systolic blood pressure (SBP), and diastolic blood pressure (DBP), and positively correlated with HDL-C. However, there is no relation between 25(OH) D and adiponectin levels among obese children and total sample.

Conclusion In spite of strong association between vitamin D and adiponectin levels with metabolic risk factors and obesity, there is no relation between 25(OH) D and adiponectin levels. In obese children, there are significant negative correlations between 25(OH) D with lipid profile, and between adiponectin levels with blood pressure. At certain adiponectin level, the relation between it and BMI disappears.

O-053 ADIPONECTIN IS ASSOCIATED WITH METABOLIC SYNDROME IN OBESE TAIWANESE ADOLESCENTS

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10.1136/archdischild-2014-307384.121

Background and aims Concurrent with the rise of the incidence in obesity, metabolic syndrome (MS) is increasingly prevalent in obese adolescents. Adiponectin is a major regulator of glucose and lipid homeostasis. Decreased adiponectin levels may be linked to MS. We aimed to test the hypothesis that adiponectin would influence the risk of MS in obese adolescents independent of its insulin sensitizer properties.

Methods This study was a cross-sectional study of the risk factors for MS in obese adolescents. A total of 593 obese subjects aged 10–18 years were recruited. International Diabetes Federation (IDF) consensus was used to define MS. We measured anthropometric, serum biochemical variables, serum adiponectin levels, and biomarkers for insulin resistance. We evaluated the independent influence of the adiponectin level on MS after controlling for the effect of insulin resistance measured by HOMA-IR.

Results 83 (14%) had MS. Obese adolescents with MS had significantly higher HOMA-IR and lower adiponectin levels than obese adolescents without MS. The adiponectin levels decreased with increasing number of metabolic syndrome components present in obese adolescents ($p < 0.001$). In multivariate logistic regression analysis, MS was significantly associated with age, body mass index, HOMA-IR, and adiponectin. For every 1 µg/mL decrease in serum adiponectin level, there was an increased risk of having MS with an odds ratio of 1.229 (95% confidence interval, 1.112 to 1.358, $p < 0.001$).

Conclusions Decreased serum adiponectin level is associated with an increased risk for MS independent of the effects of age, BMI, and insulin resistance in our population of obese Taiwanese adolescents.

O-054 NON-INVASIVE ASSESSMENT OF LIVER STIFFNESS AND HEPATIC FAT DEPOSITION BY FIBROSCAN IN JAPANESE OBESE CHILDREN

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10.1136/archdischild-2014-307384.122

Backgrounds and aims The aim of our study was to determine the usefulness and feasibility of transient elastography (FibroScan) assessing liver stiffness and hepatic fat deposition in obese children.

Methods Obese children (Obese group; BMI-SDS above 90th percentile) and non-obese children without liver disease (Control

group) were examined for liver stiffness measurement (LSM) with simultaneous controlled attenuation parameter (CAP) using FibroScan. LSM and CAP were compared with clinical, biochemical, ultrasound and histological data.

Results Obese group ($n = 40$, $12.4 \text{ y} \pm 3.6 \text{ y}$) and the control group ($n = 78$, $10.6 \text{ y} \pm 4.2 \text{ y}$) were evaluated for the study. Liver biopsy was performed in 5 patients. The CAP was significantly correlated with ultrasound fatty liver score ($r = 0.806$, $p = 0.028$) and histological steatosis grade ($r = 0.819$, $p = 0.016$). The LSM was significantly correlated with histological fibrosis grade ($r = 0.848$, $p = 0.005$). The CAP of the obese group ($293.27 \pm 55.72 \text{ dB/m}$) showed significantly higher value than control group ($179.45 \pm 44.75 \text{ dB/m}$) ($p < 0.0001$). The LSM showed significantly higher value in the obese group compared to the control group ($5.7 \pm 2.3 \text{ kPa}$ vs $3.9 \pm 0.9 \text{ kPa}$, $p < 0.0001$). In the control group, no biochemical parameters were correlated with LSM or CAP. In the obese group, the LSM correlated to the aspartate aminotransferase ($r = 0.694$, $p < 0.0001$) and the alanine aminotransferase ($r = 0.6748$, $p < 0.0001$), whereas the CAP correlated to no parameters.

Conclusions FibroScan is a non-invasive tool to assess the liver stiffness and hepatic fat deposition simultaneously thus useful as a screening tool for nonalcoholic fatty liver disease especially in obese children.

Neonatal Brain and Development Hypoxia – Ischemia

O-055 FRACTIONAL ANISOTROPY IN WHITE MATTER AND MEAN DIFFUSIVITY IN GREY MATTER CORRELATE TO NEURODEVELOPMENTAL PERFORMANCE FOLLOWING HYPOXIC-ISCHAEMIC ENCEPHALOPATHY

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10.1136/archdischild-2014-307384.123

Background and aim Biomarkers are needed to test novel neuroprotective therapies efficiently. The aim was to test the hypothesis that fractional anisotropy (FA) in white matter (WM) and mean diffusivity (MD) in grey matter (GM) correlate to subsequent developmental quotient (DQ) in infants with hypoxic-ischaemic encephalopathy (HIE).

