

Increased hepatic fat after extremely preterm birth

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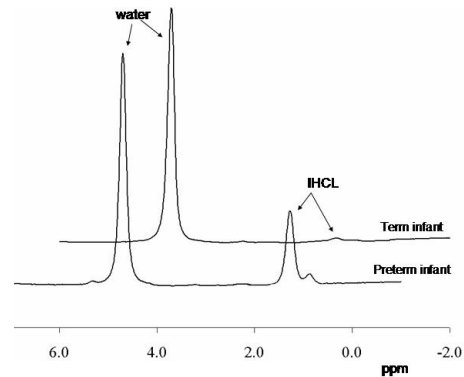
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Introduction: Nutritional support after preterm birth has largely been directed towards mimicking intrauterine growth and promoting catch-up growth, as poor growth, particularly poor head growth is associated with adverse neurodevelopmental outcome. However concern has recently been raised about the promotion of rapid weight gain in infancy¹. We have recently shown that adipose tissue (AT) distribution is altered in the preterm infant at term, with significantly increased intra-abdominal adiposity². However, little is known about the deposition of ectopic fat in these infants. The aim of this study was to apply ¹H MRS to investigate intrahepatocellular lipid (IHCL) deposition in preterm infants at the age of term.

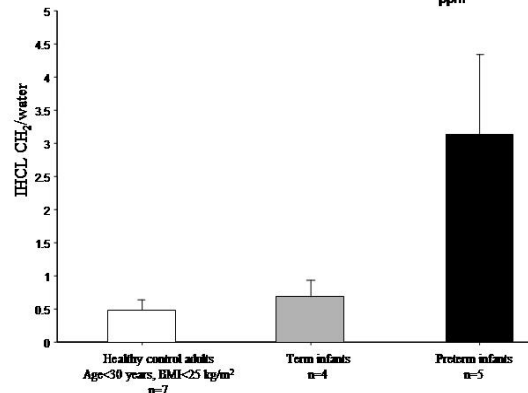
Methods: We studied 9 infants, 5 were preterm (gestational age: 30.34 ± 0.67 weeks), scanned at age term-equivalent and 4 term (GA: 40.04 ± 1.59 weeks). In parallel, IHCL data was obtained from a group of 7 healthy control adults, defined as BMI < 25 kg/m², age < 40 years, to ascertain potential age related differences in this fat depot.

MRI and MRS: All data were acquired on a 1.5T Eclipse multinuclear system. Whole body MR images were acquired and analysed using SliceOmatic. ¹H MR spectra were obtained from the right lobe of the liver using a PRESS sequence (TR 1500ms, TE 135ms) without water saturation. Spectra were analysed as previously described³, with IHCL measured relative to liver water content, after correcting for T₁ and T₂.

Results: Typical ¹H MRS spectra from a term and preterm infant are shown in Figure 1. Good quality ¹H MRS spectra were readily obtained from all infants. Term infants showed similar levels of IHCL (0.69 ± 0.47) as that observed in healthy control adults (0.51 ± 0.39; p=0.91). However, preterm infants had significantly elevated levels of IHCL (3.12 ± 2.70, p<0.05) compared with both term infants (p<0.01) and healthy control adults (p<0.01), Figure 2. The relative increase in IHCL deposition in the preterm infants did not appear to be associated with diet. Although some of the infants did receive parenteral nutrition, breastmilk comprised 94.1 ± 0.9% of total nutrition for 4/5 preterm infants. One preterm infant received a smaller proportion of breastmilk 40.9% compared with the others, but had one of the lower levels of IHCL. No association was observed between IHCL deposition and adiposity.



Discussion: In this study we have shown that IHCL can be readily detected in infants and that its relative level is similar to that observed in healthy control adults. Furthermore, the study shows that preterm infants appear to accumulate higher levels of this ectopic fat than control term infants and adult volunteers. The clinical relevance of increased lipid in the livers of preterm infants is unclear. The presence of increased liver fat has been previously reported in infant malnutrition⁴, however this is unlikely to be the cause in these well nourished preterm infants. It is possible that the increase in IHCL in preterm infants may reflect an overall alteration in the normal pattern deposition of fat, reflecting an abnormal lipid metabolism and/or adipose tissue development. Further work is underway to identify potential factors that may be responsible for this altered fat deposition in preterm babies and its longitudinal variation.



References:

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