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# Spike-timing-dependent plasticity enhanced synchronization transitions induced by autapses in adaptive Newman-Watts neuronal networks

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ABSTRACT

In this paper, we numerically study the effect of spike-timing-dependent plasticity (STDP) on synchronization transitions induced by autaptic activity in adaptive Newman–Watts Hodgkin–Huxley neuron networks. It is found that synchronization transitions induced by autaptic delay vary with the adjusting rate $A_p$ of STDP and become strongest at a certain $A_p$ value, and the $A_p$ value increases when network randomness or network size increases. It is also found that the synchronization transitions induced by autaptic delay become strongest at a certain network randomness and network size, and the values increase and related synchronization transitions are enhanced when $A_p$ increases. These results show that there is optimal STDP that can enhance the synchronization transitions induced by autaptic delay in the adaptive neuronal networks. These findings provide a new insight into the roles of STDP and autapses for the information transmission in neural systems.



## 1. Introduction

Synchronization is an important phenomenon occurring in many realistic systems of biology, ecology, and so on (Arenas et al., 2008; Suykens and Osipov, 2008). In neural systems, synchronization is correlated with many physiological mechanisms of normal and pathological brain functions (Gray and Singer, 1989; Bazhenov et al., 2001; Mehta et al., 2001) and particularly with several neurological diseases such as epilepsy and tremor in Parkinson's disease (Levy et al., 2000; Mormann et al., 2003). In neural systems, information transmission between neurons occurs at electrical and chemical synapses, and information transmission delays are inherent due to the finite propagation speeds and time delays occurring by both dendritic and synaptic processing. Physiological experiments have revealed that the transmission delays introduced by chemical and electrical synapses are several tenths of milliseconds and 0.05 ms in length, respectively (Mann, 1981; Izhikevich, 2006). Several decades ago, Van der Loos and Glaser found a unique synapse, known as autapse. They pointed out that autapse occurs between dendrites and axon of the same neuron and connects a neuron to itself, and these self-connections could establish a time-delayed feedback mechanism at the cellular level (Van der Loos and Glaser, 1972.). In addition, there are many studies reporting the possible existence of autapses in different brain regions (Tamás et al., 1997; Lübke et al., 1996; Bacci et al., 2003). In recent decade, the roles of autapses in the firing dynamics of neurons have been extensively studied. It is found that autaptic activity can enhance the precision of spike times of neurons (Bacci and Huguenard, 2006), engineer the synchronization of action potentials in cultured neurons (Rusin et al., 2011), induce rich firing patterns in a Hindmarsh–Rose model neuron (Wang et al., 2014), enhance pacemaker-induced stochastic resonance in a scale-free neuronal network (Yilmaz et al., 2016a) and propagation of weak rhythmic activity across small-world neuronal networks (Yilmaz et al., 2016b), and regulate the firing of interneurons (Guo et al., 2016a, 2016b). Recently, a phenomenon of synchronization transitions has attracted increasing attention, and synchronization transitions induced by time delay (Wang et al., 2008, 2009a,b, 2011; Gong et al., 2011; Guo et al., 2012; Qian et al., 2013; Wu et al., 2013), coupling strength (Xu et al., 2013; Sun et al., 2011), noise (Wu et al., 2014, 2015a; Wang et al., 2015a), and even autapses (Wu et al., 2015b; Wang et al., 2015b) have been observed in various neuronal networks. However, these studies are devoted to a static description of synaptic connections. In reality, neural networks are adaptive due to synaptic plasticity, and synaptic strength varies as a function of neuromodulation and time-dependent pro-

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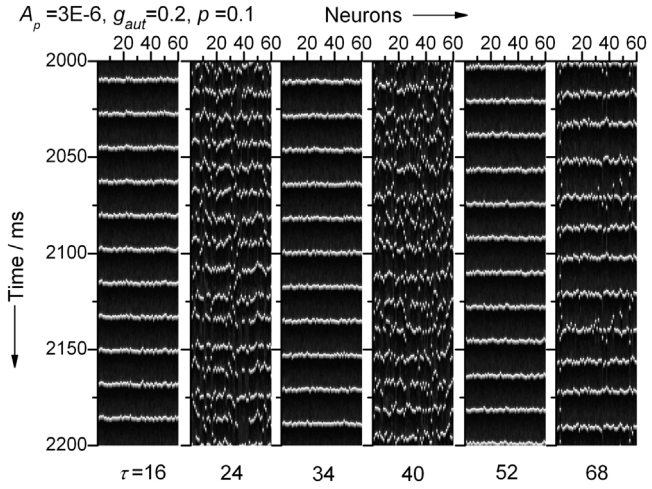


**Fig. 1.** Spatiotemporal patterns of the membrane potentials for different autaptic delays $\tau$. Other parameters are:$A_p = 3 \times 10^{-6}$,$g_{aut} = 0.2$, and $p = 0.1$. As $\tau$ increases, the neurons intermittently become synchronous and non-synchronous, exhibiting synchronization transitions.

