Propagation of Action Potential Mediated by Microtubules May Involve in The Neural Quantum Mechanism

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ABSTRACT

The traditional thoughts hold that action potential is a basis of neural information transmission. Previous studies have found that microtubules are structurally connected to some ion channels such as the subunits of sodium, potassium and calcium channels on axons, suggesting that microtubules may be related to the propagation of action potential. Moreover, recent studies have demonstrated that microtubule system network in the brain may be involved in the mechanism of photon quantum brain and the origin of consciousness. These studies indicate that the structural integrity of microtubules is closely related to the action potential. However, the detailed relationship between microtubules and action potential is not clear. Here, we found that the compound action potentials of bullfrog sciatic nerve were inhibited significantly by colchicine, a microtubule depolymerizer. The inhibitory effects presented time-dependent changes and even reached to a decrease of 57% after treatment for 480 min with 20 mM Colchicine. These results suggest that the propagation and transmission of action potentials are related to the stability of microtubule system and may involve in the neural quantum mechanism.

Key Words: action potential, microtubule, sodium channels, neural quantum mechanism.

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Introduction

Traditional theories have believed that neural communication is mediated by action potential and chemical molecules via the processes called bioelectrical and chemical transmission, respectively. In the early 1950s, Hodgkin, Huxley and Katz conducted electrophysiological experiments on squid giant axons and found that stimulating an axon with direct current resulted in a large instantaneous inflow of sodium ions (Na^+), which was followed by outflow of potassium ions (K^+) (Hodgkin and Huxley, 1952). Further studies showed that the increased permeability of cytomembrane to sodium ions, which make the membrane potentials tend to the equilibrium potential of sodium ions, provides an explanation for the formation of action potential. Then based on their experimental measurements, Hodgkin and Huxley were able to construct a mathematical model of Na^+ and K^+ conductance changes, which was later known as Hodgkin-Huxley model (HH model) and laid a theoretical foundation for elucidating the generation and transmission mechanism of action potential (Hodgkin and Huxley, 1952).

However, in 2014, a research team at the University of Copenhagen in Denmark found that the collision of two action potentials propagating in

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the same direction did not result in the annihilation of the action potentials predicted by the HH model, but presented a phenomenon of mutual penetration (Heimburg and Jackson, 2014). Other studies have observed that there is no heat dissipation in the process of action potential transmission, which is an adiabatic (non-dissipative) phenomenon (Abbott *et al.,* 1958; Howarth *et al.,* 1968; Howarth, 1975; Ritchie and Keynes, 1985; Tasaki and Byrne, 1992). However, according to HH model, ion flow through resistance (channel protein) can lead to irreversible heat dissipation of membrane (Heimburg and Jackson, 2007), which is in contradiction with the observed results. In addition, it was found that the process of action potential transmission was accompanied by the changes in physical quantities such as the thickness, area and length of nerve cell membrane (Appali *et al.,* 2012). Therefore, it is clear that the HH model based on the opening and closing of ion channels could not explain the observed experimental phenomena mentioned above.

Microtubules, which are widely existed in various cells, are the most fundamental structure of axons. Microtubules play a key role in maintaining structural stability of axons. Previous studies have found that microtubules are structurally connected to some ion channels such as the subunits of sodium, potassium and calcium channels on axons (Fukuda et al., 1981; Matsumoto et al., 1983; Matsumoto et al., 1984; Park et al., 2004) suggesting that microtubules may be related to the propagation of action potential (Matsumoto and Sakai, 1979). These studies indicate that the structural integrity of microtubules is closely related to the action potential. In addition, the changes in microtubule structure are associated with the pathogenesis of some neurological diseases. For example, hyperphosphorylation of microtubule-associated protein tau plays an important role in the pathogenesis of Alzheimer disease (Iqbal *et al.,* 2003). Some anti-tumor drugs such as colchicine that interferes with the function of microtubule can cause obvious neurological side effects (De Deyn *et al.,* 1995). Moreover, recent studies have showed that microtubule system in the brain may be involved in the mechanism of quantum brain and the origin of consciousness (Hameroff and Penrose, 2014; Chai *et al.,* 2018). These studies suggest that microtubule system may play an important role in the transmission of neural signals. Therefore, in this study, by studying the relationship between the functional changes of microtubules and the propagation of action potentials of nerve fibers, we hope establish the relationship between microtubules, action potentials and biophotons, which may provide new ideas for further understanding the mechanism of quantum brain.

