Chapter 2
Compartmentalization of Synaptic Tagging and Capture

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Abstract Testing of the synaptic tagging and capture (STC) hypothesis has produced remarkable work on the understanding of how a single neuron undergoes spatial and temporal encoding of information. Central to this work is the notion that STC processes can be compartment specific. Formed by activation of synaptic plasticity mechanisms and extending along confined dendritic domains, these compartments can work as the neuron’s information integration units. Association or dismissal of incoming information would depend on the plasticity-driven functional state of the compartment. With multiple streams of neural activity arriving at distinct synapses of a neuron, compartmentalization emerges as a key strategy to organize this information and enhance the neuron’s computing capability.

Keywords STC model • Synaptic plasticity • Compartmentalization • Neuronal integration • Information coding • Memory

2.1 Why Compartmentalization?

Information carried by distinct neural paths in the brain is integrated to generate a vast number of cognitive processes, including representations of thoughts, recollections, planning, and actions. A typical neuron in the brain receives thousands of synaptic afferents. These synaptic connections can convey relevant information from other neurons. Hence, a single neuron can potentially code thousands of streams of information, with each synapse acting as a unique integrative unit (Yuste 2013). This immense computing capability emerges as one of the most remarkable features of the neuron’s anatomy.

In addition to single synapse computation, it is also suggested neurons integrate information within confined dendritic areas comprising large groups of synapses, referred to here as compartments. Compartmentalization, is the process by which

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the biochemical and biophysical properties of a particular dendritic region (compart-artment) are modified by plasticity mechanisms. Synaptic function within the compartment will be largely conditioned by the molecular setting imposed by these plasticity mechanisms. As a result, each compartment will process distinct streams of information arriving to its synapses according to the unique molecular environment of the compartment. Streams of information that arrive at distinct compartments will be encoded in terms of the particular molecular history experienced by each compartment (Sajikumar and Korte 2011b). The potential combinations of different streams of neural activity and different compartments highlight the computational capability of compartment-specific integration of information. If compartmentalization is a mechanism for processing information, then, two important questions arise: (1) how do compartments form? and (2) what functional purpose do they serve?

2.2 STC and Compartmentalization

Clues to a compartmentalized nature of the STC process came from the characterization of synaptic capture of long-term synaptic potentiation (LTP), a form of LTP associativity, between two separate synaptic inputs located within and across the two main dendritic projections of CA1 pyramidal neurons of the rodent hippocampus, basal and apical dendrites (Alarcon et al. 2006; Fonseca et al. 2004; Sajikumar et al. 2007a, b). Prior to this work, LTP associativity had been studied primarily between two separate sets of synapses within apical dendrites in the stratum radiatum of CA1 pyramidal neurons (Barco et al. 2002; Frey and Morris 1997, 1998). Nothing was known of the properties for STC at CA1 basal synapses. Synaptic capture of LTP between two sets of synapses within the basal dendrites occurred just as it did for apical synapses suggesting that LTP associativity processes are functionally similar in both types of synapses (Alarcon et al. 2006). However, LTP associativity, in the form of synaptic capture of LTP, did not occur when tested between synapses located in separate apical and basal dendrites (Alarcon et al. 2006). The failure of synaptic capture of LTP across the basal and apical dendrites led to a fundamental notion: LTP associativity, and perhaps all of the STC processes, can be compartment specific; that is, restricted within domains of the neuron’s dendritic arbor. Further work supported this notion by demonstrating the associative properties between LTP and long-term synaptic depression (LTD) can also be compartmentalized (Pavlowsky and Alarcon 2012; Sajikumar et al. 2005).

These findings led to a revision of the original STC model. In its original form, the STC model posits that upon activation of transcription and somatic protein synthesis, plasticity products would be distributed throughout the cell and then productively incorporated into activity-tagged synapses. The notion of a compartmentalized STC process suggests that there could be activity-dependent mechanisms that restrict the availability of the plasticity products to confined dendritic domains; possibly through selective cellular sorting or local protein synthesis. Dendritic sorting
and local translation would allow plasticity products, such as mRNAs granules and proteins, to be used by particular groups of tagged synapses within a confined dendritic domain (Alarcon et al. 2006; Fonseca et al. 2004; Govindarajan et al. 2006; Pavlowsky and Alarcon 2012; Sajikumar and Frey 2004; Young and Nguyen 2005).

