not clear whether this is significant in an activated platelet. The evidence that panPKC inhibitors abolish phosphorylation of syntaxin as well as secretion is indicative that syntaxin phosphorylation could be important for secretion.³

In conclusion, we emphasize that our data² are supported by a recent independent study⁴ that confirms a positive regulatory role for PKCθ in platelets.

Reference for the studies. Informed consent was provided in accordance with the declaration of Helsinki.

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To the editor:

β1-tubulin gene mutation platelets are not macrothrombocytes

The brief report by Kunishima et al¹ describing a mutation of the β1 tubulin gene affecting microtubule assembly is of interest, but data presented do not support association of the genetic defect with macrothrombocytopenia. The authors stated both the male propositus and his mother had prominent appearance of giant platelets on peripheral blood smears, and provided light (Figure 1A¹) and electron micrographs (Figure 1B¹) to support that finding. The patient platelet in Figure 1A¹ resembles balls of yarn.³ The studies of the mutation in the present study are of interest. However, the statement “W318 β1-tubulin may interfer with normal platelet production, resulting in macrothrombocytopenia”¹¹ is not supported by the data presented.

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References

Response

W318 β1-tubulin and macrothrombocytopenia

We would like to thank Dr White for valuable comments on our study that reported the first human β1-tubulin mutation associated with congenital macrothrombocytopenia.¹ We have been working on congenital macrothrombocytopenia and analyzed more than 200 cases. We do not think that giant platelets from macrothrombocytopenia syndromes are necessarily larger than red blood cells. The mean platelet sizes...
β1-tubulin gene mutation platelets are not macrothrombocytes

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