cesses (Markram et al., 1997; Bi and Poo, 1998; Feldman and Brecht, 2005) One representative of this biological effect is spike-timing-dependent plasticity (STDP), which modulates coupling strength adaptively based on the relative timing between pre- and post-synaptic action potentials (Markram et al., 1997; Bi and Poo, 1998). A series of biological works have confirmed the existence of STDP in excitatory synapses onto neocortical (Feldman and Brecht, 2005) and hippocampal pyramidal neurons (Bi and Poo, 1998), excitatory neurons in auditory brainstem (Tzounopoulos et al., 2007), parvalbumin-expressing fast- spiking striatal interneurons (Fino et al., 2009), etc. It is suggested that synaptic plasticity may account for learning and memory (Abbot and Nelson, 2007; Kim and Linden, 2007). In the past years, the roles of STDP in the synchronization of neuronal population have been intensively investigated (Ruan and Zhao, 2009; Nowotny et al., 2003; Zhigulin et al., 2003; Karbowski and Ermentrout, 2012; Perez and Uchida, 2011; Kube et al., 2008; Mikkelsen et al., 2013; Yu et al., 2015). It was found that STDP modifies the weights of synaptic connections in such a way that synchronization of neuronal activity is considerably weakened (Kube et al., 2008); STDP induces persistent irregular oscillations between strongly and weakly synchronized states (Mikkelsen et al., 2013); STDP can largely depress the temporal coherence and spatial synchrony induced by external noise and random shortcuts in Newman–Watts neuronal networks (Yu et al., 2015). So far, however, there are few studies on synchronization transitions in adaptive neuronal networks and, particularly, there is no study on synchronization transitions induced by autapses in adaptive neuronal networks with STDP.

In this paper, we study how autapses induce synchronization transitions in adaptive Newman–Watts neuronal networks with STDP, focusing on how STDP influences the synchronization transitions. We first present synchronization transitions induced by autaptic delay when the adjusting rate of STDP is fixed, and then study the effect of STDP by investigating how the synchronization transitions vary when the adjusting rate of STDP is varied. We also study how the effect of network randomness and network size on synchronization transitions varies when the adjusting rate of STDP is varied. Finally, mechanism is briefly discussed and conclusion is given.

## 2. Model and equations

Here Hodgkin–Huxley (HH) neuron model (Hodgkin and Huxley, 1952) and Newman–Watts (NW) networks are used. According to Newman–Watts (NW) topology (Newman and Watts, 1999), the present network comprising of $N$ identical Hodgkin–Huxley neurons starts with a regular ring, each neuron having two nearest neighbors, and then links are randomly added with probability $p$ (network randomness) between non-nearest vertices. When all neurons are coupled with each other, the network contains $N(N-1)/2$ edges. The number of added random shortcuts is$M = pN(N-1)/2$. If $p = 0$, the network is a regular ring; for $p = 1$, the network is a globally coupled random network; for $0 < p < 1$, a Newman–Watts small-world network occurs. Note that there are a lot of network realizations for a given $p$.

In the presence of autapses, the dynamics of adaptive NW HH neuronal networks can be written as:

$$C\frac{dV_i(t)}{dt} = -g_{Na}m_i^3h_i(V_i - V_{Na}) - g_K n_i^4(V_i - V_K) - G_L(V_i - V_L) + I_{aut_i} + I_i^{syn} + \xi_i(t) \quad (1)$$

where $C = 1\mu Fcm^{-2}$is the membrane capacitance; $g_K = 36$,$g_{Na} = 120$, and $G_L = 0.3$mS cm$^{-2}$ are the maximal conduc-

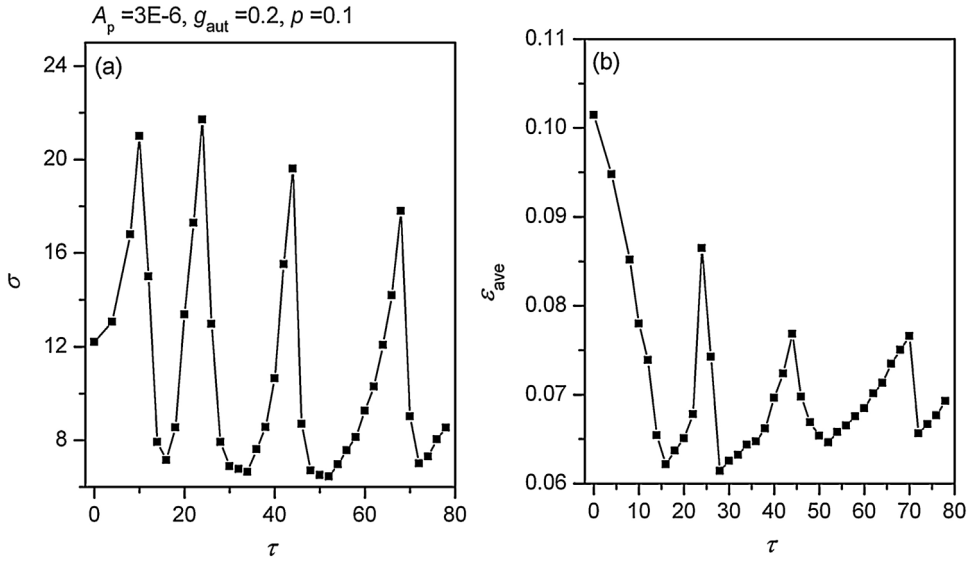


**Fig. 2.** Dependence of $\sigma$(a) and $\varepsilon_{ave}$(b) on $\tau$ at $A_p = 3 \times 10^{-6}$,$g_{aut} = 0.2$, and $p = 0.1$. As $\tau$ increases, $\sigma$ and $\varepsilon_{ave}$ pass through a few peaks, which quantifies the presence of synchronization transitions induced by autaptic delay.