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Cord blood vitamin D status: evidence for altered $CD4^+$ naïve and gut-homing T cell accumulation

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The active form of vitamin D, 1,25-dihydroxyvitamin D3 ($1,25(OH)_2D3$), has potent immunomodulatory properties that have prompted its consideration in the prevention of immune-mediated diseases. Recent evidence indicates that the gut is the primary site of tolerance induction where homing receptor switch from $\alpha4\beta7$ towards a memory phenotype occurs. However, the role of vitamin D in these processes remains unclear.

The aim of this study is to assess the association of vitamin D status to immune function at birth.

Umbilical cord plasma concentrations of 25(OH)D3 were measured at birth ($n = 47$) and related to a number of cord blood immunological endpoints: (1) enumeration of T, B, NK, iNKT, pDC, mDC cells; (2) skin and gut homing T cells; (3) $CD4^+CD25^+FoxP3^+$ regulatory T cell numbers; (4) cytokine responses to LPS and mitogen (PHA) and (5) neonatal total IgE levels.

Neonatal 25(OH)D3 was positively associated with the levels of gut homing $CD4^+CCR9^+$ T cells ($r = 0.436$, $P = 0.026$). Similar, though not statistically significant, trends were seen for $CD8^+CCR9^+$ T cells ($r = 0.365$; $P = 0.067$). Neonatal 25(OH)D3 was not correlated with either skin-homing (CLA^+) or $CD4^+CD25^+FoxP3^+$ regulatory T cell expression. TNF-α and TNF-β release after stimulation with T cell mitogen PHA, were both inversely related to neonatal 25(OH)D3 (TNF-α: $r = -0.48$, $P = 0.01$; TNF-β: $r = -0.43$, $P = 0.02$). Furthermore, IL-10 release after LPS stimulation was weakly negatively correlated with 25(OH)D3 ($r = -0.35$, $P = 0.07$).

These findings suggest that neonatal 25(OH)D3 alters $CD4^+$ gut-homing ($CCR9^+$) and naïve ($CCR7^+CD45RA^+$) T cell receptor expression and pro-inflammatory (TNF) cytokine production which could impact on neonatal tolerance mechanisms.

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