

Science

By The Students of New School International School of Georgia

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How do viruses work?

BY SABA AKHALADZE

In this article I would like to introduce you to virology[1]. In a nutshell, I will tell you how viruses are built, how they infect a cell and how we fight them off.

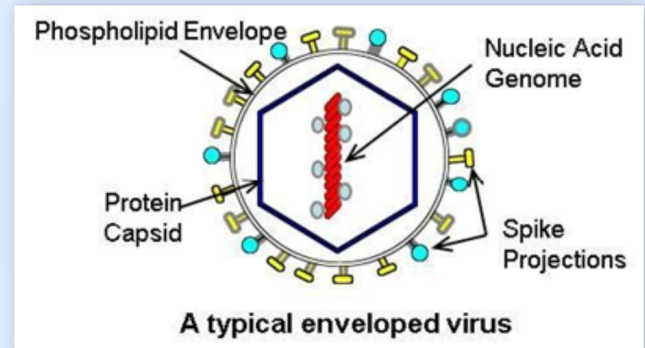
Considering today's situation, I think that this article will deliver you the knowledge you need to really understand what's happening today. I also have noticed that lack of knowledge in this subject has fed the chaos that the pandemic has caused us.

What is a virus?

To explain a virus in a sentence, it's a ball of genetic material which is encased in an envelope with protein keys attached. It infects[2] a cell and makes the cell make more viruses to infect other cells and to continue existing. Viruses can be in different shapes and sizes, depending on the host cell[3] (BD Editors).

Let's talk more about the structure of a simple virus. The outer layer is made from protein spikes (keys) and phospholipid bilayer[4]. The spikes help virus infect a host cell. Inside the membrane[5] there is a protein capsid (a shell made from proteins) in which we have the genome[6] of the virus, which

later is spilled in the host cell and new viruses are generated (Goulding).



(Goulding)

How does a virus infect a cell?

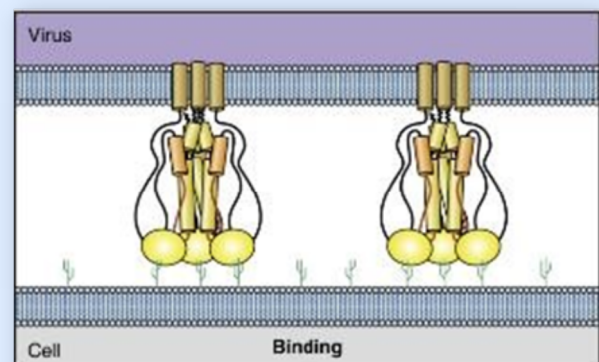
Once a virus gains access to a cell its job is to seize the function of it. After, it spills its genetic material and the cell starts to replicate the DNA, meaning the cell starts producing more viruses.

Ergo, the virus spreads (Cohen).

I will be discussing two methods for infection.

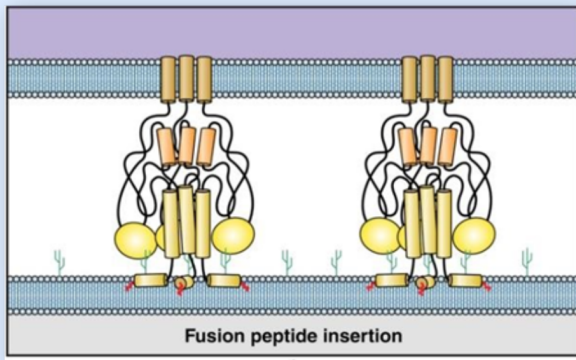
One method of infection is called fusion. It has four steps and in a nutshell the protein spikes fuse with the membrane of the host cell and make a pore[7] for the virus to enter.

The first step is binding, where the protein spikes bind with the membrane of a host cell (Cohen)



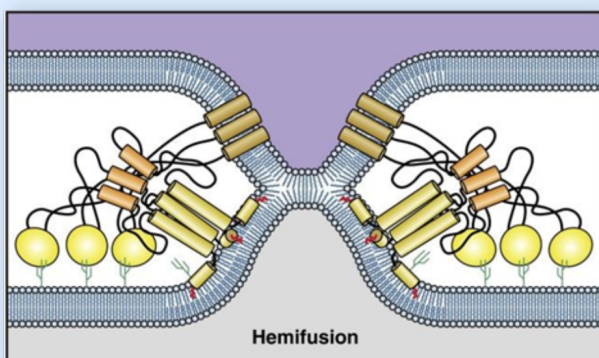
(Cohen)

The second step is the fusion peptide insertion, in this step the peptides[8] are inserted in the host cell membrane and the membrane and cytoskeleton[9] are controlled by them (Cohen).



