations were not intended for normal, healthy asymptomatic infants; such infants do not require routine screening for hypoglycemia. This distinction is important because the data in Harris et al might be erroneously misinterpreted as demonstrating that the glucose thresholds in the PES recommendations are not applicable to at risk infants.

Comparing glucose thresholds developed for at-risk babies with glucose concentrations in normal healthy babies is inappropriate. At-risk babies in the PES recommendations include those with family history of hypoglycemia disorders, high or low birth weight, maternal diabetes, fetal distress in utero, pre-eclampsia, and possibly signs of hypoglycemia in newborns. Additionally, whereas the normal infants described by Harris et al were able to raise beta hydroxybutyrate to levels above 2 mmol/L³ at-risk infants are typically unable to raise ketone concentrations to this level.^{2,3} A low glucose concentration in such at-risk infants should prompt vigilance, possible investigations and treatment as outlined.²

We respectfully recommend to those caring for at risk infants that the report by Harris et al does not justify lowering the level of glucose that triggers concern leading to investigation of the etiology of hypoglycemia, nor does it justify lowering glucose treatment targets for at risk babies.

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References

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Reply



To the Editor:

We appreciate the interest of Thornton et al in the findings of the GLOW study. We entirely agree that our study reports findings from healthy babies, and that these cannot be extrapolated to drawing conclusions about management of babies at risk of hypoglycemia. Although we have placed our findings about glucose concentrations in the context of several international guidelines for treatment of at-risk babies, including those from the Pediatric Endocrine Society,¹ and have noted that low glucose concentrations are common in healthy babies, we do not suggest this might indicate a change in the care of at-risk babies. This is why we have stated that, "We are unable to determine if ... low glucose concentrations in healthy babies ... may be associated with impairments in later childhood." Until additional data are available, clinicians should continue to rely on existing guidelines for management of babies at risk of neonatal hypoglycemia.

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