Expression of inwardly rectifying $K^+$ channels in the carotid body of rat

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Summary. The inwardly rectifying $K^+$ channels, Kir1.1, Kir2.3, Kir4.1-Kir5.1, and Kir4.2-Kir5.1, are candidate chemosensory molecules for CO$_2$/H$^+$. Here, we determined the mRNA expression and immunohistochemical localization of these channels in the carotid body (CB) and petrosal ganglion (PG) of the rat. RT-PCR analysis revealed mRNA expression of Kir4.1 and Kir5.1 in CB, and Kir1.1, Kir4.1, and Kir5.1 in PG. Immunohistochemistry identified the glomus cells in CB to express both Kir4.1 and Kir5.1 protein, while the nerve fibers in CB were immunoreactive for Kir1.1, Kir4.1, and Kir5.1. In the PG, immunoreactivity for Kir1.1, Kir4.1, and Kir5.1 was observed in some ganglion cells. Our findings suggest that Kir channels in the peripheral chemoreceptors play a role in sensing hypercapnic acidosis and maintaining the resting membrane potentials.

Key words: Hypercapnic acidosis, Inwardly rectifying $K^+$ channel, Carotid body, Petrosal ganglion, Chemoreceptor

Introduction

Carotid body is the arterial chemoreceptor that percept low pO$_2$, high pCO$_2$, and acidity of the blood. This organ contains chemoreceptive glomus cells and supporting sustentacular cells, and innervated by carotid sinus nerve derived from glossopharyngeal nerve. The two hypotheses have been proposed for signaling pathway for acute response of glomus cells against hypoxia, i.e., metabolic hypothesis and ion channel hypothesis (Lahiri et al., 2001). The metabolic hypothesis states that mitochondrial respiration mediates the activation of glomus cells for acute hypoxia. In this hypothesis, it is suggested that hem-containing proteins such as mitochondrial chytochrome aa$_3$ are the primary oxygen sensor for arterial pO$_2$ in the glomus cells (Lahiri and Acker, 1999). On the other hand, the ion channel hypothesis states that potassium channels on the glomus cell membrane are primary O$_2$ sensors that are closed by decreased oxygen. In the carotid body, it has been reported various candidates of potassium channels for this hypothesis. Studies using patch clamp technique have suggested that hypoxia-induced depolarization of glomus cells is due to inhibition of voltage-gated potassium channel, Kv4.1 and Kv4.3 in rabbit (Sanchez et al., 2002) and Kv3 channels in mouse (Pérez-García et al., 2004). Other studies have shown that background leak current are suppressed by hypoxia in glomus cells, suggesting two pore domain potassium channels, TASK, are primary sensor for hypoxia (Buckler et al., 2000).

As mentioned above, the glomus cells also response to arterial hypercapnia or acidosis. Previous studies reported that methazolamide, a carbonic anhydrase inhibitor, reduced hypercapnia-induced stimulation of carotid sinus nerve activity (Iturriaga et al., 1991, 1993). Because carbonic anhydrase catalyzes CO$_2$+H$_2$O$\rightleftharpoons$H$^+$+HCO$_3^-$, it is suggested that a decrease in intracellular pH (pHi) is the initial response of glomus cells for hypercapnic stimuli (Lahiri and Forster, 2003). During sensing of CO$_2$, K$^+$ current inhibition may be important during the membrane depolarization of chemosensory cells and subsequent events such as enhancing L-type Ca$^{2+}$ currents and neurotransmitter discharge (Summers et al., 2002). Furthermore, it is reported that extracellular pH can also suppress outward K$^+$ current in the glomus cells (Peers, 1990a; Stea et al., 1991; Buckler et al., 2000), and that part of the carotid sinus nerve discharge is known to be independent of elevated intracellular Ca$^{2+}$ in the glomus cells (Roy et al., 2000). Thus, it is possible that hypercapnic and/or
acidic stimulation directly activate glomus cells and/or nerve endings of petrosal ganglion cells in the carotid body as the acid-sensitive vagal nerve endings in the respiratory tract (Kollarik and Undem, 2002).

