Electrical membrane properties of rat subthalamic neurons in an in vitro slice preparation

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The electrical membrane properties of subthalamic (STH) neurons and their response characteristics to stimulation of the internal capsule (IC) were studied in an in vitro slice preparation. Most STH neurons recorded exhibited spontaneous repetitive firing. The input resistance of STH neurons was 146 ± 48 MΩ and showed both an anomalous and a delayed rectification when the membrane was hyperpolarized or depolarized by current injections. In neurons with the membrane potential less negative than 65 mV, depolarizing current pulses generated repetitive firing with the maximum frequency of up to 500 Hz. Two types of tetrodotoxin (TTX)-resistant cobalt-sensitive potentials, slow depolarizing potential and slow action potential, were observed in STH neurons. The slow depolarizing potential had a long duration (over 500 ms in some cases) and was able to trigger repetitive firing. The slow action potential had a duration of about 30 ms and triggered a burst of firing. The slow action potential was seen only when the neurons were hyperpolarized to more negative than 65 mV by a current injection. Electrical stimulation of IC evoked monosynaptic inhibitory postsynaptic potentials (IPSPs) in most of the neurons examined. The polarity of IPSPs was reversed in the depolarizing direction by intracellular injection of CI−. Bath application of bicuculline markedly suppressed IPSPs and unmasked monosynaptic excitatory postsynaptic potentials (EPSPs). The EPSP was able to trigger a slow depolarization with repetitive firing or a slow action potential with burst of firing when the neuron was hyperpolarized by a continuous current injection. The results demonstrated that STH neurons in an in vitro preparation have spontaneous discharges, high input resistance, capability to generate high-frequency firing, and Ca potentials. The pattern of responses of STH neurons to synaptic inputs is dependent on their membrane potentials.

INTRODUCTION

The subthalamic nucleus (STH) is a small lens-shaped nucleus which lies between the zona incerta dorsally and the cerebral peduncle ventrally. Morphological studies using Golgi, Nissl and intracellular labeling techniques indicated that the somatic shape of rat STH neurons varies from fusiform to oval or polygonal, and 2–5 primary dendrites arise from the soma. However, the distributions of the soma size and the number of primary dendrites are unimodal. Both anatomical and electrophysiological studies indicated that rat STH neurons have bifurcating axons which project to the globus pallidus (GP) and to the substantia nigra (SN). Their axon terminals form asymmetrical synapses mainly with the dendrites of the target neurons. Functional significance of STH outputs has been questioned for some time, but our recent data indicated that electrical stimulation of STH produces monosynaptic excitation to the SN neurons. STH neurons receive major afferents from GP and the cerebral cortex. In addition, STH neurons receive projections from the pedunculopontine tegmental nucleus, the dorsal raphe nucleus and the centre median parafascicular complex. Electrophysiological studies demonstrated that inputs from GP are inhibitory while those from the cerebral cortex...
and the pedunculopontine tegmental nucleus are excitatory. A review of the literature reveals that there is only a cursory report on the electrophysiological properties of STH neurons. Therefore, we have studied the electrophysiological characteristics of STH neurons in detail using an in vitro slice preparation. The data will not only add to the bank of data on the electrical membrane properties of CNS neurons but also may aid in understanding how STH neurons process their afferent inputs.

MATERIALS AND METHODS

Male Sprague-Dawley rats weighing 200–350 g were decapitated and the brains were rapidly removed. The brain was trimmed with a razor blade to a block containing the STH. Parasagittal slices of 400 μm thick were cut from the block with a Vibratome and were preincubated in oxygenated Krebs solution for about 1 h at 35 °C before recording. The recording chamber was constructed to allow Krebs solution (35 °C) to continuously flow on the bottom surface of the slice at a rate of 0.5–0.7 ml/min and to allow a warm and moist gas mixture (95% O2–5% CO2) to flow over the top surface of the slice. The Krebs solution was composed of (mM): NaCl 124, KCl 5.0, KH2PO4 1.24, NaHCO3 26, CaCl2 2.4, MgSO4 1.3 and glucose 10. Glass pipettes filled with 2 M potassium methylsulfate or 1.5 M potassium chloride with a DC resistance between 60 and 100 MΩ were used for the recording. Intracellular recordings were obtained through a high-input impedance biological amplifier with an active bridge circuit which enabled measurement of the membrane potential and the injection of intracellular constant current simultaneously. The output of the amplifier was fed into an oscilloscope and a DC pen recorder. Electrical stimulation was applied through a bipolar electrode made by twisting a pair of 80-μm-diameter nichrome wires insulated except at the tips, which were separated by 200–400 μm. The stimulating electrode was placed on the surface of the internal capsule at 0.5–1.0 mm rostral to the STH or on the cerebral peduncle immediately ventral to the SN pars reticulata. Stimulation parameters were 0.05–0.2 mA in intensity and 200 μs in duration delivered at 0.5/s.

