Modulation of TASK-2 Potassium Channel by Heterotrimeric G Protein Subunits.

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Introduction and objectives: TASK-2 potassium channel is a member of the KCNK superfamily. This superfamily are remarkable in that they posses two P-domains and four a-helices in each subunit, form dimers, and are mostly open at resting potencial. In human and mouse, TASK-2 is expressed predominantly in the proximal renal tubule. This channel, participates in ion fluxes necessary for cell volume regulation and bicarbonate reabsorption in Kidney. There is little background on molecular mechanisms that may govern these processes. Numerous studies have described the direct effect of heterotrimeric G protein subunits (Ga and ßγ) on ion channels. Preliminary evidence from our laboratory indicates that TASK-2 expressed in HEK cells, it would be modulated by a ßγ subunit of G protein. So our main goal is to identify the mechanism by which TASK-2 is regulated by G protein subunits. **Results and conclusions:** *Patch-clamp* studies revealed inhibition of TASK-2 current by GTP-γ- S and ßγ purified protein, but not by GDP-ß-S. To determine the mechanism by which TASK-2 would be regulated by G protein subunits and to identify which subtype of subunit participates in this process *Western blot* assay were performed in HEK cells and primary cultures of mouse proximal convoluted tubules (PCT) with antibody anti-ß subunit of G proteins. HEK cells expressing the ß1, ß2, ß3 and ß4 subunits of the first two being the most abundant, whereas PCT cells expressing only ß1 and ß2 subunits. Considering the abundance of these proteins in both cell types, we hypothesize that one of them is involved in modulating the activity of TASK-2.

Key words: TASK-2 potassium channel, Heterotrimeric G Protein Subunit, Kidney.
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