There are several candidates for primary sensor for acidity in carotid body. In the glomus cells, one of the K⁺ channel family, TASK, generates outwardly background K⁺ currents, and is inhibited by acidity (Buckler et al., 2000). Another candidate is inwardly rectifying potassium (Kir) channels, because previous studies revealed that several Kir subunits are sensitive to hypercapnic acidosis in the brain (see review; Jiang et al., 2001). Kir channels consist of several subfamilies (Isomoto et al., 1997; Nichols and Lopatin, 1997). These channels are important for the modulation of cell excitability and the maintenance of K⁺ homeostasis (Ruppersberg, 2000). Kir channels are found throughout the body, but are distributed in an organ- and cell-specific manner. Within the physiological range, homotetramers consisting of Kir1.1 or Kir2.3 (Zhu et al., 1999; Xu et al., 2000b) and heterotetramers consisting of Kir4.1-Kir5.1 or Kir4.2-Kir5.1 are sensitive to intracellular and/or extracellular protons (Yang et al., 2000; Zhu et al., 2000; Cui et al., 2001; Pessia et al., 2001; Casamassima et al., 2003). In situ hybridization revealed colocalization of Kir4.1 and Kir5.1 mRNAs in the chemosensitive neurons of the rat central nervous system (Wu et al., 2004). Thus, Kir is a candidate molecule for primary H⁺ sensing in central and peripheral chemosensitive cells. Furthermore, there are no reports of Kir subunit expression in the peripheral chemosensory organs.

In the present study, we examined expression and immunohistochemical distribution of the H⁺-sensitive Kir subunits, Kir1.1, Kir2.3, Kir4.1, Kir4.2, and Kir5.1, in the carotid body and petrosal ganglion, by RT-PCR to profile the mRNA expression and by immunolocalization of these subunits. We discuss the function of Kir as the primary sensor molecule for acid sensing in peripheral chemosensors.

Materials and methods

Materials

Male and female Wistar rats (n=12) were used in the present study, which was approved by the local ethics committee. For RT-PCR analysis, three rats were sacrificed in a chamber filled with diethyl ether gas, for dissection of the bifurcation of carotid arteries and petrosal ganglion. The carotid bodies were further dissected microscopically. Pairs of carotid bodies and the petrosal ganglia were used for RNA isolation. All tissue samples were frozen in liquid N₂.

RT-PCR amplification

Total RNAs from the carotid bodies and petrosal ganglion were isolated using a commercial kit (MELT™ total nucleic acid isolation system, Ambion, Austin, TX). RNA templates were incubated with DNAase I (Takara, Tokyo, Japan) for 30 min at 37°C before use. RT-PCR was performed with the Qiagen OneStep RT-PCR kit (Qiagen, Hilden, Germany) with specific primers for Kir1.1, Kir2.3, Kir4.1, Kir4.2, and Kir5.1. GAPDH expression was also monitored as a control housekeeping gene. The primers used are detailed in Table 1. Reverse transcription conditions were as follows: 30 min at 50°C for reverse transcription, and 15

