Channelopathy and Essential Needs to Search in Venomous Animals for Toxins

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Channelopathy as a new word in medical vocabulary describes those human diseases that result from defects in ion channel function. Ion channels as a pathway for the movement of ions across cell membranes play essential roles in the physiology and pathophysiology of all cells and it is therefore not very surprising that an ever increasing number of human diseases have been found to be caused by defective ion channel function that we may need to search for medicine to treat them. To understand this we need to know at least a basic understanding of the genetics, molecular structure, biophysical properties and physiological role of ion channels. Further more, ion channels play an important role in transmitting information in the nervous system. Na\(^{+}\) and K\(^{+}\) channels are involved in action potentials, and at nerve endings, channels selective for Na\(^{+}\), K\(^{+}\) and Ca\(^{2+}\) ions are involved in action potentials and the release of neurotransmitters.

Considering this point, venoms or poisons and toxins which are pure fractions isolated from each of them could be used as pharmacological tools to identify and study physiological role of ion channels, as well as introducing the different types or sub-type of ion channels activity in cell membranes. Nevertheless, they may play a medicinal role to treat different channel related diseases.

In the past, biophysical and pharmacological studies of some of the post-synaptic receptors and ion channels have been aided by the use of potent and selective toxins, e.g. α-bungarotoxin to isolate and characterize the nicotinic acetylcholine receptor-ion channels complex. Similarly, tetrodotoxin and saxitoxin have been fundamental to the characterization of Na\(^{+}\) channels in neurons. Until fairly recently, however, characterization of neuronal K\(^{+}\) and Ca\(^{2+}\) channels has been restricted by a lack of suitable selective and high affinity probes. Characterization of these ion channels and ionic currents at nerve terminals is particularly important, because, by regulating the frequency and duration of action potentials, the K\(^{+}\) and Ca\(^{2+}\) channels can regulate nerve terminal excitability and transmitter release. Because Na\(^{+}\) channel-mediated action potentials are essential to the functioning of the nervous system, it is perhaps not surprising that many toxins exist, which act at the Na\(^{+}\) channel. In contrast to Na\(^{+}\) channels, many types of K\(^{+}\) channels exist in neurons. There are three main classes based on the kind of stimulus required to convert them from the closed to the open configuration. They include voltage-dependent K\(^{+}\) channels, K\(^{+}\) channel regulated by intracellular “messengers” like Ca\(^{2+}\) and K\(^{+}\) channels which can be activated by neurotransmitters, e.g. serotonin or acetylcholine. Besides, we should never forget recent reports related to the effects of the bee venom on the treatment of rheumatoid arthritis or Multiple Sclerosis. The variety of venomous animals and their subtypes from snakes, scorpions, spiders in earth, and bees in air to puffer fish and sea anemonia in oceans, open a large research area to distinguish the effects of venom and toxins on human cell, which may help us to obtain a novel drug to treat different diseases related to channel functions.

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