The drugs used were tetrodotoxin (TTX) with a concentration of 10^-5 g/ml, tetraethylammonium chloride (TEA) at 10 mM, and bicuculline methiodide at 50–100 μM. In some animals, the internal capsule, at the level of the entopeduncular nucleus, was transected by a Halazs knife at 6–10 days prior to the recording. This was to eliminate afferents to STH originating from the structures rostral to STH.

RESULTS

The results were obtained from 98 STH neurons which had membrane potentials of more than 40 mV and generated action potentials with an amplitude greater than 40 mV. Of the 98 neurons, 92 exhibited spontaneous firing at a rate of 5–40 spikes/s. Although the membrane potential of neurons having higher spontaneous activity tended to be less negative than that of neurons having lower spontaneous activity, all the other electrical properties of these neurons were similar.

Fig 1. Input resistance of STH neurons. A membrane responses to intracellularly injected hyper- and depolarizing currents of various intensities. B. membrane responses to hyper- and depolarizing currents during application of TTX (10^-5 g/ml) to eliminate spikes. Square waves at the bottom of oscillographic records in this and all subsequent figures indicate the intensities of injected depolarizing (upward square wave) and hyperpolarizing (downward square wave) currents. Calibrations in A also apply to B C current–voltage relation for a neuron recorded in the slice superfused with TTX-containing solution. Note membrane rectification in both hyper- and depolarizing directions.
The input resistance

The input resistance of STH neurons was measured from the current–voltage relationship obtained from the membrane potential shifts to depolarizing and hyperpolarizing current pulses with a duration of 100 ms (Fig. 1A). In the neurons with spontaneous firing, the measurement of input resistance was obtained during continuous applications of a small hyperpolarizing current (i.e., less than 0.1 nA) or TTX (Fig. 1B), which eliminated spontaneous spikes. The input resistance calculated from the slope of the current–voltage curves (Fig. 1C) crossing at zero current pulse was 146 ± 48 MΩ (mean ± S.D., n = 26). The input resistance was decreased upon application of large hyperpolarizing current (an anomalous rectification) (Fig. 1A,B). A reduction of the input resistance was also observed during membrane depolarization (a delayed rectification) (Fig. 1B,C) in the slice superfused with TTX-containing solution.

Direct activation by intracellular stimulation

Injections of depolarizing current pulses to STH neurons produced either repetitive or burst discharges. The duration of action potentials was about 1 ms. When neurons with a membrane potential of 40–65 mV were activated by the injection of current pulses, single or repetitive action potentials were generated either from passive depolarization (Fig. 2A) or active slow depolarization which often outlasted the duration of the current pulses (Fig. 3A–C). Action potentials generated from passive depolarization had the highest frequency of firing at the beginning of the current pulse (Fig. 2A). The relationship between the intensity of injection current and the frequency of firing (I–f curve) obtained from the first interspike interval following the onset of the current was almost linear up to 300 Hz of firing but deviated downwards from the linearity at higher frequencies. As can be seen from the graph, the STH neuron could fire at the maximum frequency of about 500 Hz (Fig. 2B). The I–f curve obtained from the last interspike interval was almost linear up to 200 Hz and reached its peak at over 300 Hz. The slope of the linear portion of the I–f curve for 1/t1 (Fig. 2B) is about 900 Hz/nA, indicating that STH neurons are extremely sensitive to small changes in their excitatory inputs. Repetitive firings terminated by the offset of current pulses were followed by long-lasting (250–600 ms) hyperpolarizing potentials with an amplitude of 5–12 mV (Fig. 2C). The long-lasting hyperpolarizing potential was not affected by the intracellular Cl− injection (not shown) but was diminished by superfusing Ca2+-free medium (Fig. 2D).