2.3 Making a Compartment

The formation of dendritic domain-restricted compartments is thought to depend on two main factors: (1) the activation of synaptic plasticity-associated mechanisms, and (2) the geometry of the dendrite (Alarcon et al. 2006; Govindarajan et al. 2006, 2011; Makino and Malinow 2011; Sajikumar et al. 2007a). Additionally, organelle architecture within dendrites can greatly impact the shape and function of a compartment (Cui-Wang et al. 2012; Makino and Malinow 2011).

Induction of synaptic plasticity largely depends on the activation of Ca\(^{2+}\)-mediated mechanisms (Colbran and Brown 2004; Fitzjohn and Collingridge 2002; Sjostrom and Nelson 2002; Zucker 1999). Not surprisingly, the Ca\(^{2+}\) signal turns out to be the primary modulator of synaptic plasticity-induced associative processes between separate synapses of a neuron (Abraham et al. 1994; Christie and Abraham 1999; Christie et al. 1995). Key to the associative properties of synaptic plasticity may be the control of Ca\(^{2+}\) propagation within the neuron (Nishiyama et al. 2000; Raymond and Redman 2002, 2006; Sajikumar et al. 2009). Induction of synaptic plasticity that activates ryanodine receptors (RyRs) releases intracellular Ca\(^{2+}\) within stimulated synaptic spines. A stronger synaptic stimulation that activates inositol-3-phosphate receptors (IP\(_3\)Rs) propagates Ca\(^{2+}\) within dendritic branches. A much stronger synaptic stimulation that leads to L-type voltage-dependent calcium channel (VDCC) activation mediates a large Ca\(^{2+}\) influx comprising the entire somatodendritic area, including the cell nucleus (Raymond and Redman 2002, 2006). Hence, synaptic activation propagates Ca\(^{2+}\) within a neuron in an activity-dependent manner. This mechanism may be a primary factor for establishing compartment size.

Upon synaptic activation, the Ca\(^{2+}\) signaling pathway is engaged with another second messenger path: the cAMP signaling pathway. Indeed, Ca\(^{2+}\) and cAMP signaling paths activate calcium/calmodulin-dependent protein kinase II and PKA activity, respectively-protein kinase activities known to be important for STC processes (Barco et al. 2002; Young et al. 2006). Importantly, these protein kinase activities can be spatially restricted by membrane anchoring and protein clustering. For instance, spatial restriction of cAMP/PKA signaling via binding to A kinase-anchoring proteins (AKAPs) may be critical for compartmentalization (Huang et al. 2006; Nie et al. 2007). Compartmentalized protein kinase activity might, in turn, restrict the distribution and access of plasticity products to specific dendritic regions forming a compartment (Horton et al. 2005; Li et al. 2014).

Synaptic stimulation can trigger long-lasting remodeling of the actin network at both pre and postsynaptic sites (Colicos et al. 2001). The activity-mediated recruitment of cytoskeleton structures by kinase activity might enhance the transport of
mRNAs and proteins to dendritic compartments containing stimulated synapses (Kotz and McNiven 1994; Luo 2002; Rodionov et al. 2003; Sanchez et al. 2000). Activity-dependent transport of RNA granules containing a number of plasticity proteins and mRNAs depends on the expression and activity of the motor protein kinesin (Kanai et al. 2004; Kiebler and Bassell 2006; Puthanveettil et al. 2008). For instance, the activity-regulated cytoskeleton-associated (Arc) protein and mRNA accumulates selectively in activated dendritic domains (Steward et al. 1998). Another example is the transport of *Aplysia* elongation factor 1A (Ap-eEF1A) mRNAs to neurites after synaptic stimulation (Giustetto et al. 2003).

