6. CONCLUSIONS

1. We found that mast cell stimulation can induce PS externalization in the absence of secretory response.
2. We identified GPI-APs as molecules whose engagement can induce sustained and reversible non-apoptotic PS externalization.
3. GPI-AP-induced PS externalization was determined as non-apoptotic and distinct from the FcεRI-induced PS externalization.
4. The effect of multiple triggering on PS externalization was additive and dependent on a type of stimulus and cells engaged.
5. We identified PLSCR1 as a molecule that becomes tyrosine phosphorylated in mast cells stimulated through GPI-APs.
6. We found that the PLSCR1 tyrosine phosphorylation is not associated with mast cell secretory response, and with GPI-AP- or FcεRI-induced non-apoptotic PS externalization.
7. Using confocal microscopy and electron microscopy visualization of PLSCR1 in the course of mast cell activation we found that PLSCR1: (1) is not co-localized with externalized PS, (2) is not co-localized with aggregated Thy-1.1 or FcεRI, and (3) does not form self-aggregates.
8. We developed a modified one-tube semi-nested PCR-ELISA. The modified assay showed higher sensitivity and specificity than the conventional hybridization-based and a modified semi-nested-based PCR-ELISA. Due to its versatility and robustness, the modified assay is suitable for routine diagnostics of target DNA sequences in clinical as well as other specimens.