In some STH neurons with a membrane potential of 50–65 mV, depolarizing current pulses evoked slow depolarizing potentials with action potentials (Fig. 3A–C). Fig. 3A, B shows responses to 3 different intensities (Fig. 3A) and durations (Fig. 3B) of depolarizing current pulses applied to a continuously hyperpolarized neuron. Current pulses with stronger intensity or longer duration evoked all-or-none slow depolarizing potentials which clearly outlasted the duration of the applied current pulses. The slow de-
polarizing potential with the duration of more than 500 ms was observed in some neurons. As shown in Fig. 3C, the slow depolarization could trigger repetitive firings in which the frequency of firings increased along with the development of the slow depolarization. The slow depolarizing potential was TTX-resistant (figure not shown) but suppressed by superfusion of Ca^{2+}-free medium (Fig. 3D).

When STH neurons were hyperpolarized to a membrane potential more negative than 65 mV by a continuous current injection, depolarizing current pulses produced a burst of fast action potentials which were triggered from an all-or-none relatively large slow action potential with a duration of about 30 ms (Figs. 4A and B). Fig. 4A shows responses of a continuously hyperpolarized neuron to 4 different intensities of depolarizing current pulses with a constant duration. The lowest intensity of stimulation

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Fig. 3 Slow depolarizing potentials evoked by depolarizing current pulses in neurons continuously hyperpolarized by current injection. A and B: injection of depolarizing current pulses at 3 different intensities in A and 3 different durations in B. C: injection of depolarizing current pulse induced slow depolarizing potentials with repetitive spike firing. Note that depolarizing potentials in A–C outlast the duration of injected current pulses. D: recording from the same neurons as C during superfusion with Ca^{2+}-free medium. Note a great reduction in the amplitude and duration of slow depolarizing potentials after the offset of the current pulse.

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Fig. 4 Slow action potentials evoked by depolarizing current pulses in STH neurons. The neuron was continuously hyperpolarized by current injection in A–D. A and B: injection of depolarizing current pulses at various intensities in A and different durations in B. C: recordings during superfusion with a medium containing TEA (10 mM). Injection of depolarizing current pulse induced a slow action potential with spikes. Note an increase in the spike duration. D and E: recordings during superfusion with a medium containing TEA and TTX. Injection of depolarizing current pulses with various intensities during continuous hyperpolarization in D and during depolarization in E. F: addition of Co^{2+} to the superfusing medium abolished slow action potentials.
failed to evoke regenerative responses, but 3 other stimulations generated slow action potentials. The rising rate of slow action potentials was increased with the increase in stimulus intensity. Responses to two different durations of current pulses applied to the same neuron are shown in Fig. 4B. The slow depolarizing potential triggered by a short-duration pulse can be distinguished from the slow action potential triggered by a long-duration pulse (Fig. 4B). The slow action potential with a burst of firings was also triggered after the onset of hyperpolarizing pulses (Fig. 1A).

**Ionic basis of regenerative potentials**

Bath application of 10 mM TEA increased the input resistance (up to 80%) of the cell and the duration of the fast action potential due to a decrease in the falling rate (Fig. 4C). Application of TTX (10^{-5} g/ml) to the TEA-containing medium abolished fast action potentials. Under these conditions, injection of depolarizing current pulses to either continuously hyperpolarized (Fig. 4D) or depolarized (Fig. 4E) neurons triggered long-duration slow action potentials. Slow action potentials generated from the depolarized neurons were smaller in amplitude and slower in the rising and falling rates than those from the hyperpolarized neurons. Their duration often outlasted the duration of the current pulse (Fig. 4E). Both the slow depolarizing and the slow action potentials were completely abolished by an application of Co^{2+} (3 mM) to the superfusing medium (Fig. 4F). It was also often observed that application of Co^{2+} resulted in an increase in the input resistance (compare Fig. 4D,F) which is probably due to a blockade of the leak Ca conductance of the neurons.