Materials and Methods

Preparation of bullfrog sciatic nerve specimens

Adult bullfrogs (250-350 g) were purchased from a commercial supplier and kept in a tank under the room temperature (22~25 °C). Bullfrog sciatic nerve specimens were prepared according to the reported method (Kobayashi *et al.,* 1996) and placed in Ringer's solution. Ringer's solution contained (in mM): 111 NaCl, 1.9 KCl, 1.1 $CaCl_2$, 2.4 $NaHCO_3$, 0.1 NaH_2PO_4, and 11 D-glucose, pH 7.8, 280 mOsm/L.

Recording of the compound action potential of bullfrog sciatic nerve after colchicine treatment

The schematic diagram of the experimental device is shown in Fig.1. A self-made nerve specimen box and BL-420F biological signal acquisition and analysis system were used to record the compound action potential of bullfrog sciatic nerve. The nerve specimen box includes five silver wire electrodes, and two pairs of which are used as stimulating electrodes and recording electrodes, respectively, and the rest one of electrodes is grounded. The distance between the recording electrodes is approximately 10 mm, which is longer than that of the stimulating electrodes (about 5 mm). The ground wire is placed between the stimulating electrodes and the recording electrodes, but it is closer to the stimulating electrodes in order to filter stimulus artifact more effectively. BL-420F biological signal acquisition and analysis system contains a photoelectric isolated stimulator with editable waveform, and the four recording channels are independent of each other.

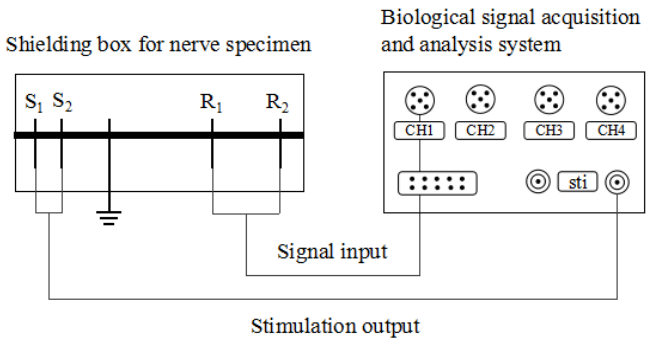

Figure 1. The schematic diagram of the experimental device. It is comprised of a nerve specimen shielding box and biological signal acquisition and analysis system. S1 and S2 are used as stimulating electrodes and R1 and R2 as recording electrodes. The middle wire is grounded to filter stimulus artifact.

The prepared bullfrog sciatic nerve was incubated in Ringer's solution for 30 min and divided randomly into control group and experimental group. The sciatic nerve in the experimental group was dipped in Ringer's solution containing 20 mM colchicine (Sigma-Aldrich), while the sciatic nerve in the control group was only placed in Ringer's solution. Then the bullfrog sciatic nerve was placed in the nerve specimen box every 60 minutes to record the compound action potential induced by direct-current electrical stimulations. The stimulation mode is set to single stimulus with the intensities of 0.1 V, 0.5 V, 1 V and 2 V, respectively, and the pulse width of each stimulus is 0.1 ms.

Data analysis

The positive phase (the first phase) amplitude peak of action potential (AP) was recorded by the software of BL-420F biological signal acquisition and analysis system. The relative amplitude peak of action potential [RV(AP) (%)] was calculated as:

$$RV(AP)\ (\%) = [AP\ (after)/AP\ (before)] \times 100\%.$$

Where AP (before) represents the amplitude peak of action potential before colchicine treatment and AP (after) shows the amplitude peak of action potential after colchicine treatment.

Statistical analysis

A two-way mixed ANOVA was employed to determine the effects of 20 mM Colchicine and time points on the amplitude peak of action potential (AP) and the relative amplitude peak of action potential [RV(AP) (%)] using the GraphPad Prism 7.0 software, and the main and interaction effects were reported as applicable. For the post-hoc analysis, paired two-tailed t-test was used to compare the effects at different time points in the treated or control group and two-tailed Student's t-test was used to compare the effects between the control and treated group at different time points using Microsoft Excel.

