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L. Di Bella, M.T. Rossi, L.Gualano, G. Scalera
(Cattedra di Fisiologia gen. Istituto di Fisiologia Umana, Via G. Campi 287, 41100 Modena, Italy).

Molecular mechanism of bone marrow thrombocytogenesis by melatonin.

Melatonin (MLT) promotes platelet outcome from rat's bone marrow megacaryocites (MG) in vitro (Di Bella et al. Boll. Soc. It. Biol. Sper., 1979, **55**, 318-330; 389-393). MLT, moreover, with stands rat's and man's platelet aggregation, as promoted by ADP addition (Di Bella et al., l.c., Com. 54, 68, 114; Cardinali, Melatonin: ..., Pergamon Press, 1980, 247-256).

Both effects may be at least partly identical, inasmuch as they are exerted on the same membranes of platelets which are not at all or hardly any more agglutinated. However , not all points over the external membrane of MG seem to be identical, at least where they unite to shut up the cytosol of the future platelet. These sites seem to be in a position as to coalesce with the next borders of the demarcation membrane system, when MLT reaches a topically adequate concentration.

Indeed in the presence of NAT inhibitors plus MLT, a multitude of forming platelets gather together on the surface of MG. This means not only that NAT is extant on the same point over the surface membrane of MG, but moreover that 5-methoxytryptamine is active in thrombocytopoiesis only when it is N-acylated (=Melatonin). The 5-methoxy-group is less important in this occurrence than N-acetyl group, inasmuch as HIOMT inhibitors are far less active on MLT induced thrombocytopoiesis than NAT inhibitors. Probable MLT sites are present on platelet membrane too; their role resides, however, in reversibly changing the platelet shape and inhibiting the secondary action of ADP on platelet aggregation. NAT and HIOMT inhibitors play therefore a negligible role in platelet aggregation.