Developmental expression of survivin during embryonic submandibular salivary gland development

ABSTRACT
Survivin is recognized to have a beneficial and anti-apoptotic effect. Our study demonstrates the existence of nuclear-localized survivin protein in presumptive ductal and proacinar lumen-bounding cells, which suggests that survin may play a crucial role in embryonic SMG epithelial cell survival, given that it is thought to mediate survior's localization into the nucleus for both entry to the cell cycle and inhibition of inflammatory processes.

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

We have demonstrated that polyclonal anti-MCM2 antibodies offer dependable staining results consistent with fixed tissues without the need to search for specific antigens. The interpretation of the results is simple because there is a significant difference between normal bronchoepithelium and premalignant lesions. MCM2 is a simple marker that can be used to assess the progression and regression of morphologically abnormal lesions in primary lung cancer prevention studies and early detection of lung carcinoma in screening studies.