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Four Voltage-Gated Potassium Currents in Trigeminal Root Ganglion Neurons

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Various voltage-gated K^+ currents were recently described in dorsal root ganglion (DRG) neurons. However, the characterization and diversity of voltage-gated K^+ currents have not been well studied in trigeminal root ganglion (TRG) neurons, which are similar to the DRG neurons in terms of physiological roles and anatomy. This study was aimed to investigate the characteristics and diversity of voltage-gated K^+ currents in acutely isolated TRG neurons of rat using whole cell patch clamp techniques. The first type (type I) had a rapid, transient outward current (I_A) with the largest current size having a slow inactivation rate and a sustained delayed rectifier outward current (I_K) that was small in size having a fast inactivation rate. The I_A currents of this type were mostly blocked by TEA and 4-AP, K channel blockers whereas the I_K current was inhibited by TEA but not by 4-AP. The second type had a large I_A current with a slow inactivation rate and a medium size-sustained delayed IK current with a slow inactivation rate. In this second type (type II), the sensitivities of the I_A or I_K current by TEA and 4-AP were similar to those of the type I. The third type (type III) had a medium sized I_A current with a fast inactivation rate and a large sustained I_K current with the slow inactivation rate. In type III current, TEA decreased both I_A and I_K but 4-AP only blocked I_A current. The fourth type (type IV) had a smallest I_A with a fast inactivation rate and a large IK current with a slow inactivation rate. TEA or 4-AP similarly decreased the I_A but the I_K was only blocked by 4-AP. These findings suggest that at least four different voltage-gated K^+ currents in biophysical and pharmacological properties exist in the TRG neurons of rats.

Key words: Trigeminal root ganglion neurons, Voltage-gated K^+ currents

Introduction

Dorsal root ganglia (DRG) neurons transmit various sensory information such as touch, pressure, pain and temperature from the peripheral region to the CNS. Similarly, a pair of trigeminal root ganglia (TRG) is responsible for the sensory inputs from the oromaxillofacial region to which the trigeminal nerve is innervated.

TRG Primary afferent neurons are a functionally diverse population of neurons that transduce and encode a variety of stimuli. Some of this diversity may reflect a differential distribution of voltage-gated K^+ currents, a class of currents that plays an integral role in the regulation of a number of neuronal response properties including spike repolarization, interspike interval, and burst adaptation [1]. In broad category, K^+ currents carried by voltage-gated potassium channels are classified into 3 types; transient A type, delayed rectifier and in-

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ward rectifier [2]. Although two [3], and subsequently three [4,5] distinct voltage-gated K^+ currents have been identified in neurons from rat sensory ganglia, the steady-state and kinetic properties of these currents appear variable between studies. For an example, a slowly inactivating transient current (A-type K^+ current) is present in sensory neurons that has an inactivation time constant that varies between 150 and 300 ms.

In addition, although many investigators have reported the existence of a single sustained delayed rectifier-type current in sensory neurons, a recent report by Akins and McCleskey (1993) suggests that some delayed rectifier-type current in dorsal root ganglion (DRG) neurons is subject to steady- state inactivation [6]. One explanation for the variability observed by these investigators is that there are more than three distinct voltage-gated K^+ currents present in sensory neurons. In fact, six voltage-gated K^+ currents were recently reported to be existed in DRG neurons [7]. However characterization and diversity of voltage-gated K currents have been not well known in TRG neurons, similar to the DRG neurons in physiological roles and anatomy. This study was aimed to investigate the diversity and characterization of voltage-gated K^+ currents in acutely isolated SD rat TRG neurons using whole cell patch clamp technique because the electrophysiological mechanisms that give rise to the diversity of response properties observed among sensory neurons requires a thorough knowledge of the properties of the currents expressed in those neurons.

Materials and Methods

Preparation of rat TRG neurons

All experiments were carried out according to the guiding principles for the care and use of animals approved by the ethics committee in Chonnam and Chosun University and the National Institutes of Health Guide for the care and Use of Laboratory Animals, and every effort was made to minimize both the number of animals used and their suffering. Rat TRG neurons were prepared by the method described previously [8]. After the decapitation of Sprague-Dawley rats (postnatal 7-14 days), a pair of trigeminal ganglia were dissected and washed several times in cold (4℃) dissociation solution containing (in mM) 140 NaCl, 5 KCl, 22 KH_2PO_4, 17 Na_2HPO_4, 5 Glucose, 59 Sucrose, pH 7.2. They were incubated at 37℃ for 40 min in dissociation solution and incubated at 37℃ for 40 min in dissociation solution containing 1% trypsin. Then they were re-triturated and washed several times in modified MEM and maintained in 5% CO_2 incubator (37℃). The isolated cells were used in electrophysiological recording within 3 hrs.

