

Overview of animal toxins.

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Abstract: Animal toxins are well recognized for their toxicity. Poisoning is through direct contact, bites or by consumption. Approximately 5 million snake bites, scorpion stings and anaphylactic reactions to insect stings occur worldwide annually, causing over 100,000 human deaths each year mostly in the tropics. Comparatively, clinical data on marine toxins are few. Animal toxins are complex consisting of polypeptides, enzymes and chemicals which can cause injury. At least 3 mechanisms are involved in pathophysiology of venom poisoning. Direct injury is induced mostly by chemicals, enzymes and polypeptides. Polypeptides exert their effects through the action on ion channels and receptors on the cell membrane. The action of polypeptides on ion channels and receptor sites through signal transduction causes the release of mediators which usually results in neuromuscular effects. Enzymes can cause membrane lysis, pore formation, cytoskeletal destruction, release inflammatory and vasoactive mediators and activate the coagulation pathway at various levels. Proteolytic enzymes and phospholipase A₂ are important causes of cellular injury and coagulopathy. Indirectly, vasoactive mediators and proinflammatory cytokines induced by enzymes can lead to haemodynamic alterations and cause organ injury. Cardiovascular symptoms are therefore common. They can also trigger the inflammatory process with generation of adhesion molecules, complement activation, acute phase proteins, free radicals, increased vascular permeability and increased blood viscosity. Local reactions include pain, swelling and redness. Haemodynamic changes may result if the process is severe. Immunologic reaction usually results in the form of allergic response such as skin rashes and oedema. In the severe form anaphylaxis with respiratory and cardiovascular symptoms may occur.