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Viral infection affects important cells' stress response

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Viral infection disrupts the normal response of mammalian cells to outside deleterious forces, cleaving and inactivating a protein called G3BP that helps drive the formation of stress granules, which shelter the messenger RNAs that carry the code for protein formation, said researchers from Baylor College of Medicine in Houston.

Only recently have scientists begun to understand the role of stress granules, said Dr. Richard Lloyd, associate professor of molecular virology and microbiology at BCM, and senior author of the report that appears today in the journal Cell Host and Microbe. The stress granules are formed when a cell is subjected to several kinds of stress, such as nutrient deprivation or virus infection.

“When the cell suffers a major insult, it stops expanding. The business of protein synthesis (in which messenger RNA or mRNA’s genetic code gets translated into proteins that carry out cellular activities) is arrested. The messenger RNA goes into storage until conditions improve for the cells,” he said. “Stress granules are a major storage site for the mRNA.”

However, in poliovirus infection (used in the laboratory because it is a prototype for many kinds of viruses), the stress granules are formed early but as the infection continues, the stress granules disperse.

Lloyd and his colleagues found that the poliovirus infection actually cuts or cleaves G3BP, a protein critical in the formation of the stress granules.

“The cells respond to the viral infection, and then virus is shutting that response off,” said Lloyd. In effect, he said, this type of cell response helps prevent the virus from translating its mRNA into virus proteins and killing the cells.

Other viruses may affect other proteins important in this type of stress response, said Lloyd. “Poliovirus has evolved to target G3BP,” he said.

When he and his colleagues mutated G3BP to make it resistant to being cut or cleaved, they found that stress granules could be formed during virus infection and that this inhibited virus growth in the cells.

“With the cleavage resistant form, the cells can continue to make stress granules, and this interferes with virus reproduction” he said.

Source: Baylor College of Medicine

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