**Responses to the internal capsule stimulation**

Stimulation of the internal capsule (IC) at 0.5–1.0 mm rostral to STH evoked hyperpolarizing potentials in most of the STH neurons examined. The stimulation also evoked a negative field potential when recordings were made from the area close to the cerebral peduncle which lies ventrolaterally to STH (e.g., Fig. 5B). The field potential was diminished in the slice preparation obtained from chronically IC-transected rats, indicating that the response was caused by an activation of the descending fibers passing through the IC. The amplitude of the hyperpolarizing potential evoked by IC stimulation was increased by injection of depolarizing current and decreased by hyperpolarizing current. The polarity of the hyperpolarizing potential could be reversed to

![Synaptic responses in STH neurons following stimulation of IC immediately rostral to STH.](image)

A. Stimulation of IC elicited large IPSPs. Injection of depolarizing current increases the amplitude of IPSPs (top trace) and hyperpolarizing current of 0.2 nA decreases the amplitude of IPSPs. Reversal of the IPSPs with 0.5 nA current injection (bottom trace). B. IPSPs evoked by various stimulus intensities (top traces). Reversal of the IPSPs with Cl^{-} injection (middle traces). Extracellular field potentials (bottom traces). C. Stimulation of IC elicited EPSPs with a spike potential. Application of 0.4 nA depolarizing current (top traces) and hyperpolarizing current (bottom traces).
Fig. 6 Effects of bicuculline on the synaptic responses induced by IC stimulation. A. Bicuculline suppressed IC-induced IPSPs and unmasked EPSPs. Depolarizing current injection produced a barrage of spikes (top trace). Hyperpolarizing current injection increased EPSP amplitudes and reduced its durations (bottom two traces). B. EPSPs evoked by various stimulus intensities. C. EPSPs evoked during injection of a continuous hyperpolarizing current triggered slow depolarizing potentials (top and middle traces).

the depolarizing direction by the injection of a strong hyperpolarizing current (Fig. 5A). The results indicated that hyperpolarizing potentials induced by IC-stimulation were inhibitory postsynaptic potentials (IPSPs). The IPSP was considered to be monosynaptically induced since the change of the latency was very small (no more than 0.3 ms) and graded upon increase in the stimulus intensity (Fig. 5B). The shortest latency observed in 19 neurons ranged between 1.2 and 2.8 ms (mean = 1.7). When KCl-filled electrodes were used, the polarity of the IPSPs reversed itself in the depolarizing direction within a few minutes after a penetration into the neuron (Fig. 5B). Bath application of bicuculline methiodide (50–100 μM) markedly or even completely suppressed the IPSPs (Fig. 6A). These results indicate that the IPSPs were GABAergic and Cl-mediated responses. It was observed during bicuculline application that IC stimulation evoked depolarizing potentials in STH neurons. The latency of depolarizing potentials (mean = 3.5 ms, n = 6) did not change more than 0.3 ms upon increase in stimulus intensities (Fig. 6B). The amplitude of the depolarizing responses was decreased by continuous injection of depolarizing current and increased by hyperpolarizing current (Fig. 6A). These results would indicate that the depolarizing responses include monosynaptic excitatory postsynaptic potentials (EPSPs). The duration of the depolarizing response could be largely altered with changes in the stimulus intensity (i.e., less than 20 ms to over 400 ms). Fig. 6C shows responses of a continuously hyperpolarizing neuron to IC stimulations with 3 different intensities. The lowest-intensity stimulation evoked depolarizing potential with a relatively small amplitude and a short duration. A slight increase in stimulus intensity, on the other hand, led to depolarizations with much larger amplitudes and longer durations. The duration of depolarizing responses was also found to be related to the membrane potential of the neuron recorded. As can be seen in Fig. 6A, membrane hyperpolarizations by constant current injections reduce the duration of depolarizing responses. In most of the neurons the EPSPs were masked by preceding larger IPSPs. In 5 neurons, however, IC stimulation evoked EPSPs followed by IPSPs (Fig. 5C). During injection of continuous hyperpolarizing current, a burst of firing was triggered from the EPSPs (Fig. 5C). In the slice preparation obtained from animals which received chronic knife cuts at the level of EP, stimulation of IC immediately rostral to STH evoked monosynaptic EPSPs overlapping with
small IPSPs in all the neurons tested \((n = 8)\). These responses were very similar to those shown in Fig 5C. In some experiments the stimulation electrode was placed on the cerebral peduncle ventral to the SN in the slice obtained from normal animals. Stimulation of the cerebral peduncle in this preparation also evoked EPSPs similar to those shown in Fig. 5C, but the latency \((1.0-2.5\, \text{ms}; n = 4)\) of EPSPs was considerably shorter than that evoked by IC stimulation.

