

Despite cells directly
seems to be by



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Despite the developments in neonatology and improvements in technology to manage preterm neonates which increased the survival of preterm neonates, however, the relative risk of neonatal death is much greater for preterm infants than for full-term (1).

Neonatal sepsis and bronchopulmonary dysplasia are the most frequent causes of death in premature newborns (2). The immune response in preterm infants is affected by prenatal factors including in utero inflammation and maternal characteristics (3, 4). Regulatory T cells (Tregs) are a population of CD4⁺ T cells that play an important role in peripheral tolerance and control of immune responses to pathogens. They make up 5% to 10% of the normal CD4⁺ T-cell population and are characterized by the expression of the CD25 surface marker and the transcription factor forkhead box protein 3 (Foxp3) (5). Tregs play a vital role in maternal-fetal tolerance, and their levels are increased during pregnancy (6, 7).

Tregs participate in abrogating immune responses and prevent exacerbated and harmful immune activation. However, Tregs may inhibit the antimicrobial immune responses and lead to ineffective clearance of pathogens resulting in persistent infection (8). Tregs were divided based on their expression of CD45RA. CD4⁺FoxP3⁺ CD45RA⁺ cells were described as naive or resting Tregs, while CD4⁺FoxP3⁺CD45RA⁻ cells were regarded as fully functional effector Tregs. CD4⁺FoxP3⁺ CD45RA⁻ cells are cytokine-secreting, non-suppressive T cells (9).

The naïve subset are de novo generated cells which are recently released from the thymus and have not yet experienced antigen exposure (10). Tregs

express several molecules associated with their suppressive function.. About 40 % of the Tregs show surface HLA-DR expression.. HLA-DR+ Tregs expressed induced a more intense and a more rapid T cell suppression than the Tregs that lack HLA-DR expression 11. Other way for Tregs to suppress effector T cells directly seems to be by transferring cyclic adenosine monophosphate (cAMP) into the responder cells 12. cAMP is a potent inhibitor of proliferation, differentiation and IL-2 synthesis in T cells 13.

CD39 is an ectoenzyme that degrades ATP to AMP and may be involved in suppression mediated by Tregs. CD39 is expressed by all Treg in mice and to a lesser and variable extent in humans 14. There are previous studies of the expression of activation and memory markers (CD45RO, CD69, HLA-DR, CD25) on total cord blood CD4+ cells in neonates 15, 16 however, there is no studies about the expression of these markers on cord blood Tregs There is little information about immunity in neonates and the capacity of their immune systems against infections especially in preterm infants. The immune response of preterm infants is suspected to be immature; however, the data on differences in immune capacity between preterm and full-term infants are scant.

The aim of this study was to study Tregs and their expression of CD45RA, HLA-DR, and CD39 in newborn infants.