Immunocytochemical evidence for co-expression of Type III IP3 receptor with signaling components of bitter taste transduction

ABSTRACT
Our data indicates that IP3R3 is the primary type of taste receptor expressed in taste cells and plays a crucial role in transducing bitter taste.

INTRODUCTION
Astrocytes are the most abundant type of cell in the central nervous system, and as such they are closely involved in modulating the activity of neuronal components and are involved with many important physio-pathological brain events including synthesis and secretion of (Neuro)trophic growth factor. Furthermore, it has been established that neurotrophin-mediated signalling may not be the exclusive factor influencing astrocyte-neuron interactions. The formation of distinct intercellular connections (gap junctions) between two cell populations, which facilitate the exchange of chemical signals (ions, small metabolites) from one cell to another and facilitate communication with adjacent neurons, may provide an additional, rapid and unique method for astrocytes to communicate with each other and interact with neighboring neurons. The modulation of astrocyte functions in mammalian symbiosis is often achieved through the use of extracellular physiological agonists, which can increase intracellular Ca2+ concentrations via voltage-dependent channels or controlled release from internal stores. The coordination of astroglial function is believed to be dependent on the transmission of Ca2+ waves through gjs. The origin and dissemination of Ca2+ waves were initially observed in brain-derived cell populations during culture, but this phenomenon has since been confirmed in more complex systems, including brain slice preparations and living rat brain. Despite the significant number of contributions published in the last decade, the mechanism responsible for Ca2+ waves' origin and propagation is still unclear. Furthermore, there is limited data available from in vivo experiments, particularly those on human astrocytes. Around ten years ago, an artificial glioblastic cell line was formed using human (GL15) cell lines. By studying the cell karyotype and immunohistochemical and cytogenetic demonstration of glial fibrillary acidic protein (GFAP) expression, they were able to characterize GL15 cells as an astrogliAL-like cell line by characterising them as such. In addition, the GL15 cellular population contained other astroglial biochemical traits that were found to be unique to astrolites, such as glutamine synthetase expression, taurine transport, transforming growth factor receptor expression and interleukin-induced cytotoxicity. The data from the previous studies indicate that astroglial phenotypes exist, but there is no conclusive evidence available to date regarding the essential physiological features of the GL15 cells related to their differentiation. As resolue to investigate the mechanism(s) of cell communication within astrocytes, we decided to focus on one of our most important concerns in physiology. GL15 cells are considered an ideal in vitro model of astrocytes due to their ability to communicate with other living cells through membrane surface receptor-operated systems and/or gjs. We define the features of this model by analyzing some morphological aspects, the mechanism of [Ca2+]i increase induced by different extracellular physiological agonists, and the expression and functional capacity of the gjs system in relation to the differentiative pathway.

CONCLUSION

The 1 subunit has the ability to selectively modulate the pore function of the DNasehirgic Receptor Plasticity Factor (DHPR) without any modification of charge movements or voltage dependence of Ca2+ transients, which is not the case for other DHRP subdomains. The charge movement protocol probably did not account for gating currents that open the Ca2+ channel, which are relatively small and only resolve depolarizations lasting >200 ms compared to the 25 mms used here (Fig. 4). The potential alteration in the voltage-dependent behavior of charges recruited through long depolarizations would be consistent with the Ca2+ current inactivation and necessitates a resolution that remains unclear in 1 null myotubes. However, the protocol measures the immobilization-resistant charge movements that are typically necessary for skeletal-type EC coupling. According to a recent report, the activation of SR Ca2+ release is not likely to be dependent on the 1 subunit. However, changes in voltage dependence below the resolution limit and effects on charge movement and Ca 2+release inactivation cannot be completely excluded. The 1 knockout mice offer a unique opportunity to learn about the protein's function in myotubes in great detail.