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Whole transcriptome analysis reveals that Zika virus halts cell cycle progression and disrupts neuronal differentiation in human neurospheres

João Lídio da Silva Gonçalves Vianez Júnior[1], Janaina Mota de Vasconcelos[1], Patricia P Garcez[2, 4], Juliana Minardi Nascimento[4, 3], Rodrigo Madeiro da Costa[4], Rodrigo Delvecchio[2], Sandro Patroca da Silva[1], Pablo Trindade[4], Erick Correia Loiola[4], Luiza M Higa[2], Juliana S Cassoli[3], Gabriela Vitória[4], Patricia Sequeira[5], Jaroslaw Sochacki[4], Renato S Aguiar[2], Hellen Thais Fuzii[6], Ana M Bispo de Filippis[5], Daniel Martins-de-Souza[3], Amilcar Tanuri[2] and Stevens K Rehen[2, 4]

[1]Evandro Chagas Institute, Brazil
[2]Federal University of Rio de Janeiro, Brazil
[3]University of Campinas, Brazil
[4]D'Or Institute for Research and Education, Brazil
[5]FIOCRUZ, Brazil
[6]Federal University of Pará, Brazil

Brazil is facing an unprecedented growth in the number of microcephaly cases in babies. This phenomenon coincided with the recent Zika virus (ZIKV) outbreak in this country. Although the Brazilian Ministry of Health was quick to recognize that ZIKV was probably the cause of microcephaly in newborns, the underlying mechanisms leading to the development of this pathology have not been established. To tackle this problem at the molecular level, we employed whole transcriptome sequencing of human neurospheres derived from neural stem cells exposed to ZIKV isolated in Brazil (Asian genotype). Differential gene expression analysis of control (MOCK) and ZIKV infected neurospheres generated a list of 26 down-regulated and 64 up-regulated genes. Among the up-regulated detected genes, the cyclin-dependent kinase inhibitor 1A (CDKN1A) and the glial fibrillary acidic protein gene (*GFAP*) were found. CDKN1A prevents the activation of the cyclin E/CDK2 complex, acting as a regulator of cell cycle progression during G1 and GFAP is a known marker of astrocytes. We also observed a decrease in the expression of the neurogenic differentiation 1 gene (*NEUROD1*) which is directly involved in the neurogenic program. Those findings suggest that ZIKV infection induces cell cycle arrest and inhibits the neuronal differentiation, resulting not only in the reduction of the size but in a deeper disruption of the normal development of the human brain.

vianez.iec@gmail.com

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