The neurotoxic phospholipases A_2 ammodytoxin and crotoxin bind to distinct high-affinity protein acceptors in Torpedo marmorata *electric organ.* I. Križaj,[1,*] J. Grazyna Faure,[2] F. Gubenšek[1] and C. Bon[2] ([1]Department of Biochemistry and Molecular Biology, J. Stefan Institute, Jamova 39, 1000 Ljubljana, Slovenia; and [2]Unité des Venins, Institut Pasteur, 25, Rue du Dr Roux, F-75724 Paris Cedex 15, France).

This study investigated the binding of radioiodinated ammodytoxin C (AtxC), a monomeric phospholipase A_2 (PLA_2) neurotoxin from *Vipera ammodytes*, and of crotoxin, a dimeric PLA_2 neurotoxin from *Crotalus durissus terrificus*, to presynaptic membranes from the electric organ of *Torpedo marmorata*. In both cases, two different families of specific binding sites were identified and characterized. The high-affinity binding sites for both toxins are proteins. In contrast, the low-affinity binding sites were not affected by proteinases, heat or low pH, suggesting the involvement of certain lipid structures in this type of specific binding. By affinity-labelling, ^{125}I-AtxC was shown to be associated predominantly with membrane proteins of apparent mol. wts of 70,000 and 20,000 and weakly with several proteins of apparent mol. wt ranging between 39,000 and 57,000. ^{125}I-Crotoxin was mainly bound to a 48,000 mol. wt membrane protein. All PLA_2s tested, except β-bungarotoxin, inhibited the low-affinity specific binding of AtxC, whereas only neurotoxic PLA_2s and surprisingly, myotoxic PLA_2-analogue ammodytin L, were able to prevent AtxC and crotoxin high-affinity binding and their cross-linking. The inhibition profiles of high-affinity binding and cross-linking were quite different for ^{125}I-crotoxin and for ^{125}I-AtxC. AtxC and crotoxin did not inhibit each other, indicating that they do not share the same high-affinity binding sites on *Torpedo* membranes. In contrast with crotoxin, the isolated basic subunit CB of crotoxin was able to inhibit completely the high-affinity binding of ^{125}I-AtxC. Therefore, the acidic subunit CA of crotoxin does not simply act as a chaperone for the CB subunit, but it also confers a distinct binding specificity to the crotoxin.

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Venom of the hunting spider Cupiennius salei (*Ctenidae*). L. Kuhn-Nentwig and W. Nentwig (Zoological Institute, University of Berne Baltzerstrasse 3, 3012 Berne, Switzerland).

The tropical hunting spider *Cupiennius salei* is easy to breed in captivity, lives for about 1 to 2 years and produces a venom which can be milked at quantities of approx. 10–15 μl every 3 weeks from adult animals. Venom production depends on age, gender and degree of hunger, and probably on reproductive status. Insects and other arthropods as the main prey group are sensitive to the venom at astonishingly different levels. Dipterans are among the most sensitive taxa; crickets, moths and some roaches are also very sensitive. Some beetles, roaches and ants are extremely insensitive to the venom, all tested species ranging over 4 log units. Our hypothesis was that this different reaction to spider venom is caused by physiological properties of the haemolymph of the prey item. *Cupiennius salei* injects only small quantities of venom into a given prey but is able to control the amount of venom injected. According to the size, activity or defensive potential of the prey item the spider releases subsequently more venom quantity. The venom contains several dozen peptides, of which 13 (mol. wt 3000–13,000) were found to have toxic properties. The most common toxin, CSTX-1, covers about half of the total toxicity and consists of 74 amino acids. CSTX-2 has 13 amino acids less, the first 61 amino acids being completely identical to CSTX-1. Since the toxicity of CSTX-2 is only 1/20 of that of CSTX-1 it was assumed that the 'tail' of 13 amino acids and the corresponding tertiary structure cause the toxicity of this peptide. Among these 13 amino acids are seven lysine, therefore this amino acid is extremely important for the toxicity of the crude venom. The toxic effect of CSTX-1 is caused by an inhibition of high-threshold Ca^{2+} channels (L-type) of glutamatergic synapses (Cruz, J., Lacerda Beirao, P. S. and Leao, R. M., unpublished). The crude venom of *C. salei* also contains other non-toxic peptides (unknown function), a very active hyaluronidase (spreading factor) but no protease, amino acids, histamine, free polyamines, other low mol. wt substances or ions (possible synergistic functions).

Enzyme analysis of Atheris *snake venom.* D. Mebs,[1] A. Fach[1] and H.-W. Herrmann[2] ([1]Zentrum der Rechtsmedizin, University of Frankfurt, Frankfurt, Germany; and [2]Kölner Aquarium am Zoo, Cologne, Germany).

The venom of the African green bush viper (*Atheris squamiger*) was found to exhibit a strong coagulant activity, when fibrinogen was used as substrate, causing incoagulable blood in a patient. Several other venom samples (from a single specimen) from *A. squamiger* and from *A. chlorechis*, *A. desaixi*, *A. hispidus*, *A. nitschei* and *A. superciliaris* were tested for the following enzyme activities: L-amino acid oxidase, casein and BAEE-hydrolysis, kallikrein, phosphodiesterase, phospholipase A_2, fibrinogen coagulase, haemorrhagic and myotoxic activity. Surprisingly, all venoms tested possessed remarkably high coagulant activity on fibrinogen. Although symptoms in human snakebite cases reported in the literature seem to be rather mild (with the exception of *A. squamiger* bites, where a fatal case has been described), these snakes should be considered to be capable of causing potentially severe envenoming. Biochemical studies on the coagulant enzyme and its inhibition by commercial antivenoms will be reported.