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α - MSH inhibits human platelets aggregation.

Melatonin (MLT) inhibits man and rat platelet aggregation by ADP (1, 2).

The action is probably dependent upon the binding of MLT with tubuline microtubules (3).

MLT is on the other hand a powerful melanophore contracting agent (4) and a potent antagonist to α -MSH, which is on the contrary a darkening agent.

Melanophore has indeed at least two receptors (5) that trigger dispersion of melanosome. The two critically important residues of the α -MSH molecule being histidine and tryptophane at position 6-10 in the molecule (6) both of which can be antagonized by an isolated indole, such as MLT, when it binds with the receptor.

The α -MSH receptor is probably located on the cell membrane, since it can be washed out with Ringer solution (5). Acetylcholine can reverse MSH-induced darkening (7), and its turnover is increased by α -MSH in the rat's hippocampus (8). Melatonin induces a correspondingly vivacious platelet output from rat's megacaryocytes in vitro in the presence of N-acetyl-transferase inhibitors (NAT: 2, 3, 1, 6) (9).

The binding of Melatonin and α -MSH with the same receptor, on different cell kinds has pushed us to investigate whether α -MSH, at the same degree as Melatonin, is in a position to influence platelet aggregation in vitro.

The following main results have been as yet obtained:

- 1) α -MSH induces a disaggregation of human PRP;
- 2) Platelet aggregation by ADP can be more or less inhibited according the molar concentration ratio of ADP to α -MSH;
- 3) At the same molar ADP/ α -MSH ratio the aggregation value grows so more, so longer is the incubation length of time of PRP with α -MSH, from two to five minutes;
- 4) Both Melatonin and α -MSH inhibit platelet aggregation at concentrations of the same order;
- 5) 3.4×10^9 molecules of α -MSH can efficiently protect human platelet against aggregation by whatever high concentrations of ADP.

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α -MSH: Melatonin: Platelet aggregation.