BRAC UNIVERSITY
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PHARMA HIGHLIGHTS

ISSUE 38 | NOVEMBER 2017

GlaxoSmithKline Wins FDA Approval to Develop 'Breakthrough' Blood Cancer Drug

The renowned pharmaceutical company, GlaxoSmithKline, received FDA approval for fast-track development of a new experimental blood cancer drug, on Thursday, November 2, 2017. The drug is currently labeled as GSK2857916.

The FDA gave the drug "breakthrough therapy designation," meaning regulatory review will be faster than it typically is for other drugs. The treatment offers monotherapy in patients with multiple myeloma who have failed at least three prior lines of therapy and are refractory to a proteasome inhibitor and an immunomodulatory agent.

The FDA's decision follows a similar action from the European Medicines Agency, which approved development for GSK2857916 last month. Both approvals follow promising Phase I clinical trial results, which are scheduled to be announced in December. GSK sold its marketed cancer drugs to industry peer Novartis AG (NVS) in 2015, but the company continues to fund early-stage research. Oncology remains an option to add to GSK's pharmaceuticals business.

GSK plans to rapidly advance clinical trials with this promising therapy, alone and in combination with other therapies.

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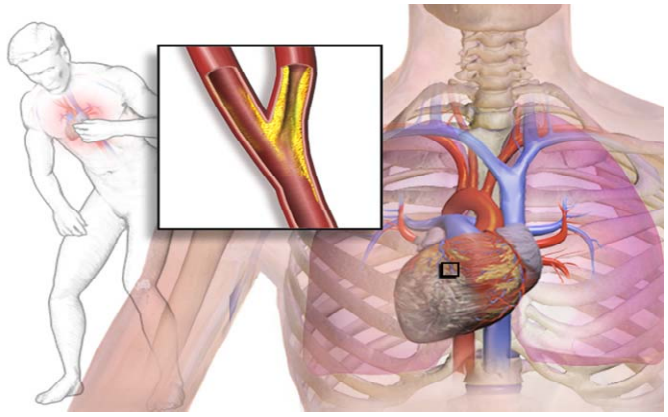
Source: The street.com

Misguided Immune Cells Treat Heart Attacks As Viral Infections

In a new study published in Nature Medicine, scientists aimed to explain why an antiviral immune response launches in the aftermath of a heart attack. Scientists found that the interferon response is turned on after a heart attack even with no viruses detected by the immune system. But instead of viral molecules triggering the defensive response, dying cell DNA initiates it.

While immune cells in the heart tissue following a heart attack can be helpful, removing “cellular debris” from dead and dying cells and helping with the beginning of the repair process, too many immune cells can promote harmful inflammation and further dysfunction in an already-damaged heart.

Researchers realized it was the interferon response that was bringing numerous immune cells to the heart during their study using single-cell RNA sequence, a technology that allowed for mapping the transcriptomes of nearly four thousand individual immune cells. A cell’s transcriptome is a collection of all of the transcribed DNA



in a cell; DNA has to be transcribed before the cellular machinery can produce proteins.

After a heart attack, DNA from dying cells acts like a virus in that it “activates an ancient antiviral program” in immune cells, the type I interferon response. Scientists refer to the cells that misguidedly activate this response “interferon inducible cells” (IFNICs). Their study of cells’ transcriptomes also lead to the novel identification of a unique type of cardiac macrophage never before seen by scientists.

Source: National Human Genome Research Institute, University of California - San Diego

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Blood Leaking Fibrinogen Is the Cause of Multiple Neurological Diseases