### 2.4 Consolidating a Compartment

Because compartment formation is a process that depends on the induction of synaptic plasticity, synaptic plasticity and compartmentalization may also share common mechanisms for consolidation. The recruitment of new protein synthesis, which is already established for synaptic plasticity, is proposed to be the chief mechanism that underlies compartment consolidation (Alarcon et al. 2006; Pavlowsky and Alarcon 2012). Compartmentalization presumes that new synthesis of protein is localized to confined dendritic domains. Indeed, local protein synthesis can be spatially restricted by the capture, activation, and translation of dormant mRNAs (Aakalu et al. 2001; Klann and Dever 2004; Ostroff et al. 2002; Richter 2001; Sutton and Schuman 2006). Protein synthesis may be locally regulated by: translocation of ribosomes into particular dendritic branches and spines, localized activation of the mammalian target of rapamycin (mTOR) pathway, localized activation of translation factors, or localized presynaptic release of brain-derived neurotrophic factor (BDNF) (Banko et al. 2005, 2006, 2007; Casadio et al. 1999, Costa-Mattioli et al. 2005, 2007; Kelleher et al. 2004).

The consolidation of compartments capable of STC processing may require new synthesis of protein. However, the long-term maintenance of a compartment may just require adjusting the rate in translation efficacy (Klann and Dever 2004). After the initiation of new protein synthesis and compartment consolidation, each compartment could adjust and exhibit a functionally defined state of translation efficacy by regulating the rate of local protein synthesis and degradation (Fonseca et al. 2006; Martin and Kosik 2002). The existence of compartments might significantly increase the efficacy of active (tagged) synapses to store information by enhancing pathways of intracellular trafficking, efficiency of mRNA translation and protein capture that otherwise would be degraded without use.

### 2.5 Functional Compartmentalization

Compartment formation, consolidation, and maintenance are not seen as immutable, irreversible processes. The size of a compartment would primarily depend on the magnitude of synaptic activation (Alarcon et al. 2006; Sajikumar et al. 2007a)
and the morphology and excitability properties of dendritic branches (Makara et al. 2009). Hence, compartmentalization can be thought to be a function of (1) the activity of plasticity mechanisms recruited within a particular dendritic area (Alarcon et al. 2006), and (2) the temporal dynamics of these mechanisms (Pavlowsky and Alarcon 2012). Therefore, the magnitude of a compartment would be defined by the activity function of the recruited plasticity mechanisms. We could visualize this activity function (wave) with its apex at the point of maximal synaptic stimulation and then extending from this center, possibly in a Gaussian-like manner, across a particular dendritic area. Increases in synaptic activation would give rise to corresponding increases in the activity function/wave and, consequently, compartment size. Importantly, this notion suggests that compartment size and compartmentalization have no fixed boundaries.

In this context, a way to define a compartment would be through its category of functional properties. For instance, electrotonic changes triggered by synaptic activation can be spine specific, branch specific, or extend across multiple dendritic branches (Frick et al. 2004; Johnston et al. 2003; Zhang and Linden 2003). Synaptically driven signaling pathways can be localized via dendritic translation (Aakalu et al. 2001; Purcell et al. 2003) or surpass dendritic boundaries and extend as far as the nucleus (Kandel 2001). A functional compartment would be operationally defined as a particular region of the dendritic arbor with modified excitability and selective access to plasticity products where late-associative processing can occur (Alarcon et al. 2006; Fonseca et al. 2004; Govindarajan et al. 2006; Pavlowsky and Alarcon 2012; Sajikumar and Frey 2004; Young and Nguyen 2005).

### 2.6 Compartmental Computation

A neuron can be activated by distinct learning-associated information streams. During initial experience, such activation elicits synaptic plasticity at a subset of synapses, a phenomenon thought to be the cellular substrate of memory (Bliss and Collingridge 1993; Kandel 2001; Malenka and Bear 2004; Neves et al. 2008). One can imagine that learning can prompt the storage of different bits of information in multiple synapses of a neuron by means of synaptic plasticity. These bits of synaptic plasticity-encoded information must be correctly associated or segregated at the cellular level to properly contribute to the neuronal ensemble that constitutes the memory engram.

STC is a process that allows the interaction between multiple forms of synaptic plasticity induced at separate synapses within a single neuron. STC compartmentalization presents an attractive mechanism for the correct integration of separate streams of information arriving at the same neuron within time periods ranging from minutes to hours (Alarcon et al. 2006; Frey and Morris 1997, 1998; Sajikumar et al. 2007a; Young and Nguyen 2005). This dynamic process might be important for the association of relevant and dismissal of irrelevant information in a setting where information encoding is done over longer periods of time. This kind of temporal integration has the behavioral connotation that allows information experienced
distantly in time to be either associated or segregated; and it adds to the welleducated mechanisms for cellular integration at the sub-second and seconds time frames by associativity models of synaptic integration (Magee 2000; Magee and Johnston 2005).