Electrophysiology

Conventional whole cell voltage-clamp techniques were used for electrophysiological recording. Patch pipettes were pulled on a Brown Flaming electrode puller (model p-87, Sutter instruments, USA) and forged using microforge (MF-83, Narishige, Japan). The pipette resistance ranged from 2-4 MΩ when filled with electrode solution (see below). The recording chamber was continuously perfused with bath solution (flow rate 0.5 ml/min). All experiments were done at room temperature (21-24℃).

The bath solution was composed of (in mM) 135 NaCl, 5 KCl, 1 $MgCl_2$, 1.8 $MnCl_2$, 5 Glucose, 10 HEPES, pH 7.4. The pipette solution contained (in mM) 10 NaCl, 140 KCl, 1 $CaCl_2$, 1 $MgCl_2$. 1 GTP, 5 ATP, 10 HEPES, 10 EGTA, pH 7.2. Both solution's pH were adjusted with Tris-base. All components of the bath and electrode solutions were obtained from Sigma Co. The K^+ channel blocking agents, 4-aminopyridine (4-AP) and Tetraethylammonium (TEA) were applied to the bath using superfusion method.

Data recording and analysis

I_K currents of TRG neurons were amplified using an Axopatch 200 B patch clamp amplifier (Axon instruments, USA), filtered at 1 kHz with an 80-dB/decade low-pass Bessel filter. Data analysis was facilitated by the use of commercially available software programs including pClamp 7 and Origin 4.1. The neuron diameter was measured with an eye piece micrometer under phase contrast illumination. Normalized peak conductances (G/Gmax) and the data describing the fractional decrease in the peak currents during the steady-state inactivation (I/Imax) were fitted with a Boltzmann function ; I/I_{max} or $G/G_{max} = 1 + \exp(V_{1/2} - Vm)/k$.

Results

Characterization of type I K^+ current

Type I K^+ current was characterized as a whole current composed of a large rapid, transient outward current (I_A) having

Table 1. Electrophysiological and pharmacological comparison of voltage-gated K^+ currents in TRG neurons

Properties	I	II	III	IV
SS Inactivation (K1/2,mV)	-68.3±2.8 (n=11)	-62.3±2.1 (n=8)	-60.7± 2.2(n=10)	-79.1±1.9 (n=11)
SS Activation (K1/2,mV)	-33.3±3.2 (n=11)	-28.7±1.7 (n=8)	-41.2± 2.4(n=10)	-26.9±2.9 (n=11)
TEA sensitivity				
IA	++	++	+	-
Delayed IK	++	++	++	++
4-AP sensitivity				
IA	+++	+++	+++	+++
Delayed IK	-	-	-	++
Prepulse (-30 mV)	+	+	+	+
Prepulse (0 mV)				
Activation time constant (ms)	16.4±1.1 (n=11)	7.9±0.9(n =8)	17.7±0.9(n=10)	6.1±0.8(n =11)
I-V relation	R	R	R	R
IA size	L	L	M	S
IA Inactivation rate	slow	slow	fast	Fast
Delayed K current size	S	M	L	L
Delayed K current inactivation rate	fast	slow	slow	slow

