received a single CBT and 124 (60%) had a double CBT. Thirty percent of CBU had 0-1 HLA mismatch (A, B, DRB1), 67% received Fludarabine and 71% received a double CBT. Median infused TNC was 3.7x10^7/kg. Pre-transplant serum was tested for HLA-Ab with a panel of fluorescent beads coated with single HLA-antigen using Luminex® platform. Results were interpreted as fluorescence intensity (MFI) against donor-specific mismatch (>1000 MFI was the threshold for positivity). Overall 48 pts (33%) had anti-HLA-Ab before CBT and those were donor specific anti-HLA-Ab (DSA) in 16 pts. Among the 16 pts withDSA (11 females, 5 males), 9 had single and 7 double-CBT (none had DSA directed to both CBT units). Seven pts had DSA vs to HLA-Class-I, 5 vs to HLA-Class-II and 4 to both HLA-Class-I and-II. DSA threshold ranged from 1620-17529 MFI. Results. Cumulative incidence (CI) of day-60 neutrophil engraftment was 78%. It was 44% for recipients with DSA and 81% in pts without DSA (p=0.006). There was no difference for pts with anti-HLA-Ab non donor-specific (77% vs 69%). Multivariate model showed DSA (RR 2.7, p=0.01) and CBT before 2008 (RR 1.49, p=0.03) independently associated with GF. Seven pts withDSA engrafted, 4 after double CBT and chimerism analysis showed the engraftment of the CBU with DSA in 1 case. Among 50 pts who failed engraftment, 9 (20%) pts had DSA specific for donor HLA-Class-I (n=4) or Class-II (n=2) or both Class-I and Class-II (n=3). CI of platelet recovery at day-180 was 62%, 12 of 16 patients with DSA did not achieve platelet recovery. CI of 1-year TRM was 35%. DSA was associated with higher TRM (p=0.002). Overall survival at 3-years was 44%, it was 41% and 45% for pts with non-malignant and malignant disease respectively. OS was 47% for recipients without DSA and 25% for those with DSA, p=0.006. In multivariate analysis, the absence of DSA was the only factor associated with better survival (RR 2.41, p=0.005). Conclusions. Donor-specific anti-HLA-Ab in recipients of CBT is associated with failed engraftment and lower survival. Screening for DSA may be included in the algorithm of donor choice for cord blood transplantation.

**Background.** The impact of the type of reduced intensity conditioning regimen used on immune recovery after allogeneic hematopoietic cell transplantation (allo-HCT) is poorly determined. Aims. We analyzed immune reconstitution in patients enrolled in a BHS-HCT sponsored randomized study comparing two non-myeloablative conditioning regimens for allo-HCT for which cell samples were prospectively collected. Patients and Methods. The conditioning regimen consisted of either 2 Gy TBI with 90 mg/m^2 fludarabine (=TBI arm, n=21), or 8 Gy TLI plus thymoglobulin (ATG) 7.5 mg/kg (=TLI arm, n=19). Median ages at HCT were 59 yrs and 61 yrs in the TBI and TLI arms, respectively. Immune reconstitution was assessed by flow-cytometry phenotyping, signal joint T-cell Receptor Excision Circle (sTREC) quantification, and T-cell spectratyping. Written informed consent has been obtained for each patient included. Results. Absolute T cell counts were lower in the TLI arm than in the TBI arm on day 28 after HSCt (P=0.04) but not thereafter. Further, B cells, as well as CD4+, CD4+CD45RA+ and CD4+CD45RO+ T cell reconstitution lagged behind in the TLI arm compared to the TBI arm the first year after HCT (B cells: p=0.0295 and others: p<0.0001). In contrast, reconstitution of CD8+ T cells, NK cells, Tregs and iNKt cells were similar in the 2 groups. For the thymic function, while sTREC levels were higher in the TBI arm than in the TLI arm on day 100 (P=0.002) and on day 365 (not significant) after HCT, the increase in sTREC levels from day 100 to day 365 was similar in the 2 groups of patients. The diversity of the TCR repertoire was similar in the 2 groups of patients on day 100 after HCT. Finally, we found that ATG persists in patients up to 17 days after allo-HCT in TLI patients (median of [ATG] at day 17=0.62 mg/l (TLI arm, n=19) while sTREC levels were higher in the TBI arm than in the TLI arm on day 100 (P=0.002) and on day 365 (not significant) after HCT, the increase in sTREC levels from day 100 to day 365 was similar in the 2 groups of patients. The diversity of the TCR repertoire was similar in the 2 groups of patients on day 100 after HCT. Finally, we found that ATG persists in patients up to 17 days after allo-HCT in TLI patients (median of [ATG] at day 17=0.62 mg/l and for one patient at day 20=0.53). Conclusions. These preliminary results suggest that ATG may be responsible for the delay of immune reconstitution of CD4+ T cells in the TLI arm. Furthermore, ATG probably destroyed grafted sTREC+ T cells, explaining the difference of sTREC levels at days 100 and 365 between the two groups while sTREC increment from day 100 to day 365 was similar in the 2 groups. Finally, TLI conditioning has no impact on immune regulatory populations (Treg and iNKt) after the transplantation.