Results

Determination of stimulus intensities

The compound action potential was induced at different stimulus intensities (0.1 V, 0.5 V, 1 V and 2 V). The results showed that the stimulus intensity of 1 V could induce the maximum amplitude of the compound action potential, while applying greater stimulus intensity up to 2 V could not increase the amplitude of the compound action potential, suggesting that the stimulus intensity of 1 V has reached the maximum effect (Fig. 2). Therefore, 1 V was selected as the effective stimulus intensity for inducing the compound action potential.

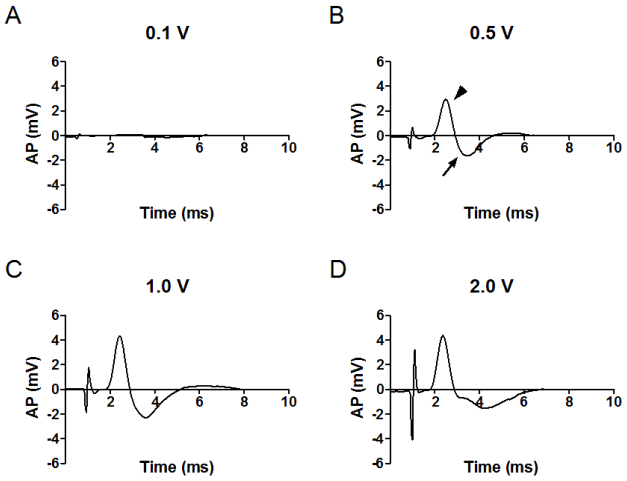

Figure 2. A representative bullfrog sciatic nerve showing the compound action potentials (AP) induced by a direct current (DC) single-phase square wave (0.1 ms pulse width) at different stimulation intensives (0.1 V in **A,** 0.5 V in **B,** 1 V in **C** and 2 V in **D**). The arrowhead and arrow indicate the positive phase (the first phase) and the negative phase (the second phase) amplitude peak of action potential (AP), respectively.

Time-dependent changes in the compound action potential of bullfrog sciatic nerve after colchicine treatment

Previous studies have demonstrated that 20 mM colchicine had significant inhibitory effect on sodium current (Matsumoto *et al.,* 1984) and our preliminary experiment also found that there were no significant differences in the concentrations over 20 mM. Therefore, we carried out a two-factor mixed-design ANOVA experiment and treated the bullfrog sciatic nerve continuously with 20 mM Colchicine to observe the time-dependent changes in the compound action potential of bullfrog sciatic nerve as compared to control.

The interaction effects treated with and without 20 mM colchicine on AP [$F(8, 64) = 19.35$, $p < 0.001$] and RV(AP)(%) [$F(8, 64) = 19.09$, $p < 0.001$] were found to be significant. A significant time-dependent influence of 20 mM colchicine on AP [$F(8, 64) = 7.15$, $p < 0.001$] and RV(AP)(%) [$F(8, 64) = 6.42$, $p < 0.001$] was found. The amplitude of action potential remained almost unchanged after treatment for 120 min with 20 mM Colchicine although a slight increase was observed after 60 min, but then presented a gradual downward trend after 120 min. (Fig. 3, Fig. 5, Table 1 and Table 2). The RV(AP)(%) reached to 93%, 85%, 77%, 69%, 64%, 61% and 57%, respectively,