Abbreviation: SS, steady-state; R, rectification; L, large; M, medium; S, small

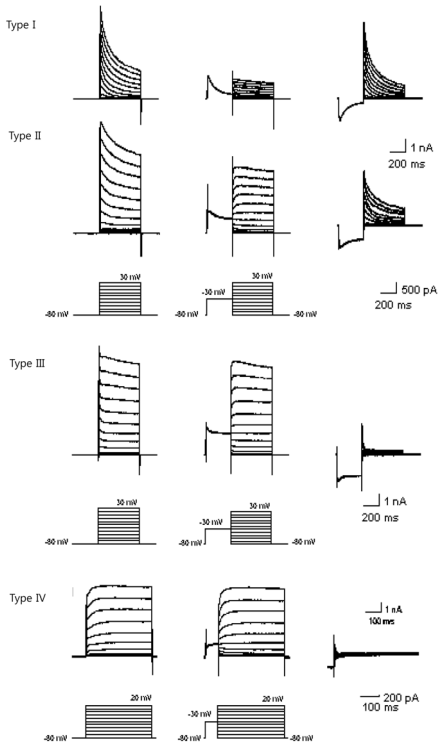

Fig. 1. Variability of the outward currents in different rat TRG neurons. Membrane potential held at -80 mV. The activation protocol consisted of 500ms or 1.5s depolarizing voltage steps to potentials ranging between -80 and +90 mV, after a 300 ms step to either -80, -30 and 0 mV.

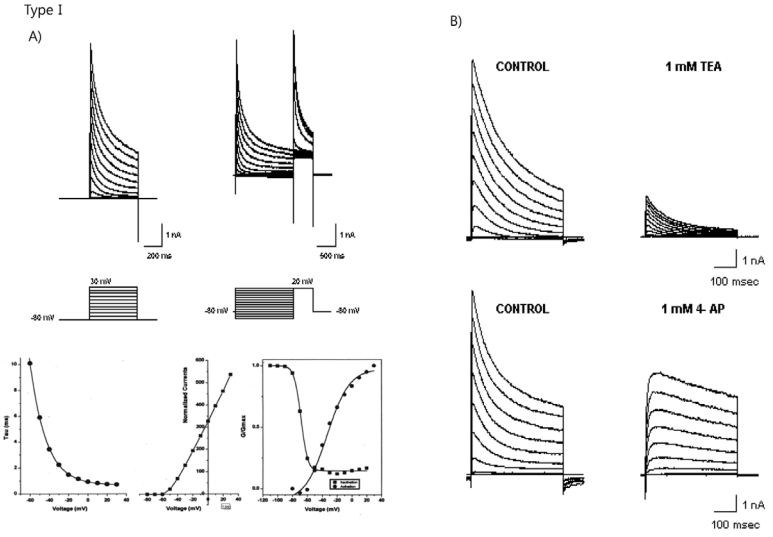

Fig. 2. Electrophysiological properties of Type I K^+ currents and effect of TEA and 4-AP on type I currents. A) The normalized peak currents vs. prepulse potential is plotted. The peak currents evoked from each prepulse potential in the steady-state inactivation protocol was normalized with respect to the peak current evoked from -120 mV($V_{1/2(\text{inact})}$ = -33.3±3.2 mV, n=11). Normalized peak conductance (G/Gmax) was fitted with a Boltzmann function ($G/Gmax = 1 + exp(V_{1/2}-Vm)/k$, $V_{1/2(\text{act.})}$ = -68.3±2.8 mV, n=11). B) Membrane voltage was held at -80 mV and stepped from -80 mV to 30 mV in 10 mV increment for 400 msec and held at -80 mV again. Sampling rate was 500 μs(2 kHz). The applicated concentrations of Tetraethylammonium (TEA) and 4-aminopyridine (4-AP) were indicated. 4-AP block selectively a transient component of the outward currents, sparing a sustained component while a transient current and sustained current were sensitive to the TEA.

slow inactivation rate and a small delayed sustained outward current (I_K) with fast inactivation rate (Table 1, Fig. 1 and 2). I_A was isolated as the difference between the current evoked from a holding potential +80 mV and that evoked from a holding potential of -30 mV (Fig. 1). Type I I_A was a relative large current that had the relative slow inactivation rate even though its activation rate is faster than type II.

Half-maximum activation and inactivation of I_A were -33.3 mV and -68 mV (Table 1, Fig. 2). Activation time constant of I_A showed an exponential dependence on membrane potential change e-fold in 16.6 mV. I_K in type I was relatively small current with the fast inactivation rate. Type I_A was mostly blocked TEA and 4-AP while I_K was significantly inhibited by TEA but not by 4-AP (Table 1, Fig. 2).

