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addition not only to the current vaccine against Hepatitis B virus but also other vaccines that require the induction of a more diverse range of immune responses to provide protection.

Platelet activating factor and its association with other inflammatory mediators in the pathogenesis of dengue

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Objective: Platelet Activating Factor (PAF), was shown to be an important mediator of vascular leak in acute dengue. Therefore, we set out to investigate the factors that lead to PAF production and its association with other severity markers in acute dengue. **Methods:** PAF production by primary human monocytes was assessed by co-culturing dengue virus (DENV) infected monocytes with lipopolysaccharide (LPS) levels reported in patients with acute dengue, or dengue immune sera at varying concentrations. Gene expression analysis was done for MAPK1, MAPK3, MAPK14, NLRP-3, TLR-4, NF-ΚB, RIG-I.
Results: PAF was only produced by DENV infected monocytes in the presence of LPS and was not induced by the infection alone or in the presence of dengue immune sera. However, IL-6, IL-10 and TNFα were induced in DENV infected monocytes in the absence of LPS and also in the presence of dengue immune sera. PAF, IL-6, IL-10 and TNFα levels were significantly higher in DENV infected monocytes co-cultured with LPS, than monocytes co-cultured with LPS alone. Although the NFKβ-1 pathway is known to be involved in inducing proinflammatory cytokines and PAF, only RIG-I was significantly up regulated ($p < 0.05$) when DENV infected monocytes were co-cultured with LPS. MAPK1, MAPK3, MAPK14, NLRP3 or TLR-4 mRNA not induced by DENV infection of monocytes or by co-culturing DENV infected monocytes with LPS.
Conclusion: Although LPS appears to act synergistically with the DENV to induce PAF and other cytokine production, the mechanisms by which this occurs needs further investigation.

The atypical chemokine receptor ACKR4 shapes early antibody responses

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B cell differentiation during T-dependent antibody responses proceeds down extrafollicular plasmablast (EFPB), germinal centre B (GCB) cell or early GC-independent memory B (EBM) cell pathways that differ in their spatio-temporal emergence, effector function and cellular longevity. Adoption of these fates is controlled to a large extent by B cell trafficking receptors, which are dynamically expressed following antigen-engagement enabling access to antigen, interactions with T cells, and positioning in distinct functional niches within secondary lymphoid organs that promote rapid or long-term antibody production. In this study, we reveal a novel role for Atypical Chemokine Receptor 4 (ACKR4, gene name Ccrl1) in regulation of EBM/EFPB/GCB cell responses during T-dependent humoral immune responses. Chimeric mice restricting ACKR4-deficiency to the hematopoietic compartment formed enhanced T-dependent EFPB and GC responses. Using a combination of competitive mixed bone-marrow chimeras,
B cell-specific μMT bone-marrow chimeras and the SW_{HEL} BCR transgenic system, we demonstrate that B cell-intrinsic ACKR4 expression functions to limit PB, EBM and GCB cell responses prior to fate trifurcation of antigen-engaged B cells. Accordingly, forced expression of Ccrl1 in B cells using novel $Cd23^{Cre/+}Ccrl1^{Tg/o}$ mice reduced T-dependent EFPB and GC

