Data in Brief

Transcriptomic analyses of primary astrocytes under TNFα treatment

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Abstract

Astrocytes, the most abundant glial cell population in the central nervous system, have important functional roles in the brain as blood brain barrier maintenance, synaptic transmission or intercellular communications [1,2]. Numerous studies suggested that astrocytes exhibit a functional and morphological high degree of plasticity. For example, following any brain injury, astrocytes become reactive and hypertrophic. This phenomenon, also called reactive gliosis, is characterized by a set of progressive gene expression and cellular changes [3]. Interestingly, in this context, astrocytes can re-acquire neurogenic properties. It has been shown that astrocytes can undergo dedifferentiation upon injury and inflammation, and may re-acquire the potentiality of neural progenitors [4,5,6,7].

To assess the effect of inflammation on astrocytes, primary mouse astrocytes were treated with tumor necrosis factor α (TNFα), one of the main pro-inflammatory cytokines. The strength of this study is that pure primary astrocytes were used. As microglia are highly reactive immune cells, we used a magnetic cell sorting separation (MACS) method to further obtain highly pure astrocyte cultures devoid of microglia.

Here, we provide details of the microarray data, which have been deposited in the Gene Expression Omnibus (GEO) under the series accession number GSE73022. The analysis and interpretation of these data are included in Gabel et al. (2015). Analysis of gene expression indicated that the NFκB pathway-associated genes were induced after a TNFα treatment. We have shown that primary astrocytes devoid of microglia can respond to a TNFα treatment with the re-expression of genes implicated in the glial cell development.

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1. Direct link to deposited data

Deposited data can be found at: http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE73022.

2. Experimental design, materials and methods

2.1. Cell culture and experimental design

Primary mouse astrocytes cultures were prepared from newborn C57BL/6JOlHsd mice brains as previously described [8]. After removing meninges and large blood vessels, brains were minced in phosphate-buffered saline solution by mechanical dissociation. Cells were cultivated in Dulbecco’s Modified Eagle Medium supplemented with 10% fetal bovine serum, 100 U/mL penicillin and 100 μg/mL streptomycin at 37 °C in a humidified atmosphere.
Microarray gene expression data was normalized using the GC-RMA procedure with default parameters for background correction, quantile normalization, and probe replicate summarization [10]. Differentially expressed genes between control and TNFα conditions were then determined using the empirical Bayes moderated t-statistic (eBayes) [11]. P-value significance scores for these genes were adjusted for multiple hypotheses testing according to the Benjamini-Hochberg procedure [12].

A heat map and dendrogram cluster visualization for the top 150 most significant known genes (Fig. 1) was obtained using standard hierarchical average linkage clustering with a Euclidean distance metric.

A volcano plot for the analysis of differential gene expression between TNFα and control samples was obtained. For each transcript, the negative decadic logarithm of the adjusted p-value significance score was plotted against the logarithm of the fold change. Several genes (green dots) are significantly altered (adjusted p < 0.05) and display an absolute log fold change above 1 in expression (Fig. 2).

Alterations in known cellular pathways and processes were identified and visualized by applying the MetaCore™ GeneGo software onto the differential expression statistics obtained from the eBayes analysis [11]. The genes were pre-filtered using a significance threshold (adjusted p-value < 0.05) before applying the default GeneGO pathway analysis. Pathway analysis with GeneGO revealed that pathways related to glial differentiation, immune response and apoptosis were modulated (Fig. 3).

3. Conclusion

Herein we describe the transcriptional analysis of primary astrocytes following a TNFα exposure. These expression data could be useful to describe the effect of the NFκB activation on primary astrocyte cultures devoid of microglia. Taking advantage of the MACS technology, in contrast to the main studies reported in the literature, we were able to characterize pure populations of astrocytes under inflammatory conditions.

We show that TNFα increases the expression of genes associated with the NFκB pathway and induces the re-expression of genes implicated in glial developmental processes.

These data highlight the importance of the NFκB pathway during the conversion of astrocytes into reactive cells and, particularly, its active role in the dedifferentiation process [13].

Conflict of interest

The authors declare that there are no conflicts of interests.

Acknowledgments

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Fig. 3. Cellular pathways enriched in significantly differentially expressed genes between TNF and control sample. These pathways were identified using the GeneGO pathway analysis software.
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