Characterization of type II K^+ current

Type II K^+ current was characterized as a whole current composed of a large I_A with slow inactivation and a relative medium size I_K with slow inactivation rate (Table 1, Fig 1). Type II I_A was a relatively large current which had relatively slow inactivation rate and blocked with -30 mV pre-pulse. Half-maximum activation and half-maximum steady-state

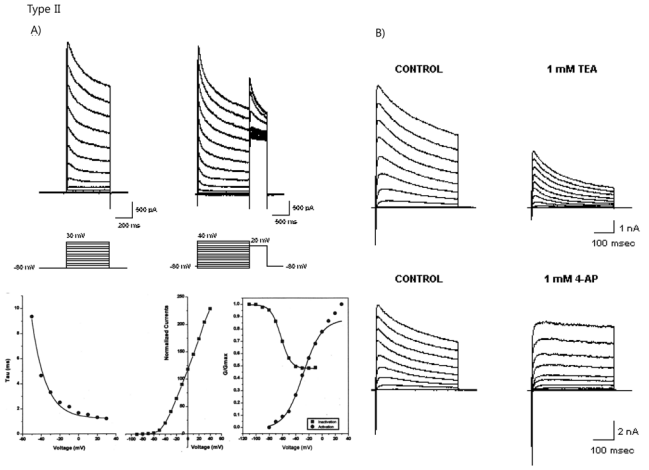

Fig. 3. Electrophysiological properties of Type II currents and effect of TEA and 4-AP on type II currents. A) Normalized peak currents (I/Imax) and normalized peak conductance (G/ Gmax) was fitted with a Boltzmann function ($K_{1/2(act)}$ = -28.7± 1.7 mV, $K_{1/2(inact)}$ = -62.3±2.1 mV, n=8). B) Membrane voltage was held at -80 mV and stepped from -80 mV to 30 mV in 10 mV increment for 400 msec and held at -80 mV again. Sampling rate was 500 μs(2 kHz). The applicated concentrations of Tetraethylammonium (TEA) and 4-aminopyridine (4-AP) were indicated. 4-AP block selectively a transient component of the outward currents, sparing a sustained component.

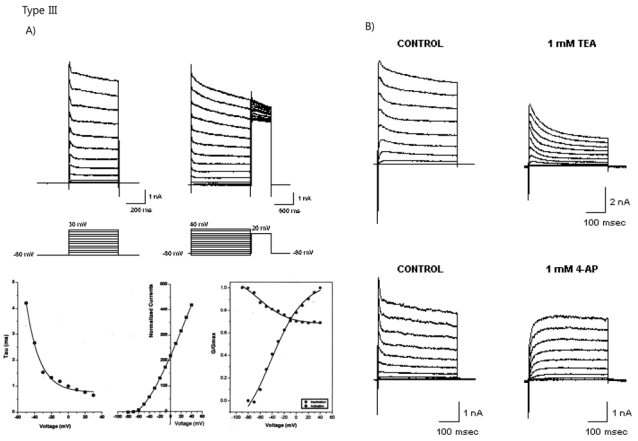

Fig. 4. Electrophysiological properties of Type III currents and effect of TEA and 4-AP on type III currents. A) Normalized peak currents (I/Imax) and normalized peak conductance (G/ Gmax) was fitted with a Boltzmann function ($K_{1/2(act)}$ = -41.2± 2.4 mV, $K_{1/2(inact)}$ = -60.7±2.2 mV, n=10). B) Membrane voltage was held at -80 mV and stepped from -80 mV to 30 mV in 10 mV increment for 400 msec and held at -80 mV again. Sampling rate was 500 μs(2 kHz). The applicated concentrations of Tetraethylammonium (TEA) and 4-aminopyridine (4-AP) were indicated. 4-AP blocked selectively a transient component of the outward currents, sparing a sustained component.

inactivation of I_A were -28.7 and -62.3 mV (Table 1) Activation time constant showed an exponential dependence on membrane potential change *e*-fold in 14.7 mV (Table 1, Fig. 3). I-V relationship curve showed linear rectification and voltage dependency (Fig. 3).

I_K was small and had slow inactivation rate. Sensitivities of TEA and 4-AP to the I_A or I_K were similar to those of the type ; 4-AP almost blocked I_A but not I_K whereas TEA significantly inhibited I_A and I_K (Table 1, Fig. 3).

