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Sommario/riassunto

The cytoplasmic free Ca2+ concentration ([Ca2+]i) is a key determinant of neuronal information transfer and processing. It controls a plethora of fundamental processes, including transmitter release and the induction of synaptic plasticity. This enigmatic second messenger conveys its wide variety of actions by binding to a subgroup of Ca2+ binding proteins (CaBPs) known as "Ca2+ sensors". Well known examples of Ca2+ sensors are Troponin-C in skeletal muscle, Synaptotagmin in presynaptic terminals, and Calmodulin (CaM) in all eukaryotic cells. Since the levels of [Ca2+]i directly influence the potency of Ca2+ sensors, the Ca2+ concentration is tightly controlled by several mechanisms including another type of Ca2+ binding proteins, the Ca2+ buffers. Prominent examples of Ca2+ buffers include Parvalbumin (PV), Calbindin-D28k (CB) and Calretinin (CR), although for the latter two Ca2+ sensor functions were recently also suggested. Ca2+ buffers are distinct from sensors by their purely buffering action, i.e. they influence the spatio-temporal extent of Ca2+ signals, without directly binding downstream target proteins. Details of their action depend on their binding kinetics, mobility, and concentration. Thus, neurons can control the range of action of Ca2+ by the type and concentration of CaBPs expressed. Since buffering strongly limits the range of action of free Ca2+, the structure of the Ca2+ signaling domain and the topographical relationships between the sites of Ca2+ influx and the location of the Ca2+ sensors are

central determinants in neuronal information processing. For example, postsynaptic dendritic spines act to compartmentalize Ca2+ depending on their geometry and expression of CaBPs, thereby influencing dendritic integration. At presynaptic sites it has been shown that tight, so called nanodomain coupling between Ca2+ channels and the sensor for vesicular transmitter release increases speed and reliability of synaptic transmission. Vice versa, the influence of an individual CaBP on information processing depends on the topographical relationships within the signaling domain. If e.g. source and sensor are very close, only buffers with rapid binding kinetics can interfere with signaling. This Research Topic contains a collection of work dealing with the relationships between different [Ca2+]i controlling mechanisms in the structural context of synaptic sites and their functional implications for synaptic information processing as detailed in the Editorial.