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3280 - Category: CELLULAR IMMUNOLOGY (CE)
The Impact of Vitamin D Deficiency on Thromboinflammation in Individuals with Obesity

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Obesity is a chronic low-grade inflammatory condition associated with increased thrombotic risk. Vitamin D deficiency is frequently observed as a factor that aggravates the inflammatory and procoagulant profile in obese individuals. Our aim was to investigate the influence of vitamin D on tissue factor (TF) and inflammatory mediator secretion by monocytes in different pro-inflammatory contexts related to obesity. We analyzed TF expression in monocytes, neutrophils and plasma, as well as plasma thromboinflammatory markers, correlating these findings with vitamin D levels. We also evaluated the release and cellular origin of pro-coagulant extracellular vesicles (EVs) ($AnnV^+TF^+$) using flow cytometry. Plasma exchange experiments exposed whole blood from healthy donors to plasma from obese or eutrophic individuals, with or without vitamin D deficiency. Finally, inflammatory stimuli (lipopolysaccharide (LPS) and leptin) were applied in vitro to monocytes from obese and eutrophic subjects, in the presence or absence of $1,25(OH)_2D_3$ and secretion of TF, IL-1β, IL-8 and TNF-α was measured by ELISA. The study was approved by the ethics committee 5.736.117. Results demonstrated that individuals with obesity and vitamin D insufficiency had higher TF expression in monocytes and increased release of monocyte-derived procoagulant EVs. Moreover, plasma from obese individuals with vitamin D deficiency induced higher TF expression in monocytes from healthy donors compared to plasma from eutrophic individuals, suggesting a role for soluble procoagulant mediators. The effects of inflammatory stimuli on TF, IL-1β, IL-8 and TNF-α secretion by monocytes from obese individuals stimulated with LPS or leptin were reduced by vitamin D treatment. These findings support that the obesity inflammatory microenvironment, exacerbated by vitamin D deficiency, contributes to thromboinflasmmatory pathways and that vitamin D may modulate monocyte procoagulant responses.
Keywords: Obesity;Immunometabolism;Vitamin D.

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IL-10 Modulates Metabolism and Dendritic Cell Function During Efferocytosis of Staphylococcus aureus-Infected Cells

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During Staphylococcus aureus methicillin-resistant (MRSA) skin infection, neutrophils engulf the bacteria and then undergo apoptosis, promoting the accumulation of MRSA-infected apoptotic cells (MRSA-iAC). Previous studies have shown that efferocytosis of apoptotic cells (AC) induces metabolic reprogramming in macrophages. However, the impact of iAC-efferocytosis on cell metabolism and its regulation of DC function remains to be investigated. Our group demonstrated that MRSA-iAC efferocytosis reduces mitochondrial respiration and causes mitochondrial dysfunction in DC after 6h. However, after 18h, we observed partial restoration of mitochondrial dysfunction and an increase in IL-10 production. It has been shown that IL-10 regulates mitochondrial integrity and glycolysis in macrophages in the presence of LPS. Therefore, we hypothesized that IL-10 may play a role in restoring mitochondrial function and modulating DC activation during the efferocytosis of MRSA-iAC. BMDC from WT and IL-10-deficient (KO) mice were co-cultured with MRSA-iAC, and the role of IL-10 in metabolism and DC activation was evaluated by Flow cytometry, ELISA and RT-PCR. MRSA-iAC efferocytosis increased MHC-II, CD86, and PDL-1, while IL-10 deficiency reduced only MHC-II and CD86. MRSA-iAC also enhanced IL-1β, IL-6, TNF-α, and IL-10 production; IL-10 absence further increased TNF-α and IL-1β but did not affect IL-6 or nitrite. Also, IL-10–deficient DCs did not affect mitochondrial mass and had a minimal impact on mitochondrial membrane potential; however, after 18h, the deficiency of IL-10 showed an accumulation of dysfunctional mitochondria during MRSA-iAC efferocytosis, compared to WT. On the other hand, the absence of IL-10 increased Glut1 expression during MRSA-iAC efferocytosis, compared to control and DC+AC. Taken together, our findings suggest that IL-10 downregulates DC activation, suppressing the glycolytic pathway, and also impairs the accumulation of dysfunctional mitochondrial. Keywords: Dendritic Cells; Immunometabolism;IL-10.