As previously described, the induction of plasticity-associated metabolic activity within dendrites can give rise to functional compartmentalization. These functional dendritic compartments may be key to the proper association and segregation of learning-associated information and its decoding (Alarcon et al. 2006; Fonseca et al. 2004; Govindarajan et al. 2006; Morris 2006; Reymann and Frey 2007; Sajikumar et al. 2007a; Young and Nguyen 2005). For instance, neurons in the CA1 area of the hippocampus form part of different ensembles and circuits that underlie behavior (Amaral and Witter 1989; Kramar and Lynch 2003). CA1 principal neurons receive inputs from different brain areas to morphologically defined dendritic domains which are potential substrates for compartmentalization: basal dendrites (within the stratum oriens) receive information from the contralateral hippocampus; proximal apical dendrites (within the stratum radiatum) receive ipsilateral afferents from neighboring CA3 neurons via Schaffer-collateral fibers; and distal apical dendrites (within the stratum lacunosum-moleculare) receive inputs from layer III of the entorhinal cortex via the temporo-ammonic pathway (Amaral and Witter 1989; Deuchars and Thomson 1996; Dolleman-Van Der Weel and Witter 1996; Ishizuka et al. 1990; Pikkarainen et al. 1999). The anatomical distinction between these dendritic domains is also functional as they differ on their biophysical properties (Arai et al. 1994; Cavus and Teyler 1998; Haley et al. 1996; Jarsky et al. 2005; Kawakami et al. 2003; Kloosterman et al. 2001; Kramar and Lynch 2003; Leung and Shen 1999; Nicholson et al. 2006). Inputs to these dendritic domains may contain spatial, relational, and other relevant forms of information that need to be integrated for proper encoding of memory traces. Compartment specificity might enable a neuron to compare information arriving into its distinct functional compartments from different brain areas and estimate its relevance.

A proposition for compartmentalization and memory encoding at the single neuron level is the “clustered plasticity” hypothesis (Govindarajan et al. 2006). The hypothesis posits that information encoding occurs within clusters of synapses located within particular dendritic branches. Physical distance, morphology and structural restrictions would constrain the spreading of plasticity products between clusters of synapses located at separate dendritic branches. Synapses within each cluster might be tagged by different plasticity-inducing stimuli, but only the strongest plasticity-dependent metabolic cascade would dominate the molecular environment of the cluster. Branch-specific local translation mechanisms would be key to this process (Govindarajan et al. 2011). This branch-specific protein synthesis-dependent homogenization of synaptic weights of a synaptic cluster would produce a more efficient action potential firing during recall compared with conventional dispersed plasticity models (Govindarajan et al. 2006, 2011).

Compartment-specific homogenization of synaptic weights is seen in the interaction between LTP and LTD, two opposite forms of synaptic plasticity (Han and Heinemann 2013; Pavlowsky and Alarcon 2012). Plasticity-induced protein synthesis
underlies the antagonistic interaction between LTP and LTD elicited at two synaptic inputs to the same dendritic region in CA1 principal neurons of the mouse hippocampus (Pavlowsky and Alarcon 2012). Interestingly, compartment-specific interactions between LTP and LTD can also be cooperative (Pavlowsky and Alarcon 2012; Sajikumar and Frey 2004). The type of interaction, antagonistic or cooperative, is regulated in a time-dependent fashion (Pavlowsky and Alarcon 2012). Antagonistic interactions, which disfavor the coexistence of LTP and LTD, occur when both forms of synaptic plasticity are induced within less than an hour from each other. Cooperative interactions between and coexistence of these opposite forms of synaptic plasticity begin to be seen after a longer time interval between inductions (Pavlowsky and Alarcon 2012).

