Marinobufagenin (MBG), a bufadienolide cardiac inotrope, is a component gaining more and more interest in the early diagnosis of volume expansion-mediated hypertensive states.

This endogenous mammalian vasoconstrictive compound, is a selective inhibitor of the $\alpha_1$ subunit of Na$^+$,K$^+$-ATPase making it implicated in series of pathophysiological circumstances and leading to hypertension and natriuresis. Indeed, increased plasma MBG has been observed in mammals presenting volume-expansion, especially when it is associated with fluid retention, like in preeclampsia (PE) [1-3].

In an animal model of PE, some authors have demonstrated that the urinary excretion of the circulating cardiotonic steroid MBG is elevated prior the development of the syndrome [4]. These observations have even been confirmed in human patients [5-6], leading us to consider MBG as a biomarker for PE.

This consideration implicates an accuracy and sensitive analytical method of MBG plasma levels in order to further investigate the implications of MBG in PE, and to establish a diagnosis for this syndrome.

Given that no standard compound of MBG already exists and that the main source for MBG is located in the parotid and skin gland secretions of the toad *Bufo Marinus*, we have developed a successful extraction method of MBG from the crystallized venom of this toad.

In order to quantify MBG levels, a pre-extraction step from rat and human plasma has been carried out through a solid phase extraction (SPE) HLB (hydrophilic lipophilic balanced) cartridge with an extraction yield of 88%.

The reversed-phase LC-UV method to allow quantifications of MBG in the nanomolar range is under development.

References