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<th>Product length (bp)</th>
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<td>Kir1.1 (X7234)</td>
<td>5'-CAGAAGTTGATGAAACGGGACG-3' (sense)</td>
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<td>148</td>
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<td></td>
<td>5'-GTGCTAGAGATTTGCATG-3' (antisense)</td>
<td>1419-1438</td>
<td></td>
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<tr>
<td>Kir2.3 (U27582)</td>
<td>5'-GGACATCCTTCCACGGGACGTCG-3' (sense)</td>
<td>507-525</td>
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<td>5'-GAACCCATAACCGTAGTCG-3' (antisense)</td>
<td>767-786</td>
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<tr>
<td>Kir4.1 (X86818)</td>
<td>5'-GGACATCCTTCCACGGGACGTCG-3' (sense)</td>
<td>836-857</td>
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<td>1041-1060</td>
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<td>1096-1114</td>
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<tr>
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<td>330</td>
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<td></td>
<td>5'-AGATGAGACGCTGTCG-3' (antisense)</td>
<td>858-876</td>
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<tr>
<td>GAPDH (AF106860)</td>
<td>5'-GGAGTGTCTTCCACGGGACGTCG-3' (sense)</td>
<td>1130-1148</td>
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<td></td>
<td>5'-AGATGAGACGCTGTCG-3' (antisense)</td>
<td>1513-1530</td>
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min at 95°C for initial PCR activation. After reverse transcription, PCR amplification was performed 40 times as follows: 30 sec at 94°C for denaturation, 30 sec at 55°C for annealing, and 1 min at 72°C for extension. After PCR amplification, samples were applied for 10 min at 72°C for final extension. PCR end products were visualized on 2% agarose gels with ethidium bromide. For negative-control experiments, the mRNA template was omitted.

**Immunohistochemistry**

Sections were stained for immunohistochemistry by the avidin-biotin-peroxidase complex (ABC) method. The sections were treated in 0.3% H₂O₂ in methanol to block endogenous peroxidase activity and incubated for 60 min with non-immune donkey serum (1:50). They were then rinsed in PBS and incubated overnight at 4°C with antisera against Kir1.1, Kir2.3, Kir4.1, Kir4.2, or Kir5.1 (Table 2). After incubation, the sections were washed again in PBS, and then treated with biotinylated donkey secondary antibody for 30 min at room temperature. Finally, the sections were incubated with the ABC kit reagent (Elite ABC kit, Vector, Burlingame, CA) for 30 min at room temperature. The immunoreaction sites were visualized by incubation with Tris-HCl buffer containing 3,3′-diaminobenzidine tetrahydrochloride (0.2 mg/ml) and 0.003 % H₂O₂. For negative controls, sections were incubated with preabsorbed antibodies or PBS. Details of antibodies and concentrations of antigen for preabsorption testing are summarized in Table 2.

**Double immunofluorescence**

Cryostat sections were also used for double immunofluorescence for Kir1.1, Kir4.1 or Kir5.1 with tyrosine hydroxylase (TH) and for Kir4.1 with Kir5.1. Sections of the petrosal ganglion were also stained for Kir1.1, Kir4.1 and Kir5.1 with glial fibrillary acidic protein (GFAP). After incubation with normal donkey serum, sections were incubated with antisera against Kir1.1, Kir4.1 or Kir5.1 together with antibody against mouse monoclonal antibody against TH or GFAP for 12 hours at 4°C. Other sections were incubated with antisera against Kir4.1 with that against Kir5.1. Then, the sections were incubated with a mixture of FITC- and TRITC-labeled donkey secondary antibodies for 2 hours at 25°C. Some sections were counterstained with DAPI (1 µg/ml) for 15 min, and were coverslipped with glycerol-PBS, and examined under an epifluorescence microscope. Details of antibodies were tabulated in Table 2.

**Results**

**RT-PCR**

PCR products for Kir4.1 and Kir5.1 mRNAs were detected in the carotid body, while no mRNA expression of Kir1.1, Kir2.3, or Kir4.2 was observed (Fig. 1, left panel). In the petrosal ganglion, PCR products for Kir1.1, Kir4.1, and Kir5.1 were detected (Fig. 1, right panel). Appropriate sized transcripts of the housekeeping gene, GAPDH, were detected in all samples. No signal was detected in the negative controls without mRNA template (data not shown).