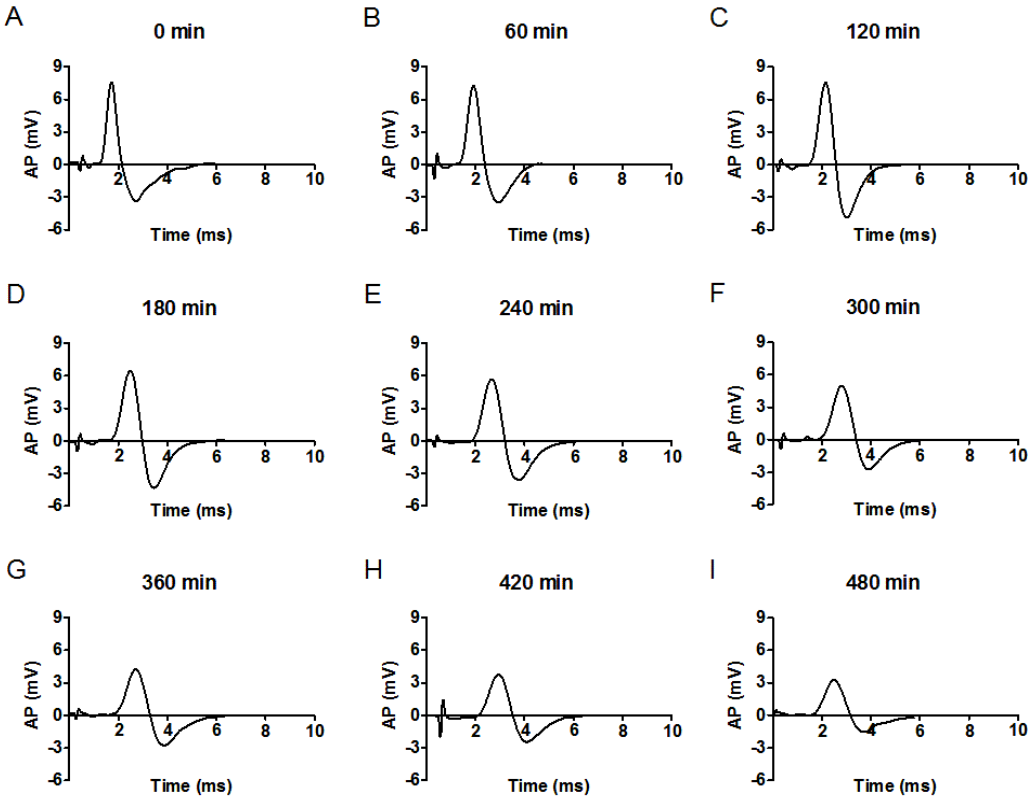

Figure 3. A representative bullfrog sciatic nerve showing the time-dependent changes in the compound action potentials after incubation in Ringer's solution treated with 20 mM Colchicine. **A-I** presents the recorded action potentials at 0, 60, 120, 180, 240, 300, 360, 420 and 480 min, respectively.

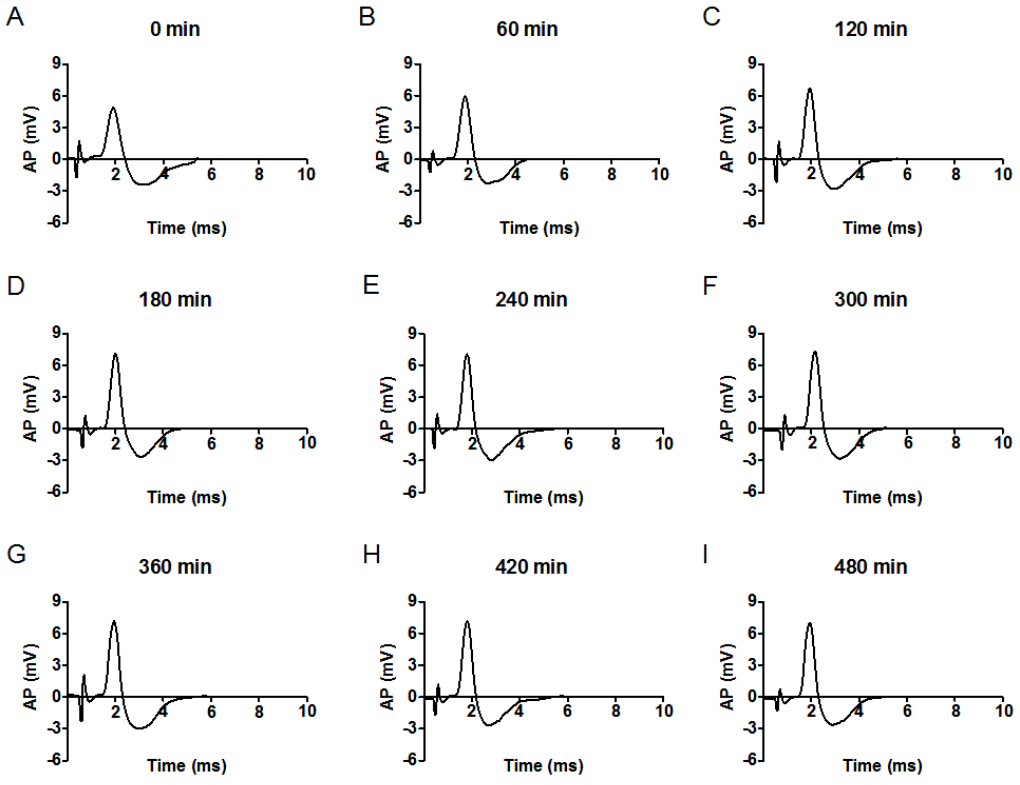

Figure 4. A representative bullfrog sciatic nerve showing the time-dependent changes in the compound action potentials after incubation in Ringer's solution. **A-I** presents the recorded action potentials at 0, 60, 120, 180, 240, 300, 360, 420 and 480 min, respectively.

