BMC Pharmacology

Poster presentation

NO/cGMP signalling in platelets

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from 4th International Conference of cGMP Generators, Effectors and Therapeutic Implications
Regensburg, Germany. 19–21 June 2009

Published: 11 August 2009

BMC Pharmacology 2009, **9**(Suppl 1):P62 doi:10.1186/1471-2210-9-S1-P62

This abstract is available from: http://www.biomedcentral.com/1471-2210/9/S1/P62

Background

NO/cGMP-signalling is important for the inhibition of platelet aggregation. The cGMP dependent protein kinase I plays thereby the key role in the signal cascade of NO/cGMP. It is already known that the interaction of the cGKI substrate IRAG with the IP_3RI is essential for the NO/cGMP dependent inhibition of platelet aggregation. To specify the relevance of IRAG signalling for platelet function, IRAG deficient murine mutants were analysed. Diverse agonists like thrombin, thromboxane A2 and collagen induced platelet aggregation and granule secretion.

Results

In wild-type platelets the activation was inhibited by NO-donors and a cGMP-analogue whereas in IRAG-deficient platelets the inhibiting effect of NO/cGMP was almost abolished. Furthermore NO/cGMP dependent inhibition of agonist-induced activation of the fibrinogen receptor GPIIb/IIIa and the resulting fibrinogen binding was strongly reduced in IRAG deficient platelets compared to wild-type platelets.

Conclusion

These findings delineate the predominant physiological role of IRAG for the NO/cGMP dependent inhibition of platelet aggregation and granule secretion.