How could the temporal control of compartment-specific interactions lead to proper information encoding? Interactions among different forms of synaptic plasticity may underline a form of competition by synapses and memories for access to retrieval resources (Diamond et al. 2005). Compartmen tal interference between learning-associated plastic events that occur within nearby time frames could provide a mechanism for disruption of unwanted information. Activity-dependent disruption of unwanted information seems to be a step necessary for the stabilization of a memory trace (Levy and Steward 1979; Martin and Morris 2002; Thomas et al. 1994; Villarreal et al. 2002; Xu et al. 1998). This mechanism is thought to prevent adding existent but irrelevant information to a memory experience. But this restriction may not be permanent. After a period of time, once the relevant plasticity-associated trace is consolidated, another one could be associated with it (Pavlowsky and Alarcon 2012).

### 2.7 Compartmental Encoding

In the hippocampus, encoding of information at specific compartments is mainly defined by the anatomy of hippocampal circuits (Amaral and Witter 1989; Kramar and Lynch 2003; Morris 2006). Changes in neural activity that modulate hippocampal oscillations (e.g., theta, gamma) (Atallah and Scanziani 2009) are suitable candidates to modulate the induction of synaptic plasticity in these synaptic paths (Colgin et al. 2009; Isomura et al. 2006). Indeed, changes in hippocampal oscillations do occur with learning (Bastiaansen et al. 2002; Jones and Wilson 2005; Montgomery and Buzsaki 2007). As induction of synaptic plasticity develops in various temporal fashions in multiple synapses, neurons could utilize compartmentalization mechanisms to integrate the information associated to these plastic events. Spatial and temporal interactions among plastic synapses of a neuron might enable the processing of information arriving from different brain areas into the neuron’s distinct functional compartments and associate or segregate such information (Alarcon et al. 2006; Barco et al. 2002; Govindarajan et al. 2006; Sajikumar and Korte 2011a; Sajikumar et al. 2007a). Conceivably, the relationship between changes in input activity, hippocampal oscillations, and compartmentalization could
shape a subset of the neuron population to specifically encode information related to a given behavioral experience (Diba and Buzsaki 2008; Fenton et al. 2008; Geisler et al. 2010; O’Neill et al. 2008). These neurons could be part of a particular population ensemble that could generate particular output spike activity stamps (Broome et al. 2006; Dragoi et al. 2003; Marder and Buonomano 2003) that will impact the decoding of information in order to produce behaviorally relevant outputs (Benchenane et al. 2010; Lansink et al. 2009; Sirota et al. 2008).

2.8 Experience-Dependent Compartmentalization

Experimental and theoretical work has characterized the properties of plasticity-induced dendritic compartments. However, the emergence of a functional role for compartmentalization upon physiologically relevant behaviors for information processing (e.g., learning) is still understudied. Encouragingly, recent reports have begun to tackle this deficit and suggest that experience induces plasticity in a dendritic compartment-specific fashion that could reflect information processing that takes place during learning, development, and sensory processing. Exposure to an enriched environment leads to compartmentalized changes in the distribution of dendritic spike propagation within particular dendritic branches of CA1 principal neurons (Makara et al. 2009), indicating that the electrical properties of individual dendritic branches can be modified by in vivo experience. Similarly, spontaneous activity that occurs during development is shown to functionally cluster CA3 synapses (based on glutamate receptor activity and insertion, and intracellular Ca^{2+} distribution) on developing dendrites (Kleindienst et al. 2011). Lastly, sensory experience was shown to produce synaptic potentiation of nearby (clustered) dendritic synapses (Makino and Malinow 2011). Interestingly, this clustered synaptic potentiation was eliminated when animals were deprived of sensory experience (Makino and Malinow 2011).

These outstanding studies strongly demonstrate that experience drives compartmentalized synaptic changes in neuronal dendritic domains; suggesting compartmentalization of plastic events may prove fundamental for the development and function of neuronal circuits.

2.9 Multiple Levels of Integration of Information

Multiple compartments with different functional sizes are thought to dramatically enhance the associative properties of a neuron that receives multiple streams of information in its different synapses. According to the functional compartmentalization model, the size of a compartment depends on an activity function/wave; there is no restriction in the size of a compartment, and therefore, in the extent of
plasticity-mediated associativity within it. A neuron’s functional compartmentalization could be confined to a small cluster of synapses within a dendritic branch, or to larger portions of the dendrite (including primary and secondary branches). In theory, compartmentalization could extend throughout the entire dendritic tree and even across the whole neuron. What would be the distinction of processing information within compartments, across compartments and at the cell-wide levels? And, what are the functional and behavioral consequences of each form of information processing?

