Results We found significantly higher serum ADMA levels but not serum hs-CRP levels in NBF when compared to BF group (p < 0.05). According to BMI data starting from the age of 12 months more overweight/obese children were found in NBF children when compared to BF. Serum ADMA was inversely associated with HDL-cholesterol levels and breastfeeding duration in studied children (p < 0.05). Positive correlation was found between ADMA and body fat mass (p < 0.05).

Conclusion In NBF children increased circulating ADMA is observed, however further studies are needed to assess whether breastfeeding duration affects body fat and other measures of body composition at older ages.

Background and aims Anti cholinergic and sympathomimetic eye drops are widely used to achieve mydriasis. Normally systemic effects of these eye drops are ignorable but adverse events in preterm infants are reported. In this study during routine screening for retinopathy of prematurity (ROP), preterm infants were searched for the systemic effects of eye drops.

Methods The standard protocol was to instill 3 drops per eye which is a mixture of short acting tropicamide 0.5% with long acting cyclopentolate 1% and phenylephrine 2.5% ophthalmic solution in equal volumes. Each drop instilled at a 15 min interval before examination. Body temperature, heart rate, respiration, blood pressure, spO2, presence of flashing were recorded three times; before the instillation of eye drops, just after the examination and after an hour. Parents were informed about the adverse side effects and presence of complaints were asked after 24 h with the telephone interview. Data were analysed by 2 two-way ANOVA and independent samples t-test.

Results Forty eight (27 male+21 female) infants with birth weight 1498 ± 432(270–2500)g and gestational age 31.7 ± 3.3 (25–37)weeks were examined at postmenstrual age of 41.95 ± 4.74(34–37)weeks. Body temperature, heart rate, respiration, blood pressure, spO2, presence of flashing were recorded. Each drop instilled at a 15 min interval before examination. Body temperature, heart rate, respiration, blood pressure, spO2, presence of flashing were recorded three times; before the instillation of eye drops, just after the examination and after an hour. Parents were informed about the adverse side effects and presence of complaints were asked after 24 h with the telephone interview. Data were analysed by 2 two-way ANOVA and independent samples t-test.

Background and aims Rapid diagnosis of mitochondrial disorder is difficult in newborn infants with metabolic crisis. We studied whether respiratory chain disorder can be assessed from circulating platelets.

Methods A full-term girl of consanguineous parents was born after uncomplicated pregnancy (2690 g, Apgar 10/10/10). On day one she was transferred to NICU with metabolic acidosis (pH 7.11, pCO2, 2.8, BE -24, lactate 19 mmol/l). CSF/plasma lactate ratio (5.3/6.9) was increased. Cerebral MRI revealed diffuse changes in pyramidal tract and internal capsule. On day 4 she developed hepatic failure, conjugated hyperbilirubinemia and slight hyperammonemia. Urine metabolic analysis revealed increased 3-methylglutaconic acid (160 mmol/mol creatinine), and 4-hydroxyphenyllactate suggestive of mitochondrial disorder. Respirometry (Oroboros Oxygraph, SUTI-protocol) was performed on blood cells. Isolated mitochondria from fibroblasts and liver were assessed with Blue Native-PAGE (BN-PAGE) for respiratory chain complex assembly. Intensive care was withdrawn because of deterioration, and postmortem biopsies performed.

Results Respirometry on platelets showed a borderline oxygen consumption. Histology of muscle was normal, liver was cholestatic with iron accumulation. In fibroblasts, respiratory chain complex assembly was normal, but in liver levels of Complexes I, III and IV were decreased. Whole genome sequencing identified the candidate genes Sycp2, Clybl and Foxred1. The deficient complexes all possess mtDNA encoded subunits thus nuclear encoded translation mutation or other mtDNA related mutation might be causative.

Conclusions Respirometry from blood cells might suggest mitochondrial dysfunction that can be verified by structural analyses of respiratory chain complexes from the target organ. Causative mutation might be achieved with next generation sequencing.