Background and aims Quantitative measurement of brain maturation is increasingly performed in preterm infants using diffusion tensor imaging (DTI). To study white matter properly, reliability of underlying DTI data is of paramount importance, as acquisition and processing steps can substantially affect DTI analyses. We systematically reviewed literature to raise awareness regarding these matters.

Methods We systematically reviewed studies published between 1991 and September 2013, in which DTI scanning of preterm infants was performed within 28 days after term-equivalent age. Based on our inclusion criteria, 75 preterm DTI studies were considered relevant and further analysed. We primarily focused on use of dedicated neonatal equipment, DTI acquisition parameters and processing methodology.

Results There was wide variation among different studies in acquisition and processing methodology, and frequently incomplete reporting of these settings. 25.3% reported the use of dedicated neonatal equipment. Data quality assessment was not reported in 34.7%. Correction for artefacts and exclusion of datasets was not reported in 45.3% respectively 30.7%. Only 54.7% of the studies reported specific correction methods. Tensor estimation methodology was reported in 82.7%. Fast but less accurate tensor calculation algorithms were applied more frequently than advanced algorithms.

Conclusion DTI acquisition and processing settings are described incompletely in current literature, and vary considerably among different neonatal DTI research groups. In addition, described settings do not frequently meet the highest standards possible. Hence the prematurity population should be regarded as one of the most challenging groups to image using DTI, maximal awareness regarding these matters is a prerequisite.

Neonatal Immunity

Newborns, in particular preterm neonates, suffer a high frequency of microbial infections. MAIT cells are innate-like T cells expressing a semi-invariant Vα7.2-Jα33 TCR which recognises MICA/B-restricted, microbial-derived riboflavin (vitamin B2) metabolites unique to bacteria and yeast.

We studied 151 newborns admitted in the Neonatology Department at Robert Debré Hospital divided into four groups according to gestational age (group 1: 24–27 wks; group 2: 28–31 wks; group 3: 32–36 wks; group 4: >37 wks). The rate and kinetics of MAIT cell expansion and maturation were determined longitudinally at birth (day 0), day 3, day 30 and day 60. We performed multiparametric 10-colour flow cytometry analyses using combinations of antibodies to CD45, CD3, CD4, CD8, TCR Vα7.2, CD161, CD45RA, TCR Vα24 and TCRγ6 on 100 ml residual whole blood (left over of blood count), allowing characterisation of MAIT cells in parallel with other non-conventional and conventional T cells.

Our results show that the frequency of MAIT at birth is low and significantly differs according to gestational age (median at D0 group 1: 0.21%; group 2: 0.14%; group 3: 0.12%; group 4: 0.06%). Of note, this frequency remains relatively stable over the first 2 months of life. However, the phenotype of MAIT cell changes after birth with rapid maturation and increased proportion of CD8αα cells. Significant difference was observed between preterm neonates with and without perinatal infection. Analysis of MAIT cell frequency in 20 twin pairs showed it was very similar, suggesting that it might be controlled by a genetic and/or early environmental factor.

In conclusion, the frequency of MAIT cells at birth is inversely correlated with gestational age, and is correlated with the presence of perinatal microbial infection in preterm neonates. Whether it may reflect the presence of microbial products in amniotic liquid, and/or differences in the gut microbiota immediately after birth is under investigation.