**Immunohistochemistry**

Glomus cells in the carotid body were immunopositive for Kir4.1 and Kir5.1, but not for Kir1.1, Kir2.3, and Kir4.2 (Table 1, Fig. 2). The staining of Kir4.1 in the glomus cells was relatively intense, while that of Kir5.1 was only weakly positive. A few nerve fibers around the glomus cells were also

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**Table 2. Antibodies used in the present study.**

<table>
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<th>Code</th>
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<td>APC-001</td>
<td>rabbit</td>
<td>1:50</td>
<td>3 µg/1 µg</td>
<td>A</td>
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<tr>
<td>AB5376</td>
<td>rabbit</td>
<td>1:100</td>
<td>1 µg/1 µg</td>
<td>B</td>
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<tr>
<td>APC-035</td>
<td>rabbit</td>
<td>1:200</td>
<td>1 µg/1 µg</td>
<td>A</td>
</tr>
<tr>
<td>AB5880</td>
<td>rabbit</td>
<td>1:100</td>
<td>1 µg/1 µg</td>
<td>B</td>
</tr>
<tr>
<td>SC-22434</td>
<td>goat</td>
<td>1:50</td>
<td>1 µg/1 µg</td>
<td>C</td>
</tr>
<tr>
<td>MAB318</td>
<td>mouse</td>
<td>1:250</td>
<td></td>
<td>B</td>
</tr>
<tr>
<td>GA-5</td>
<td>mouse</td>
<td>1:100</td>
<td></td>
<td>D</td>
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<tr>
<td>Biotinylated anti-rabbit IgG (H+L) 711-065-152 donkey</td>
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<td>Biotinylated anti-goat IgG (H+L) 705-065-147</td>
<td>1:500</td>
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<td>FITC-conjugated anti-rabbit IgG (H+L) 711-095-152 donkey</td>
<td>1:100</td>
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<td>TRITC-conjugated anti-rabbit IgG (H+L) 711-025-152 donkey</td>
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<td>FITC-conjugated anti-mouse IgG (H+L) 715-095-150 donkey</td>
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<td>TRITC-conjugated anti-goat IgG (H+L) 705-025-147 donkey</td>
<td>1:100</td>
<td>E</td>
<td></td>
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A, Alomone, Jerusalem, Israel; B, Chemicon, Temecula, CA; C, Santa Cruz Biotechnology, Santa Cruz, CA; D, LabVision, Fremont, CA; E, Jackson ImmunoResearch, West Grove, PA.
immunoreactive for Kir1.1 and Kir4.1 (Fig. 2D, F). The sections immunostained for Kir5.1 showed a dense network of varicose nerve fibers around the glomus cell clusters (Fig. 2C, F). Sustentacular cells and Schwann cells were not immunoreactive for any of the Kir subunits.

Most of the nerve cell bodies in the petrosal ganglion were immunoreactive for Kir1.1, Kir4.1, and Kir5.1, although some small neurons showed no staining (Fig. 2G-I). A few nerve fibers were immunoreactive for Kir1.1, Kir4.1 (Fig. 2G,H), while many nerve fibers also showed staining of Kir5.1 (Fig. 2I). The petrosal ganglion was not immunopositive for either Kir2.3 or Kir4.2.

Immunoreactivity was diminished in the negative control sections incubated with preabsorbed antibodies, and no signal was observed in the controls without primary antisera (Fig. 2J-L).

**Double immunofluorescence**

Immunoreactive sites for TH, the general marker for glomus cells, were also immunoreactive for Kir4.1 (Fig. 3A-C). Weak immunoreactivity for Kir5.1 is also observed in the TH immunoreactive glomus cells (figure not shown). No immunoreaction site for Kir1.1 was observed in the TH-immunoreactive sites. In the case of the sections stained for Kir4.1 and Kir5.1, numerous nerve fibers immunoreactive for Kir5.1 were observed around the Kir4.1 immunoreactive glomus cells (Fig. 3D-F). Almost Kir5.1 immunoreactive nerve fibers were not immunoreactive for Kir4.1.