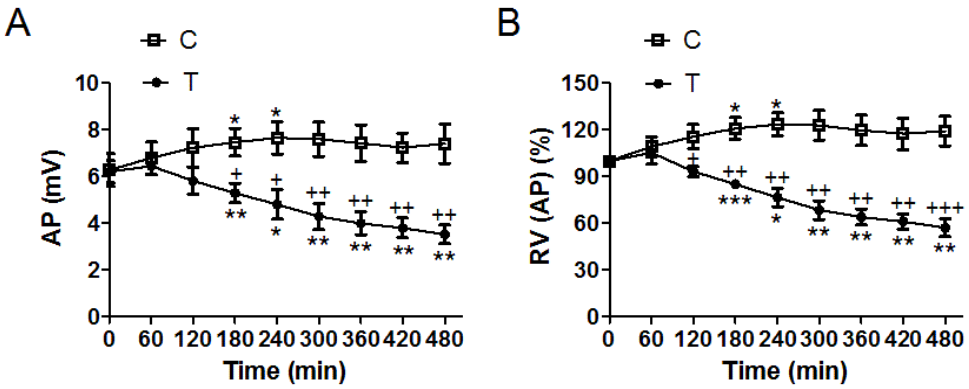

Figure 5. (**A, B**) The time-dependent changes in AP (**A**) and RV(AP)(%) (**B**) of bullfrog sciatic nerve after incubation in Ringer's solution treated with and without 20 mM Colchicine. AP: the compound action potential, RV(AP)(%): the relative amplitude peak of action potential. Data are shown as the mean±s. e. m. n=the number of bullfrog sciatic nerve. Statistical analysis was carried out to compare the time-dependent changes (*) and the differences between the treated group and the control group (+). Significant differences (* or + p<0.05, ** or ++ p<0.01, *** or +++ p<0.001) are noted from 180-480 min (see also Table 1 and Table 2).

Table 1. The time-dependent changes in the positive amplitude of compound action potential of bullfrog sciatic nerve in treated and control group.

Time	**Treated group** (n=5) (*p*=*)	**Control group** (n=5) (*p*=*)	*P*=+
0 min	6.21±0.48	6.29±0.71	0.9312
60 min	6.46±0.38 (0.5463)	6.79±0.69 (0.2299)	0.6797
120 min	5.82±0.58 (0.1107)	7.24±0.82 (0.0698)	0.1968
180 min	5.29±0.41 (0.0017)	7.47±0.58 (0.0199)	0.0159
240 min	4.81±0.63 (0.0129)	7.66±0.69 (0.0179)	0.0160
300 min	4.29±0.55 (0.0052)	7.60±0.73 (0.0553)	0.0067
360 min	4.01±0.49 (0.0028)	7.43±0.78 (0.0848)	0.0060
420 min	3.80±0.42 (0.0033)	7.24±0.63 (0.1509)	0.0019
480 min	3.53±0.40 (0.0044)	7.41±0.84 (0.1015)	0.0031

Data are shown as the mean±s. e. m. n=the number of bullfrog sciatic nerve. Statistical analysis was carried out to compare the time-dependent changes (*) and the differences between the treated group and the control group (+).

Table 2. The time-dependent changes in the relative positive amplitude of compound action potential of bullfrog sciatic nerve in treated and control group.

Time	**Treated group** (n=5) (*p*=*)	**Control group** (n=5) (*p*=*)	*p*=+
0 min	100%±0.00	100%±0.00	
60 min	105.42%±7.45% (0.5070)	109.15%±6.29% (0.2196)	0.7122
120 min	93.27%±3.22% (0.1048)	115.66%±7.77% (0.1140)	0.0287
180 min	85.22%±1.54% (0.0007)	120.98%±7.02% (0.0403)	0.0011
240 min	76.63%±5.84% (0.0161)	123.52%±7.44% (0.0341)	0.0011
300 min	68.58%±5.96% (0.0062)	122.97%±9.51% (0.0731)	0.0013
360 min	64.12%±5.08% (0.0021)	119.67%±9.72% (0.1130)	0.0010
420 min	61.23%±4.87% (0.0013)	117.68%±10.15% (0.1566)	0.0010
480 min	57.15%±5.65% (0.0016)	119.24%±9.42% (0.1106)	0.0005

