CHAPTER 2
REVIEW OF LITERATURE
2.1 *Bungarus caeruleus* (Common Indian krait)

Indian Krait measures 1.2 to 1.73m in length and is lustruous black or bluish black above with paired narrow white crossbars indistinct or absent anteriorly. *Bungarus caeruleus* can be easily identified by its enlarged hexagonal vertebral scales, entire sub caudals, uniform white belly and narrow white cross bars on the back. Indian krait’s habitat is wide range of biotopes in peninsular India from Sind (Pakistan) to West Bengal plains and in the south upto the cape Srilanka. It is commonly encountered in fields, low scrub jungles and in the vicinity of human habitation and dwellings (REID 1968), and its bite is often fatal to man. Venom glands present in the temporal region of the head secrete a clear, amber coloured fluid. Venom yield appears to depend on the condition of the snake however independent on its size varying between 0.2 to 5.4mgm of dried venom (Deoros, 1965). Krait is one of the deadliest venomous snakes among the poisonous snakes of the world (Haast etal 1953).

Changing trends in toxinological research in the last 3 decades has reported significant research contributions to gain an insight into the chemically structure of the toxin and its structure function relationships (Zeller, 1948. 1951; Meldrum, 1965a, Bouquet, 1964, 1966; Sarkar and Devi, 1968; Jimenes-Porras, 1970.

Master and Rao (1962) performed starch gel electrophoresis of the venoms of Indian krait and India saw scaled viper (*echis carinatus*) and
identified the presence of certain toxins and enzymes namely 5’ Nucleotidase, Coagulase and Phosphodiesterase.

Schnider singh et.al (1970) have reported the presence of multiple sex chromosome in the common Indian krait (*Bungarus caeruleus*). Bon C and Changeux J.P in (1975) have reported the presence of caeruleotoxin, an acidic neurotoxin from the venom *Bungarus caeruleus* capable of blocking the response to a cholinergic agonist without binding to the cholinergic receptor site.

Lee C.Y., et.al reported chromatographic separation of the venoms *Bungarus caeruleus* and pharmacological characterization of its components in 1976.

Kehoe et.al in 1976 reported their research on the effects of alpha toxins from Bungarus multicintus and *Bungarus caeruleus* on cholinergic response in aplysia neurons.

Bon C and Changeux J.P in 1977 reported their research findings on the chemical and pharmacological characterization of toxic polypeptides from the venom of *Bungarus caeruleus*.

In the same year 1977 Abe T et.al isolated and characterized presynaptically acting neurotoxin from the venom of Bungarus snakes.
In 1978 Chatwal G.S and Gaitonde B.B purified a non toxic PLA₂ from the venom of Indian krait *Bungarus caeruleus*.

In the same year 1978 Moody T.W and Raftery M A characterized polypeptides neurotoxins from the venom of *Bungarus caeruleus*.

Again in 1978 Painter S.D and Greenberg M.J reported their research findings that elapid α toxins had no effect on the cholinergic responses of bivalve myocardia.

Noble R.L et.al in 1979 compared the venom of *Bungarus caeruleus* with venom from which putative cholinergic ionophore marker was isolated.

In the decade from 1990 to 2000 Tan N.H and Ponnudoria G in 1990 performed a comparative study of the biological properties of krait (*Bungarus caeruleus*).

In the same year 1990 a comparative study between a common krait and Srilankan cobra (Naja naja naja) on envenomation and efficacy and complications of therapy with Haffkine antivenom was reported.

In 1999 Sharma S et.al isolated, purified crystallized and reported their preliminary x ray analysis of β 1 bungarotoxin from *Bungarus caeruleus* (India krait).
In the same year Selvanayagam Z.E. et.al reported their ELISA findings for the detection of venoms from 4 medically important snakes of India.

Again in 1999 Sigh G et.al reported neuromuscular transmission failure due to common *Bungarus caeruleus* envenomation,

Singh G et.al in the year 2001 determined the sequence and crystal structure of a basic PLA$_2$ from common krait (*Bungarus caeruleus*) at 2.4 a resolution and identified and characterized its pharmacological site.

Kularatne S in the year 2002 reported a prospective clinical study on common krait (*Bungarus caeruleus*) bite in Anuradhapura Srilanka.

### 2.2 Pathophysiology of snake envenomation

Elapidine venom produces severe tissue lesions and necrosis and is thus "myotoxic". Enzymatic or non-enzymatic, myotoxic venom components are extremely potent amongst the most active, known natural products (D. Mebs, 1986). Venom components on purification produce numerous pathophysiological conditions like hemorrhagea, edema, cardiopulmonary abnormalities and myonecrosis (myotoxicity).

M. Homma, A.T Tu (1971) have demonstrated experimental snake envenomation. Myotoxic activity of Phospholipase A2 isolated from snake venoms and their neutralization by polyvalent antivenoms have been reported (D. Mebs 1986).
Coral snake phospholipase A2 induced skeletal muscle damage has been reported by (O. Arroyo et.al 1987). Tissue necrosis phenomena is of specific clinical interest, because studies have shown that anti-venom doesn’t completely prevent the same.

C.L Ownby et.al (1983) have tested the ability of prairie rattlesnake antiserum to neutralize local myotoxicity and also the lethal effects of myotoxin. Snake venoms are also known to alter significantly, normal renal functions progressing into kidney failure in experimental models and also in snake bite victims and thus are said to be nephrotoxic in nature. (P.A Limpa., 1999 V Sitprija, 1999 B.V Mittal 1994).