Compartment-specific associativity can be overridden (Alarcon et al. 2006). As described above, compartment-specific synaptic capture of LTP is observed within basilar or apical CA1 dendrites, but not from one dendrite to another. However, when a stronger synaptic stimulation (different from the one normally used for synaptic capture of LTP experiments) was used to activate (tag) the capture of plasticity products at synapses in the opposite dendrite, LTP associativity across these formerly independent compartments was observed (Alarcon et al. 2006; Sajikumar et al. 2007a). Compartment-specific and across-compartments plastic associativity may depend on the cumulative recruitment of synaptically driven molecular mechanisms; therefore, they are a function of the strength of synaptic activation (Alarcon et al. 2006; Fonseca et al. 2004; Sajikumar et al. 2007a; Young and Nguyen 2005). Different activity thresholds that regulate the magnitude of compartmentalization could, in theory, allow for the expression of separate, independent, functional compartments within a single neuron or facilitate the functional overlapping between two or more compartments (Schacher et al. 1997; White et al. 1990).

Associative processes that encompass the whole cell would require stronger synaptic activation to initiate a larger recruitment of plasticity mechanisms– not at the branch or the dendrite level but at the cellular level. Somatic depolarization that strongly increases neuron’s excitability is known to promote associative phenomena at the cell-wide level in CA1 pyramidal neurons. After strong somatic depolarization, synaptic capture of LTP is observed at any set of synapses in either basal or apical dendrites (Alarcon et al. 2006; Dudek and Fields 2002). Conceivably, somatic depolarization that generates spike back-propagation into both basal and apical dendrites (Kloosterman et al. 2001) could mitigate the functional boundaries of previously established compartments. This biophysical process could trigger cellular mechanisms that may facilitate the unbiased sorting and distribution of gene products within the cell. Likewise, enhanced CREB-mediated gene expression initiated in the absence of synaptic activity increases overall cell excitability, and that too promotes cell-wide synaptic capture of LTP (Alarcon et al. 2006; Barco et al. 2002; Casadio et al. 1999; Dong et al. 2006; Lopez de Armentia et al. 2007; Marie et al. 2005). Increased cell excitability and gene expression resulting from substantial neuromodulatory activity, as in aroused, highly attentional or alarmed behavioral states, could expedite cell-wide associative processes (Berke and Hyman 2000; Reymann and Frey 2007). Moreover, environmental enrichment is shown to increase intrinsic excitability (Irvine et al. 2006), which could facilitate cell-wide facilitation of associative processes (Adams and Dudek 2005; Lee et al. 2005).
Vis-à-vis plasticity-mediated associative processes, the computing capability of a neuron could involve compartment-specific, inter-compartmental, and cell-wide integration. These levels of integration could represent separate, independent mechanisms for encoding information in different neurons. That is to say, some neurons (upon particular synaptic activation) could undergo one type of integration, while others (upon different regimes of synaptic activation) could undergo a different type of integration. Alternatively, these different levels of integration could be dynamic transitional stages; adding more richness to the neuron’s temporal and spatial integrative capability. For instance, compartment-specific changes in synaptic function elicited by sensory stimulation progress into a cell-wide change in synaptic function after sensory deprivation (Makino and Malinow 2011). Moreover, whereas cognitive behaviors may induce selective compartment-specific changes of synaptic function, stressful experiences would cause extreme neuromodulatory function to break down compartmentalization and promote cell-wide changes (Sajikumar et al. 2007b). Cell-wide and compartmental processes may be part of overlapping learning mechanisms which can be essential for behavior (Hulme et al. 2013). Transitions between compartment-specific, inter-compartmental, and cell-wide integration stages may also serve for the temporally correct processing of information. Acquisition of information may require the use of one integrative mechanism, whereas consolidation of information may require another. Hence, the cellular mechanisms that subserve the processing of information at each level as well as the transit from one level to another become most relevant for the proper association and segregation of information. Failure of these mechanisms may underlie the manifestation of disorders that have improper information processing as their core feature.

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