Data are shown as the mean±s. e. m. n=the number of bullfrog sciatic nerve. Statistical analysis was carried out to compare the time-dependent changes (*) and the differences between the treated group and the control group (+).

at 120, 180, 240, 300, 360, 420 and 480 min after treatment with 20 mM Colchicine (n=5). In contrast, in the control group, the effects were only slightly increased but not decreased during the first 120 min, and remained almost unchanged after 120 min as shown by the post-hoc analysis (Fig. 4, Fig. 5, Table 1 and Table 2). These findings suggest that 20 mM Colchicine had significant time-dependent inhibitory effects on the compound action potential.

Discussion

Previous studies have found that 10 mM colchicine can reduce the amplitude of action potential of squid giant axons and increase the threshold to evoke action potential significantly through intra-axonal recording (Matsumoto and Sakai, 1979). Voltage clamp experiments on squid giant axons showed that sodium current were almost suppressed after 20 mM colchicine treatment (Matsumoto *et al.,* 1984).

In the present study, by using bullfrog sciatic nerve specimens, we also found that the compound action potentials were significantly inhibited by 20 mM colchicine, which not only further confirmed the previous findings, but also implied a new mechanism related to Neuroquantology.

Colchicine is a kind of typical microtubule depolymerizer and some studies suggest that sodium channels and microtubules may be structurally connected (Iliev and Ivanov, 2009), however, there is no experimental evidence that colchicine acts directly on sodium channels. Therefore, we speculate that the decrease in the amplitude of action potentials caused by colchicine treatment has a direct relationship with the stability of microtubule structure. One possible explanation is that the structural and functional integrity of microtubules is closely related to the plasticity of sodium channels and the depolarization of membrane potential by a direct electrical stimulation results in a direct activity of microtubule, which then activates the sodium channels indirectly. Therefore, our findings suggest that microtubule may play a key role in the generation and transmission of action potentials, which may provide a novel explanation for the origin of electroencephalogram (EEG) (Hameroff and Penrose, 2014).

EEG is originated from the neural autonomous discharge, which is a basic phenomenon of brain functions. It has been noticed for a long time that neural cells show obvious electrical activities even without external stimulation, but the underlying mechanism has not been fully understood. A previous proposal raised by Penrose and Hameroff suggests that microtubule quantum vibrations may be a possible source of electroencephalogram of specific frequency (Gamma wave of 40 HZ) (Hameroff and Penrose, 2014). They further put forward that the formation of the consciousness may be involved in the quantum coherence and entanglement between microtubules (Hameroff and Penrose, 2003; Hameroff and Penrose, 2014; Hameroff et al., 2014). Tuszynski et al believed that anesthetics could cause the disappearance of consciousness by acting on quantum channels in the microtubules of the brain (Craddock *et al.,* 2015), in which biophotons may be involved.

It has been demonstrated in our previous studies that biophotons could transmit along the nerve fibers (Sun *et al.,* 2010), and the glutamate induced biophotonic activities and transmission in mouse neural circuits could be significantly inhibited by hyperphosphorylation of microtubule associated protein tau (Tang and Dai, 2014). Our recent study has also revealed that the biophotonic activity and transmission caused by the synergistic effects of different neurotransmitters may play an important role in the origin and the altered states of consciousness (Chai *et al.,* 2018). In addition, the structural characteristics of neurites including axons and dendrites, microtubules and mitochondria in axons have been proved theoretically to have the physical basis of biophotonic transmission (Jibu *et al.,* 1994; Craddock et al.,2009; Rahnama *et al.,* 2011; Kumar et al., 2016; Zangari *et al.,* 2018). The characteristics and behaviors of biophotons such as entanglement, coherence and superposition may satisfy the condition of quantum communication of neural network (Gibney, 2016; Tarlacä and Pregnolato, 2016). Our findings in this study that colchicine inhibits the generation and conduction of action potential along nerve fibers may bring new links among the structural and functional integrity of microtubules, biophotonic activity and transmission as well as the generation and conduction of action potential, which may be of great significance for us to understand the mechanism of 'photon quantum brain' (Chai *et al.,* 2018)

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