ISOLATION AND PURIFICATION OF A ANGIOTENSIN-CONVERTING ENZYME POISON OF THE THALASSOPHRYNE NATTERERI

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The study of venoms animals being done mainly in order to understand its deleterious actions in humans, thus enabling the development of specific treatments. Furthermore, there is in the venom of many animals the presence of substances with therapeutic potential (Lewis and Garcia, 2003). The Thalassophryne nattereri belongs to Batrachoididade family. Fish of this family are known as sapo-fish due to its appearance (Froese et al, 2008).

The angiotensin converting enzyme (ACE) (dipeptidyl carboxypeptidase, CD143 EC 3.4.15.1) is a key enzyme of the renin-angiotensin system that regulates blood pressure. It is a zinc-dependent peptidyl dipeptidase with a broad spectrum of effects. Removing the His-Leu dipeptide in the C-terminal, ACE converts angiotensin I (Ang I) to angiotensin II (Ang II) and inactivates bradykinin, regulating thereby the blood pressure and electrolyte homeostasis. As a result, ACE has become a promising target in the treatment of hypertension, heart failure, and diabetic nephropathy (Zhang et al., 2013).

Fractionation the in venom of T. nattereri in Hitrep CMFF16 / 10 showed a peak (CM1), with angiotensin-converting activity, converting Ang I to Ang II. The 12% polyacrylamide gel revealed a band for CM1.

The molecular mass estimated for the band CM1 fraction is close to 40 kDa, approaching much the size of natterinas, toxins found in the venom of T. nattereri. The isolated protein also shown to be inhibited by captopril and the EDTA and is characterized as a classic angiotensin converting enzyme.

Thus, the enzyme isolated and purified from the venom of T. nattereri shows a different physiological effect of the other studied poisons, causing a hypertensive effect on the victim.

These results strongly suggest that T. nattereri employs compounds which cause disruption of cardiovascular function as part of its complex poisoning strategy leading to the production of bioactive peptides.