**DISCUSSION**

**Slice preparations**

We employed an in vitro slice preparation since STH is small and located deep in the brain making it difficult to reach the nucleus and to obtain a stable intracellular recording in in vivo preparations. The in vitro slice preparations, on the other hand, allow easy placement of the recording and stimulating electrodes under visual guidance and stable intracellular recordings without pulsations. Slice preparations also allow an investigator to manipulate the chemical environment of the recording neurons by changing the chemical composition of superfusing media.

**Passive membrane properties**

The mean input resistance \((146\, \text{M} \Omega)\) of rat STH neurons obtained from the present in vitro slice preparation was about 8 times higher than the value obtained from rat STH neurons in an in vivo preparation. Factors that could be responsible for the differences in the values obtained between in vitro and in vivo preparations include the following. The composition of the extracellular fluid is different in the in vitro preparation from that in the in vivo preparation. The nucleus is isolated from the extrinsic circuits which results in a lack of tonic synaptic inputs in in vitro preparation. Another difference is that the neuronal processes \((\text{e.g., dendrites and axons})\) may be severed in the slice preparation, reducing the area of the surface membrane which would influence the input resistance. The resting membrane potentials of the neurons analyzed in the in vivo preparation varied from 15 to 40 mV, while those of the neurons analyzed in this study were more than 40 mV. The difference in the membrane potential also would influence the input resistance since the present in vitro study revealed that the membrane of STH neurons has strong delayed rectification.

**Spontaneous firings**

Most of the STH neurons studied had spontaneous firings of 5–40 Hz. Spontaneous extracellular unit discharges \((23.3 \pm 9.8\, \text{Hz}; \text{mean} \pm \text{S.D.}; n = 26)\) were also frequently encountered in STH of slice preparations (Nakamshi et al., unpublished observation). We considered that the spontaneous firing was caused by a relatively low resting membrane potential of STH neurons, low threshold for Na-spikes and a strong delayed rectification, probably due to voltage-dependent K-conductance, which may prevent spike accommodation. The membrane potential of STH neurons reported in this study falls between 40 and 65 mV. These relatively low resting membrane potentials of STH neurons, however, are not likely to be a result of damage to the neuron by electrode penetration since \(n\) the recording was stable and could be maintained for at least 1 h, \(n\) the input resistance was relatively high \((70–250\, \text{M} \Omega)\), and \(n\) no large changes in the rate of spontaneous discharge were noted after penetration of the neuron. Linás and Ya-rom have described a sequence of events that causes the slow rhythmic discharge \((4–10\, \text{Hz})\) of inferior olivary neurons. The events include Na spike, high-threshold Ca spike, Ca-dependent K conductance and low-threshold Ca-spike. However, spontaneous firings of STH neurons may not be due to Ca-dependent potentials since \(n\) the firing frequency is relatively high and has a wide range \((5–40\, \text{Hz})\), \(n\) substitution of Mg\(^{2+}\) in the Krebs solution for Ca\(^{2+}\) does not cause a big change in the frequency nor in the pattern of spontaneous unit firing (Nakamshi et al., unpublished observation), \(n\) no spike after depolarization which might correspond to a high-threshold Ca spike can be detected (i.e., the duration of the action potential was about 1 ms).

