Apical accumulation of MARCKS in neural plate cells during neurulation in the chick embryo

ABSTRACT
The accumulation of MARCKS at the apical region of neural plate and lens placode cells during bending processes is observed. This symmetric subcellular distribution starts before the beginning of curved neural plates. These results indicate potential downstream regulatory actions of these proteins on specific functions of the actin subacical meshwork.

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

According to earlier investigations on FMR proteins, the FXR1 protein is primarily cytoplasmic, but undifferentiated cells in various tissues of human foetuses and mouse embryonic stem cells occasionally exhibit nuclear localization. By examining the model system of C2C4 myoblasts that can be manipulated in vitro to differentiate into mytubes, we provide strong evidence that FXR1P isoforms are actually stored in the nucleus as well as in other forms. We propose that this nucligen-cytoplasmic partitioning of FYR1,P may be controlled by factors regulating cell differentiation. Moreover, we also postulate that FMRP isoforms, which have been kept secreted until now because of the scarcity of available antibodies, may play a nuclear role in mRNA maturation during specific phases of neuronal differentiation and plasticity. To sum up, the model system presented here is a potent resource for ongoing research on the structure-function relationships among various FMR family members, as the role of FXR1P and FYR2P in Fragile X Mental Retardation is yet to be determined.