In the petrosal ganglion, the satellite cells immunoreactive for GFAP were also positive for Kir1.1, Kir4.1 and Kir5.1 in addition to nerve cell bodies (Fig. 3G-I).

**Discussion**

The results of the present study revealed that Kir channels were expressed in carotid body at mRNA and protein levels. The pKa value (midpoint of channel inhibition) of Kir4.1 homomeric channels for intracellular acidity is pH 6.03, which is lower than the physiological decrease that occurs in acidosis, whereas the pKa of Kir4.1-Kir5.1 heteromeric channels is pH 7.45 (Xu et al., 2000a). In the astrocytes in neocortex and olfactory bulb of mouse, Kir4.1-Kir5.1 heteromeric channel was identified (Hibino et al., 2004). In the present study, both Kir4.1 and Kir5.1 channels were distributed in the glomus cells of the carotid body. Therefore, it is suggested that Kir4.1 and Kir5.1 form heteromeric channel in the glomus cells to decrease potassium current during hypercapnic acidosis.

In the rat brain, Kir4.1 and Kir5.1 subunits do not form heteromeric channels according to a previous biochemical analysis (Tanemoto et al., 2000). The weak immunoreactivity for Kir5.1 in the glomus cells, which was revealed in the present study, may indicate some part of the Kir4.1 form homomeric channels. It has been reported the homomeric Kir4.1 channels play an important role in regulation of membrane potentials of the glial cells in the brain (Butt and Kalsi, 2006). Thus, the homomeric Kir4.1 channels may maintain resting membrane potentials also in the glomus cells. Electrophysiological studies identified different
Fig. 2. Immunoreactivity for Kir1.1 (A, D, G), Kir4.1 (B, E, H), and Kir5.1 (C, F, I) in the carotid body (A-F) and the petrosal ganglion (G-I). Rectangles in panels A-C are enlarged in panels D-F. Note the presence of small number of nerve fibers immunoreactive for Kir1.1 (arrowheads in D) around the immunonegative glomus cells (asterisks in D). Note also Kir4.1 immunoreactivity in the glomus cells (arrow in E) and in some nerve fibers (arrowheads in E). Note the dense network of varicose nerve fibers immunoreactive for Kir5.1 (arrowheads in F) around clusters of the glomus cells (arrows in F). In the petrosal ganglion, both nerve cell bodies (arrows in G-I) and nerve fibers (arrowheads in G-I) are immunoreactive for Kir1.1, Kir4.1, and Kir5.1. Note also that some ganglion cells are immunonegative for all Kir-specific antibodies (asterisks in G-I). No positive reaction is shown in the carotid body sections those stained with preabsorbed antibodies (J-L).
Kir channels in carotid body

Fig. 3. A-F. Double immunofluorescence for Kir4.1 combined with TH (A-C) and Kir5.1 (D-F). Panels C and F are merged image of A-B and D-E, respectively. Glomus cells are immunoreactive for both Kir4.1 and TH (arrows in A-C). Kir4.1-immunoreactive glomus cells are weakly immunoreactive for Kir5.1 (arrows in D-F). Nerve fibers around the glomus cells are immunoreactive for Kir5.1 (arrowheads in E, F). G-I. Double immunofluorescence for Kir1.1, Kir4.1 and Kir5.1 combined with GFAP. Schwann cells are immunoreactive for these channels (arrows). Nuclei are stained with DAPI.
potassium currents in the glomus cells including Ca\(^{2+}\)-
dependent K\(^+\) current (Peers, 1990b; Pardal et al., 2000),
voltage dependent K\(^+\) current (López-López et al., 1993;
Sanchez et al., 2002) and HERG-like current (Overholt et al.,
2000) and background leak current (Buckler et al.,
2000). The potassium current in the glomus cells might
be regulated by multiple channel systems, and Kir
channels may take part in the modulation of potassium
current of glomus cells.

