TILIPO 33, A MEMBER OF LIPOCALIN FAMILY, IS A POTENT INHIBITOR OF COLLAGEN-INDUCED PLATELET AGGREGATION

[1]Torquato, R.J.S., [1]Lovato, D.V., [1]Pimentel, C.C., [2]Santoro, M.L., [1]Tanaka, A.S.

[1]Departamento de Bioquímica, UNIFESP-EPM, São Paulo, SP, Brazil. [2]Laboratório de Fisiopatologia, Instituto Butantan, São Paulo, SP, Brazil.
E-mail: torquato. bioq@epm.br

Vertebrates developed many mechanisms, such as vasoconstriction, platelet aggregation and blood coagulation, to control blood losses after vascular injury. Platelet activation is a process, which can be initiated by several agonists, e.g. ADP, thrombin, collagen and PAF. Platelet aggregation is one of the challenges that hematophagous animals have to deal with during blood meals. Triatomines control this process by producing a protein family named lipocalin in their saliva. The present work shows the characterization of a potent inhibitor of collagen-induced platelet aggregation, named tilipo 33, from *Triatoma infestans* salivary glands. Previously, we have described the expression and purification of four different lipocalins from a cDNA library of *T. infestans* salivary glands, named tilipo 33, 37, 39 and 77. Among them, tilipo 33 and 37 were expressed in high levels and were well characterized. They were expressed by *E coli*, in protein concentrations of 3-4 mg/L, and purified by affinity and size exclusion chromatographies. Purified tilipo 33 showed a strong inhibitory activity (IC_{50} ~60 nM) on platelet aggregation induced by 10 µg of collagen, whereas tilipo 37 could not inhibit it. On the other hand, tilipo 33 did not inhibit convulxin-induced platelet aggregation, and slightly affected that platelet aggregation induced by ristocetin. **Supported by: FAPESP/CNPq.**