CORRIGENDUM

Potassium Channels as a Potential Target Spot for Drugs

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Corrigendum to: Djokic V, Novakovic R. Potassium Channels as a Potential Target Spot for Drugs. London: IntechOpen; 2020. DOI: 10.5772/intechopen.92176.

The publisher is correcting [1] following an authors’ request.

An error occurred with regard to the list of authors. By mistake the name of the author Vladimir Djokic was omitted from the list of authors.

Following correction have been made to the main text of [1]:

After the sentence “Ion channels play key roles in membrane potential generation and many cellular activities such as signal transduction, neurotransmitter release, muscle contraction, hormone secretion, volume regulation, growth, motility, and apoptosis” following reference has been added: Kim JB. Channelopathies. Korean J Pediatr 2014;57:1-18.

After the sentence “It is widely known that potassium channels (K channels) are transmembrane proteins that allow the flow of potassium across the membrane to regulate ion homeostasis, cell proliferation, migration, cell volume, and specific processes such as muscular contraction.” following reference has been added: Ashcroft FM. Ion Channels and Disease. Academic Press 2000.

Sentence “The biophysical properties, physiological regulation, and pharmacological properties of Kv channels are dependent on the combination of α subunits. The combination on four α subunits may be homo- or heteromultimers [10].” has been replaced with a following sentence: “The biophysical properties, physiological regulation, and pharmacological properties of Kv channels are dependent on the combination of α subunits. The combination on four α subunits may be homo- or heteromultimers.”.

Sentence: “Even more complex to these heteromultimers is their interaction with smaller accessory proteins including β subunits, KChIP, KcHAP, and minK proteins, miRP peptide, and others [11].” has been replaced with a following sentence: “Even more complex to these heteromultimers is their interaction with smaller accessory proteins including β subunits, KChIP, KcHAP, and minK proteins, miRP peptide, and others [10, 11].”

Sentence “These differences in channel sensitivity attribute to the different expressions of Kv channel subtypes, the use of different animal species in studies, differences in sex, cell isolation techniques, and imaging conditions.” has been replaced with a following sentence: “These differences in channel sensitivity attribute to the different expressions of Kv channel subtypes, the use of different animal species in studies, differences in sex, cell isolation techniques, and imaging conditions [10, 18].”

After the sentence “They play a role in the regulation of smooth muscle tone, and change in the gene encoding them leads to a decrease in the activity of functional KCa1.1 channels lead to constrictions [23, 24].” following reference has been added: Zhu Y, Ye P, Chen SL, Zhang DM. Functional regulation of large conductance Ca2+-activated K+ channels in vascular diseases. Metabolism: Clinical and Experimental 2018;83:75-80.

Paragraph “The pharmacology of KCa1.1 channels spread from nonspecific blockade with TEA and alkaloids, like paxillin, to more effective specific inhibitors scorpion toxins, such as iberiotoxin and charybdotoxin. Although these
compounds do not have pure therapeutic potential, they are very useful tools for studying the function of these channels. Several small-molecule KCa1.1 channel openers have been detected for both native and cloned channels. For example, benzimidazole NS-1619 activates KCa1.1 channels, but its functional effects also include inhibition of Ca2+ currents and Kv channels [20]. And many other substances can modulate the activity of KCa1.1 channels. Although these compounds do not have pure therapeutic potential, they are very useful tools for studying the function of these channels. Several small-molecule KCa1.1 channel openers have been detected for both native and cloned channels. For example, benzimidazole NS-1619 activates KCa1.1 channels, but its functional effects also include inhibition of Ca2+ currents and Kv channels. And many other substances can modulate the activity of KCa1.1 channels, such as estrogen, reactive oxygen species and ethanol [20, 27, 29].

In the sentence “Estrogen binds at a high micromolar concentration to the KCa1.1 channel, primarily to the β1 subunit, and directly regulates channel expression as well as its function. Heme proteins and reactive oxygen species are proven modulators of these channels since both inhibit their activity in vascular smooth muscle cells by acting on the α subunit. Ethanol also inhibits KCa1.1 activity in vascular smooth muscle cells causing vasoconstriction to increase [27]” have been removed.

In the sentence “On the other hand, activation of protein kinase C (PKC) and vasoconstrictor induced increases in intracellular Ca2+ caused by noradrenaline, vasopressin, endothelin, and angiotensin II were accompanied by inhibition of the KATP channel [35, 36]” following reference have been added: Ko EA, Han J, Jung ID, Park WS. Physiological roles of K+ channels in vascular smooth muscle cells. J Smooth Muscle Res. 2008;44:65–81. doi: 10.1540/jsmr.44.65.

In the Table 1, column 2 in the Table 1 following changes have been made:
Word dendrotoksin has been replaced with dendrotoxin.
Word margatoksin has been replaced with margatoxin.
Word tetraetilamonijum has been replaced with tetraethylammonium.
Word noksiustoksin has been replaced with noxiustoxin.
Word maurotoksin has been replaced with maurotoxin.
Word koreolid has been replaced with correolide.
Word fampridin has been replaced with fampridine.
Word linopirdin has been replaced with linopiridine.
Word astemizol has been replaced with astemizole.
Word dizopiramid has been replaced with disopyramide.
Word retigabin has been replaced with retigabine.

Changes have been made in online and print versions of the chapter.
This corrigendum is published in agreement with the authors.
The publisher regrets any inconvenience this might have caused to the readership.

References

[1] Djokic V, Novakovic R. Potassium Channels as a Potential Target Spot for Drugs. London: IntechOpen; 2020. DOI: 10.5772/intechopen.92176.