

Poster presentation

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P10-09. Altered release of RANTES from human, normal platelets: contribution of distinct HIV-1MN gp41 peptides

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Background

Blood platelets link the processes of haemostasis and inflammation. Platelets can further, sense danger by the display of e.g. TLR, and our group demonstrated that they can even discriminate between danger signals in order to secrete cytokines/chemokines differentially. This study examines the secretion of normal platelets after exposure to recombinant HIV-1MN-gp120 or gp41 peptides.

Methods

We used platelets sampled from healthy, HIV-1,2 negative, normal blood donors. Platelet-Rich Plasma (PRP) was incubated after addition thrombin receptor agonist peptide or HIV env glycoprotein. PRP were centrifuged and platelet free supernatant was stored. Two recombinant gp120MN (produced in insect cells using the baculovirus expression system) and synthetic peptides of gp41MN, and mAbs to gp41: D50 and 2F5, were all obtained through the AIDS Research and Reference Reagent Program. Soluble CD62p and RANTES production in cultures were measured by specific ELISA.

Results

Platelets (n = 10) with various HIV-1MN gp120 or gp41MN peptides had no significant effect on sCD62P release. None of two recombinant gp120MN tested caused any modulation of RANTES release. In contrast, the stimulation of platelets with gp41 peptides evidenced that peptides 2025 (GKLICTTVPWNASWSNKSL) and 2031 (LLELDKASLWNWFDITNWL) led to a significant

reduction of RANTES release, whereas the other 8 tested peptides were ineffective. RANTES production could be significantly restored if platelet cultures with peptides 2025 and 2031 were performed in the concurrent presence of D50 or 2F5 mAb anti-gp41.

Conclusion

This data indicates that certain peptides of HIV env-gp41 exert a negative signal, the precise nature of which remains to be ascertained and further investigated, for RANTES production by normal platelets in *in vitro* stimulation conditions. Our data provide novel information on possible primary interactions between platelets and HIV in their early encounter in the circulation. Therefore, further studies are needed to clarify its consequence: fair or – more likely – foe?