**Ca-potentials**

Recent electrophysiological studies revealed that the neurons in many areas of the central nervous system are able to generate two or 3 different types of Ca-potentials. Recent electrophysiological studies revealed that the neurons in many areas of the central nervous system are able to generate two or 3 different types of Ca-potentials. The Ca potentials are thought to subserve a variety of functions in neuronal activities such as an intrinsic oscillatory mechanism.
synaptic inputs and a release of neuroactive substances. In this study we have revealed that STH neurons possess two distinct TTX-resistant potentials, namely the slow depolarizing potential and the slow action potential. These potentials were considered to represent activation of inward Ca currents since their generation was blocked by superfusion of Ca2+-free medium or application of Co2+ which is known to block Ca conductance. The slow depolarizing potential, with the characteristics of slow rising, long duration and relatively small amplitude, had similar characteristics to the chick sensory neurons and the Ca-dependent plateau potential recorded in the dendrites of cerebellar Purkinje cells. The slow depolarizing potentials are generated by depolarizing current pulses in many STH neurons having a membrane potential of more than 50 mV. The present study suggested that EPSPs could trigger the slow depolarizing potential, since the duration of depolarizing responses induced by IC stimulation was greatly altered by the changes in the stimulus intensities or membrane potential levels. Then, the slow depolarizing potential may play an essential role in the response to excitatory inputs since it could strongly enhance the duration and the amplitude of postsynaptic excitation which leads to triggering repetitive spikes.

It has been reported that stimulation of the cerebral cortex evoked a large, long-lasting depolarizing potential accompanied by a multiple firing of spikes in STH neurons. These response patterns are clearly in contrast to those of excitatory responses recorded in neonatal neurons. The response of the neonatal neurons after stimulation of the cerebral cortex is a short-duration depolarizing potential with one or two spikes. It may be that the difference in the response pattern in the two nuclei is a result of the generation of slow depolarizing potentials in STH neurons.

In this study, we have observed another Ca-dependent potential different from the slow depolarizing potential. We call this potential 'slow action potential', distinguished from the slow depolarizing potential by its fast rate of rise, a short duration and a large peak amplitude. In the preparation superfused with Krebs solution, generation of this potential was seen only in the neurons with a membrane potential more negative than 65 mV. These phenomena indicate that the Ca conductance responsible for the slow action potential is inactivated in the depolarized membrane, as has been previously observed in other central nervous system neurons. In preparations superfused with TEA-containing Ringer, however, the slow action potential was generated from neurons having membrane potentials less negative than 65 mV. The experiment with TEA revealed that the termination of the slow action potential involves activation of K conductances (i.e., probably both voltage- and Ca-dependent K conductances) since TEA effectively prolonged the duration of the potential. This result was consistent with the reports for other CNS neurons.

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The enhancement of excitation by the slow spike potential differs from that by the slow depolarizing potential, since the slow spike potential occurs only in the neuron which has a resting membrane potential of more than 65 mV and can trigger bursts of spikes.

**Origins of IC stimulation-induced IPSPs and EPSPs**

Stimulation of IC evoked short-latency monosynaptic IPSPs with overlapping EPSPs in STH neurons. The IPSPs were considered to be GABAergic and Cl-mediated since they were blocked by application of bicuculline and their polarity was reversed by intracellular injections of Cl-. These IPSPs were considered to be induced by stimulation of the axons originating from the GP and/or the entopeduncular nucleus (EP) since a chronic transection of the IC at the level of the EP resulted in a drastic reduction in the IPSPs induced by IC stimulation. A support for this interpretation also comes from a recent in vivo electrophysiological study which showed that stimulation of GP produced monosynaptic large-amplitude and short-duration IPSPs in STH neurons. These IPSPs are similar to those obtained in this study. The origin of EPSPs evoked by IC stimulation, however, remains unclear. Kitai and Deniau reported that stimulation of the cerebral cortex evokes monosynaptic EPSPs in STH neurons. However, we consider that the EPSPs observed in the present study are not cortical in origin for the following reasons: (1) the EPSPs evoked by cortical stimulation had much shorter latency (mean 2.5 ms) than the latency (mean 3.5 ms) of the EPSPs observed after IC stimulation, and (2) the EPSPs to IC stimulation were not...
abolished by chronic IC transection. Possible sources for the EPSPs are the activation of axon collaterals of STH neurons projecting to GP, the pedunculopontine tegmental nucleus, and the raphe nucleus. More studies are needed to clarify the origin of these EPSPs.

In summary, the present study demonstrated that STH neurons are under an influence of GABAergic and Glu-mediated inhibitory inputs originating from the globus pallidus. These neurons, however, are capable of high-frequency firing and possess features to self-regulate and intensify their excitatory inputs (e.g., cortical origin) through Ca-dependent potentials.

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