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PKCε expression is required during proplatelet formation in murine model

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Megakaryocytes (MK) remodel their cytoplasm into long proplatelet extensions to generate platelets [1]. We have previously demonstrated that PKCepsilon expression is strictly regulated during megakaryocytopoiesis (MKpoiesis), and its forced expression in the late phases of MK differentiation impairs platelet production [2,3]. However, our preliminary data suggest that PKCepsilon positive platelets may be released around the acute event of myocardial infarction, affecting their aggregation potential. Primary fetal liver (FL) cells isolated from CD1 pregnant mice are the preferential model to study the platelet formation mechanism [4]. Therefore, here we focused on the mouse PKCepsilon positive model to elucidate the role of PKCepsilon in MK maturation.

Our data show that not only PKCepsilon expression increases during mouse MK differentiation, but also its forced down-regulation strongly reduces pro-platelet formation. Therefore, PKCepsilon is strongly required for murine proplatelet production. On the basis of these results and other known model systems, we show that PKCepsilon has a relevant role in the completion of platelet release.

References

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