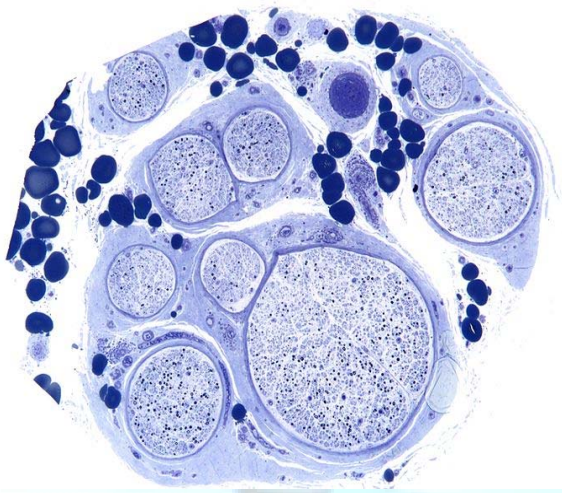
The body has built-in mechanisms to handle brain injury, but when something disrupts the repair process, neurological diseases like multiple sclerosis (MS), stroke, and Alzheimer's develop. With a new study published in the journal Neuron, scientists from the Gladstone Institutes discovered the role of a blood protein in blocking repair processes in the brain.

Without the myelin sheath, nerve fibers experience a plethora of problems centered on impaired signal transmission: cognition, sensation, movement, and more. When Gladstone researchers realized that it was the blood-clotting protein fibrinogen that was causing the repair problems, they set out to revolutionize the way scientists approach treating neurological disorders.

Fibrinogen, made in the liver, is one of 13 coagulation factors that promote blood clotting. In the right context, blood clotting can save lives by preventing excessive bleeding. In the wrong context, blood clotting can prevent the flow of blood, carrying oxygen and nutrients, to important bodily organs like the brain and heart.

Source: Gladstone Institutes, Healthline

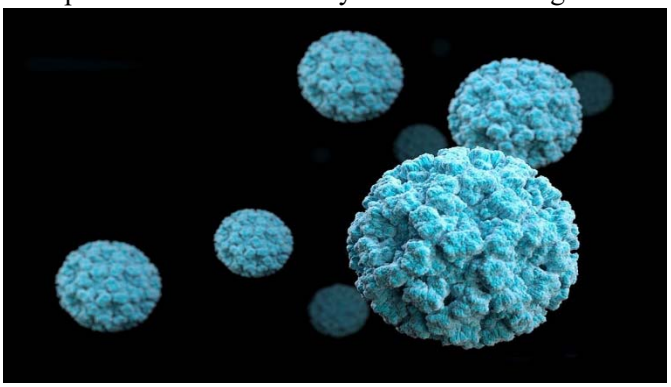
They found that fibrinogen "leaks" into the central nervous system and disrupts the way nerve cells produce myelin, preventing repair. Normally, adult stem cells travel to damaged nerve sites and transform into new myelin-producing cells. However, fibrinogen prevents adult stem cells from making the reparative transition into myelin-producing cells. Now that they know what's stopping repair, scientists are hopeful that they can find a way to better approach treating neurological diseases like MS and Alzheimer's.



Norovirus Sneaks Around in the Gut to Avoid Detection

Norovirus is infamous for its invasion of cruise ships, daycares, and other places where a viral infection is particularly devastating. In a new study from the University of Pennsylvania School of Medicine, scientists strip away some of the mystery surrounding this virus, revealing how it manages to avoid the immune system and persist for months at a time.

Norovirus is extremely contagious, and with so my strains of the virus in the world, you can become infected multiple times. Scientists say it is the "leading cause of nonbacterial gastroenteritis in the world," a condition people often refer to as the "stomach flu."



In the new study, published in the journal Immunity, researchers used a mouse model of norovirus infection, watching how the virus slips through the cracks, avoiding the immune attack even when the mice had been immunized against the virus. It turns out the norovirus takes cover in a rare gut cell that has virtually no contact with the immune system. Researchers say this may explain why current vaccines don't work against norovirus, even when researchers see an antibody response after administration.

Norovirus hides in rare gut cells, a type of cell representing an extremely small minority of the billions of other cell types in the gut. These cells do not "talk" to T cells, so norovirus can replicate in peace. This finding means future vaccines would need to act fast, before norovirus moves to the gut to hide in cells. And now that researchers know where norovirus is hiding, they can develop drugs, potentially T cell-based, to go and find it.

Source: CDC, University of Pennsylvania School of Medicine