Buerk et al. (1998) reported that carotid sinus nerve
activity showed synergic action between hypoxic and
hypercapnic stimuli but dopamine release did not.
Furthermore, the intracellular Ca\(^{2+}\) responses in isolated
glomus cells to hypercapnic acidosis and hypoxia are
multiplicative in most cases (Dasso et al., 2000). Ituriaga
et al. (1991, 1993) reported that methazolamide,
inhibitor of carboxic anhydrases, decreases hypoxia-
or hypercapnia-induced an increase of carotid sinus nerve
activity. Activation of the petrosal ganglion cells that
cocultured with glomus cells is also reduced by
inhibition of carboxic anhydrase (Zhang and Nurse,
2004). Through the immunohistochemistry, we
previously reported that carboxic anhydrase isozymes I,
II, and III, which increase intracellular H\(^+\) under
hypercapnia, are also localized in rat glomus cells
(Yamamoto et al., 2003). Taken together, it is suggested
that elevation of intracellular H\(^+\) by carboxic anhydrase
under hypercapnia induce activation of glomus cells.
Kir4.1-Kir5.1 heteromeric channels might act in
sensitization of glomus cells to hypercapnia. On the
other hand, TASK-like potassium current was inhibited
by hypoxia and acidity (Buckler et al., 2000) and
expression of the TASK-1 and/or TASK-3 channels in
glomus cells was shown by in situ hybridization and
immunohistochemistry (Buckler et al., 2000; Yamamoto
et al., 2002). These channels may act co-operatively with
Kir4.1-Kir5.1 to inhibit potassium currents under
hypercapnic acidosis.

In the petrosal ganglion cells, the electrophysiological study revealed the delayed
rectifying K\(^+\) current and Ca\(^{2+}\) activated K\(^+\) current (Stea
and Nurse, 1992) and immunohistochemical analysis
showed several Kv channels (Andrews and Kunze,
2001). In the present study, a part of petrosal ganglion
cells expressed Kir1.1, Kir4.1 and Kir5.1. Thus, Kir
channels may be another modulator of potassium
currents in the petrosal ganglion cells. The
heterogeneous expression of Kir1.1, Kir4.1, and Kir5.1
in the petrosal ganglion cells may reflect functional
differences of the neurons. Because numerous nerve
fibers immunoreactive for Kir5.1 in the carotid body.
Kir5.1 in the petrosal ganglion cells may play important
roles for chemosensory transduction from glomus cells.
It has been reported that the binding of Kir5.1 with PSD-95
was prevented by protein kinase A (PKA)-mediated
phosphorylation of its carboxyl terminus in the
HEK293T cells cotransfected with Kir5.1 and PSD-95
(Tanemoto et al., 2002). In the carotid body, Kir5.1
homomeric channels may modulate the potassium
currents of nerve terminals at the synaptic sites. Acid-
induced activation of nerve endings via Kir channels
may be limited, because Kir1.1- and Kir4.1-
immunoreactive nerve fibers in the carotid body were
very small in number. It has been reported that acid
sensitive cation channels, i.e., TRPV1 and ASIC3, are
immunohistochemically distributed (Matsumoto et al.,
2001; Fukuda et al., 2006) in the petrosal ganglion cells.
The Kir channels in the perikarya of petrosal ganglion
cells may maintain resting membrane potentials.

Hibino et al. (1999) reported that Kir4.1
immunoreactivity were localized in the satellite cells of
the cochlear ganglia, vestibular ganglia, trigeminal
ganglia, and superior cervical ganglia, but not in those
of the dorsal root ganglia and myenteric ganglia. Based
on the immunoreactivity for Kir4.1 in the present study, the
characteristics of satellite cells in the petrosal ganglion
may be similar to those in other viscerosensory and
sympathetic ganglia. Kir4.1 in the satellite cells may in
fact regulate extracellular K\(^+\) concentration in
combination with Kir1.1 and Kir5.1.

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