Characterization of type III K^+ current

Type III K^+ current was characterized as a medium I_A with slow inactivation rates and a large I_K with fast inactivation rates (Table 1, Fig. 1 and 4). Type III I_A was relatively medium size current and showed fast activation rate blocked by -30 mV pre-pulse. Half-maximum activation and half-maximum inactivation of I_A were 41.2 mV and -60.7 mV. Activation time constant of I_A showed an exponential dependence on membrane potential change *e*-fold in 9.46 mV. The I_K was relatively large current with slow inactivation rates. Type III I_A was completely blocked by 4-AP and partially inhibited by TEA whereas I_K was only significantly blocked by TEA but not by 4-AP (Fig. 4).

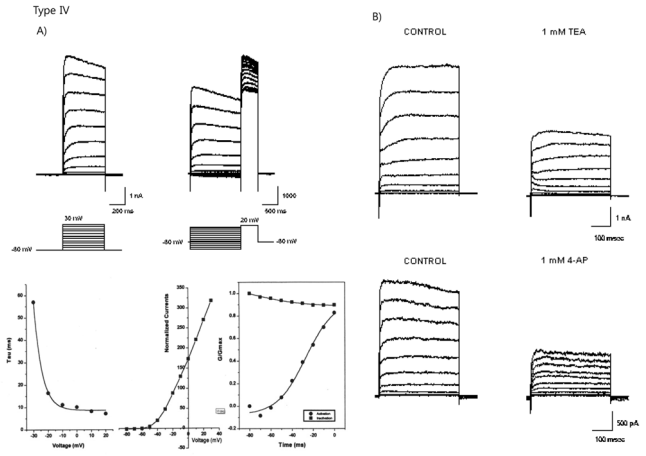

Fig. 5. Electrophysiological properties of Type IV currents and effect of TEA and 4-AP on type IV currents. A) Normalized peak currents (I/Imax) and normalized peak conductance (G/Gmax) was fitted with a Boltzmann function ($K_{1/2(act)}$ = -26.9± 2.9 mV, $K_{1/2(inact)}$ = -79.1±1.9 mV, n=11). B) Membrane voltage was held at -80 mV and stepped from -80 mV to 30 mV in 10 mV increment for 400 msec and held at -80 mV again. Sampling rate was 500 μs (2 kHz). The applicated concentrations of Tetraethylammonium (TEA) and 4-aminopyridine (4-AP) were indicated. 4-AP blocked selectively a transient component of the outward currents, sparing a sustained component.

Characterization of type IV K^+ current

Type IV K^+ current had a very small I_A current with fast inac tivation or only had a I_K current with slow activation and inactivation rates (Table 1, Fig. 1 and 5). Type IV I_A was only a small size current with fast inactivation rates which blocked by -30 mV pre-pulse. Half-maximum activation and half-maximum inactivation rates were -26.9 mV and -79.1 mV. I_K was relatively a large current and showed the slow inactivation rates.

The I_A was only sensitive to 4-AP but I_K was significantly blocked by both TEA and 4-AP (Fig. 5)

Discussion

Primary afferent neurons of the mammalian DRG and TRG are classified on basis of the morphological and electro physiological characteristics of neurons. Large neurons with myelinated Aα and β fibers propagating touch and pressure have a fast conducting velocity whereas small neurons with myelinated Aδ and unmyelinated C fibers propagating pain and temperature have a slow conducting velocity [9]. Rat TRG neuron isolated by enzyme in this study showed various sizes ranging from 15 to 40 μm. However, properties of the neuronal action potential are known to be associated with the cell types at lesser extent; long action potential with inflected repolarization is mainly recorded from Aβ and C neurons and short action potential without plateau during falling phase is recorded from Aα and Aδ [10].

Intracellular recordings in the mammalian TRG neurons have shown that three types of action potential exist; 1) fast spikes, most of which are completely blocked by tetrodotoxin (TTX), 2) CO^{2+}-and TTX-resistant humped spikes [11] and 3) slowly decaying TTX-resistant and Cd^{2+}-sensitive action potentials [12]. These diversities of neuronal size and action potential in TRG may reflect that various types of voltage-dependent ion channels are expressed in TRG. In fact, at least two types of voltage-gated sodium current (I_{Na}) distinguished by their sensitivity to TTX [13-15] and T, L, N and P types of voltage-gated calcium current (I_{Ca}) have been reported to exist in DRG and TRG. However, diversity and characteristics of voltage-gated potassium currents have been not well studied in sensory neurons because characteristics of voltage gated K^+ current are changed according to developmental stage and have complicated properties [16].

