TASK CHANNELS IN CAROTID BODY GLOMUS CELLS IN THE RAT MODEL OF CHRONIC INTERMITTENT HYPOXIA

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Acute ventilatory response to hypoxia is generated by carotid body glomus cells. The response is enhanced in chronic intermittent hypoxia (CIH) resembling nocturnal apneic episodes, but the mechanisms of the enhancement are unclear. TASK channels shape the background membrane K^+ current. The TASK1–TASK3 heterodimer most effectively inhibits outward K^+ current leading to cell depolarization. These channels are assembled in the endoplasmic reticulum (ER) from where they traffic to the cell surface. p11 protein serves as a retention factor that holds channels in ER. Here we assessed the presence and trafficking of p11 and TASK channels in glomus cells in the rat model of CIH compared to chronic normoxia, using immunohistochemistry. We failed to find differences in cytosolic localization and intensity of p11 and TASK immunoreactive materials in CIH. Thus, the reason for the readjustment of the carotid body’s output power controlling hypoxic sensitivity in CIH does not involve the TASK channels’ function.