Methods We studied 40 infants with HIE (median [range] age 39^{+5} [36^{+4} – 42^{+3}]), who underwent MRI within 21 days of birth and neurodevelopmental assessment at ≥ 12 months. Infants with a DQ > 2 SDs below the mean were considered to

Abstract O-055 Figure 1 Graphs showing receiver operating characteristic curves to estimate outcome with FA in WM (A), MD in thalami (B) and cortex (C)

Abstract O-055 Table 1 Results of receiver operating characteristics analysis to estimate outcome with FA in WM and MD in thalami and cortex. SE—standard error, CI—confidence intervals

	AUC	SE	Significance	95% CI
White matter FA	0.859	0.057	0.000	0.747-0.970
Thalami MD	0.775	0.077	0.003	0.625-0.926
Cortex MD	0.803	0.069	0.001	0.667-0.939

have an unfavourable outcome. Cortical, thalamic and WM tissue labels were generated by neonatal atlas-based automated segmentation of the T2-weighted images [1]. Tissue labels were propagated onto the diffusion maps. FA and MD values were determined. Linear regression was performed to assess the correlation between FA/MD and DQ. Estimation of unfavourable outcome with FA/MD in WM and GM was examined using receiver operating characteristic (ROC) analysis.

Results Following correction for multiple comparisons and age at scan significant correlation was found between DQ and FA in WM ($p = 0.0002$), and MD in thalami ($p = 0.0002$) and cortex ($p = 0.005$). ROC analysis to estimate unfavourable outcome showed larger area under the curve (AUC) for FA in WM, than MD in GM (Figure and Table).

Conclusion These data show that FA in WM and MD in GM may be used as biomarkers of outcome following HIE.

REFERENCES

1 Makropoulos *et al.* 2013

O-056

DOCOSAHEXANOIC ACID (DHA) GIVEN AFTER NEONATAL HYPOXIA-ISCHEMIA ATTENUATES LIPID PEROXIDATION AND BRAIN DAMAGE IN BOTH CORTICAL- AND WHITE MATTER TISSUES

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10.1136/archdischild-2014-307384.124

Background and aims Docosahexanoic acid (DHA) is a major component of membrane phospholipids and critical for fetal neuro-development. It accumulates during late pregnancy and may protect against oxidative damage to biomembranes. Isoprostanes, neuroprostanes, and neurofurans have all become attractive biomarkers of oxidative damage and lipid peroxidation in brain tissues. Lately also F₂-dihomo-isoprostanes have emerged as an *in vivo* biomarker of free radical damage to myelin in white matter.

Methods Global hypoxia was induced in newborn piglets (age 12–36 h) until Base Excess -20 mmol/L or mean arterial blood pressure <20 mmHg. One group ($n = 11$) was resuscitated with ambient air and another ($n = 10$) received in addition DHA 5 mg/kg 4 h after start of resuscitation. The piglets were followed for 9½ h after end of hypoxia.

Results Treatment with 5 mg/kg DHA 4 h after severe hypoxia significantly attenuated lipid peroxidation in tissues from cortex and hippocampus. There were less Isoprostanes in cortex and hippocampus in compared with reoxygenation with air (21%) alone, $p = 0.041$ in cortex and $p = 0.006$ in hippocampus. Neuroprostanes were lower in cortex in the DHA treated group, $p = 0.047$. F₂-dihomo-Isoprostane, an indicator of white matter damage, was significantly lower in tissue from hippocampus in the DHA treated group, $p = 0.035$.

Conclusions Treatment with DHA after severe neonatal hypoxia attenuates lipid peroxidation and brain damage in both grey- and white matter tissues. These novel findings add new knowledge on oxidative injury and points at a possible therapeutic intervention after perinatal asphyxia or severe hypoxia in both term and preterm babies.

Neonatal Brain and Development, Intervention and Outcome

O-057

MELATONIN AS A NOVEL NEUROPROTECTANT IN PRETERM INFANTS – A DOUBLE BLINDED RANDOMISED CONTROLLED TRIAL (MINT STUDY)

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10.1136/archdischild-2014-307384.125

Background Experimental studies suggest that melatonin is neuroprotective. Preterm infants are deprived of the normal intrauterine exposure to maternal melatonin and may benefit from supplementation to adult physiological levels.

Aim To prove that melatonin supplementation to adult concentrations decreases prematurity associated white matter injury assessed by tract based spatial statistics at term equivalent age.

Methods The study was a phase 2 exploratory; multi-centre double-blinded randomised placebo-controlled 2-arm trial, evaluating the neuroprotective effect of melatonin in preterm infants less than 31 weeks gestation. The 2 study drugs, melatonin (active) and normal saline (placebo) were given as an intravenous infusion once a day for 7 days starting by 48 h after birth. The dose of melatonin (0.1 mcg/kg/hr for 2 h) was derived from our previous pharmacokinetic study. Analysis was by intention to treat. Magnetic resonance imaging (MRI) was done at term corrected age. A 5% difference in the fractional anisotropy (FA) on MRI was taken as the primary endpoint.

Results Fifty-eight preterm infants participated in the study; 30 received melatonin and 28 received saline. Four babies died in each group. The 2 groups did not show any differences in the demographic data or in the short term adverse events. Seventeen infants from the melatonin group and 19 from the placebo group had MR images which were analysed. There was no difference in the FA in the 2 groups.

Conclusions Melatonin supplementation to adult physiological levels during the first week after birth did not alter FA in the preterm white matter at term equivalent age.

O-058

BRAIN MORPHOMETRY IN YOUNG ADULTS BORN SMALL-FOR-GESTATIONAL-AGE AT TERM

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10.1136/archdischild-2014-307384.126