Voltage-gated K^+ currents are traditionally classified in terms of three families, distinguished mainly by their responses to changes in membrane potential ; 1) transient (A-type) K^+ current activated by depolarization and decayed spontaneously and rapidly while the depolarization is maintained, 2) delayed rectifier K^+ current with a brief delay following the onset of a membrane depolarization and sustained while the depolarization is maintained and 3) inward rectifier opened by a large hyperpolarization [17]. Transient A type and delayed rectifier K^+ current are known to exist in neuronal cell. Even in this experiment, whole K^+ currents were composed of a transient and a delayed outward current in TRG neurons but inward rectifier currents were not observed at voltage steps more than -80 mV.

Gold et al. (1996) recently reported that DRG sensory neurons express at least different six voltage-gated K^+ currents in electrophysiological and pharmacological properties; three transient and three sustained currents [7]. However, characterization and diversity of voltage-gated K^+ current in TRG have been not studied. Therefore, in this study voltage-gated K^+ currents in TRG were firstly separated on

basis of total shape of whole current composed of a transient outward current and a delayed sustained outward current unlike a previous study of which each transient and sustained K^+ currents were independently separated in DRG neurons. Furthermore, electrophysiological kinetics and pharmacological properties of various voltage-gated K^+ current were examined in this study.

In this study, at least four different voltage-gated outward K^+ currents were observed in TRG neurons; 1) type I outward K^+ current characterized as a whole current composed of a large rapid, transient outward current (I_A) with slow inactivation rates and a small delayed, sustained outward current (I_K) with fast inactivation rate, 2) type II K^+ current characterized as a whole current composed of a large I_A with slow inactivation rate and ad medium size I_K with slow inactivation rate, 3) type III K^+ current composed of a medium size I_A with fast inactivation and a large I_K with very slow inactivation, and 4) type IV K^+ current composed of a very small I_A current with inactivation and a large I_K with very slow inactivation.

In the present study, four voltage-gate K^+ currents found in TRG neurons showed apparent difference in electrokinetic and pharmacological properties. The degree of I_A size among four K^+ current types in TRG neuron was in order of type I > type II > type III > type IV. I_A inactivation rates of type I and II K^+ currents were relatively slow or a little slow while those of type III and IV were rapid. I_A currents in all K current types were sensitive to 4-AP, a voltage-gated K channel blocker, indicating that 4-AP significantly decreased I_A current evoked by voltage clamping in all types. These results were consistent with previous reports that 4- AP is sensitive to the transient A current [2,18]. Even if TEA, a voltage-gated K^+ channel blocker, has been known to be more sensitive to I_K than I_A [19], TEA significantly decreased I_A in type I and II K^+ currents of TRG. In the present study, type I and II K^+ currents were found in large size TRG neurons (>30 μm) while type III and IV K^+ currents were observed in small size TRG neuron (<30 μm). These results suggest that type I and II K^+ currents may play a role in regulation of spike frequency and resting membrane potential in TRG large neurons with fast conducting velocity, whereas type III and IV K^+ currents may play a pivotal role in regulation of those in small neuron with slow conducting velocity.

On the other hand, physiological role for various voltage-gated K^+ currents in TRG was not clear in this experiment. However, it was speculated how various K^+ currents with respect to somatic sense properties might regulate neuronal excitability. For an example, a slowly inactivating transient K^+ current in sensory neurons functions to limit excitability, because a selective block of the current decreases both action potential-threshold and accommodation [5]. These changes are associated with sensitization of nociceptor by hyperalgesic inflammatory agents such as prostaglandin and serotonin, suggesting that these agents might modulate I_A currents in nociceptive neurons. In addition, various delayed I_K current may be responsible for repolarizing changes of the action potential, resulting into longer action potential and increased neurotransmitter release.

The role of K^+ currents in the cell body is less clear, because there is evidence that antidromically conducting action potentials fail to penetrate the cell body [20]. However, there is a growing body of evidence to suggest that the cell body becomes a source of excitability after injury [21-23]. Thus an injury-induced modulation of the K^+ currents presented in the cell body could contribute to these changes in excitability.

In summary, at least four different voltage-gated K^+ currents in electrophysical and pharmacological properties existed in TRG neurons of the rats.

Acknowledgements

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