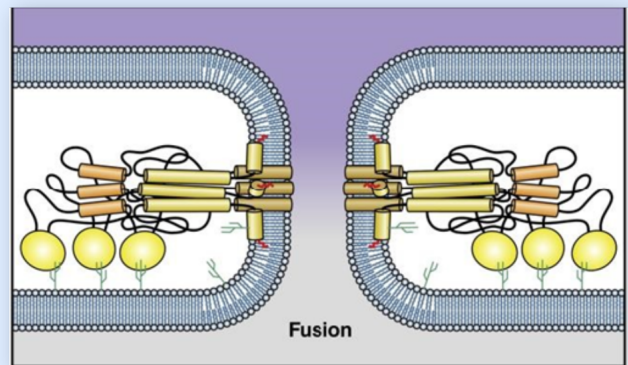
(Cohen)

The third step is called hemifusion. In this step the spike proteins start moving and changing the shape of both, host cell membrane and virus membrane to start fusing both cells together. The word hemi-fusion tells us the whole process, partially fused, where only a small part of the membrane is separating virus from the host cell (Cohen).



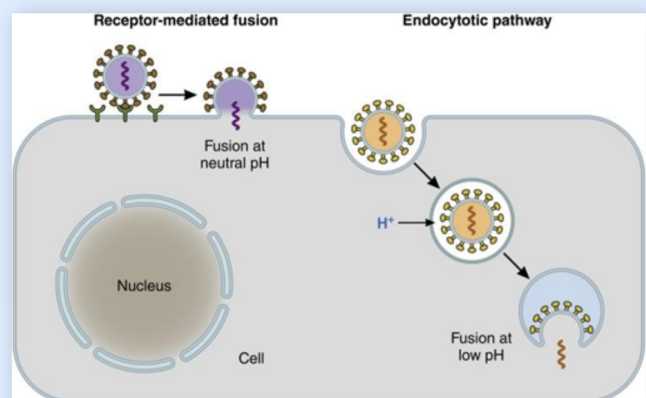
(Cohen)

The fourth and the final step is fusion. In this step the spikes finally made a pore through both membranes and those spikes have fused the virus and the host cell together. Now the virus can spill its genetic materials in the host cell and take over its function to produce more viruses (Cohen).



(Cohen)

The second method is called endocytic[10] pathway. In this infection method the virus uses its spikes to trick the host cell into thinking its something else, something it might need. This method has three steps. The first is receptor mediated fusion. Here the spikes get attached on the cell receptors[11] and tell the cell to open its membrane and let the virus in. After this its straightforward, the virus enters the cell, opens its membranes and spills the genetic material (Cohen).

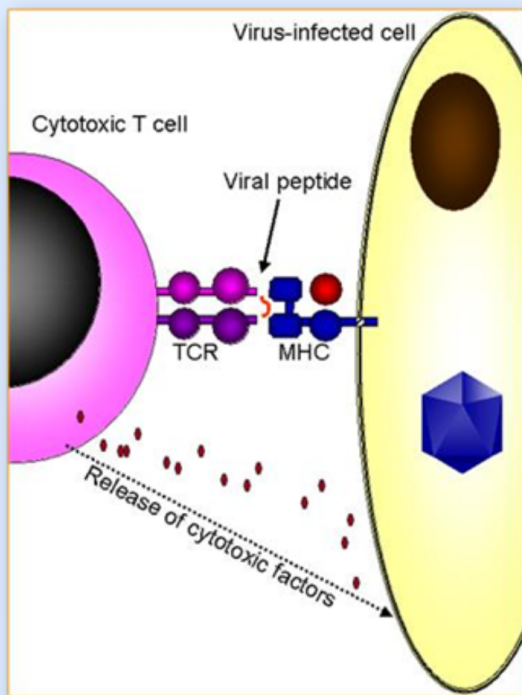


(Cohen)

How do we fight off viruses?

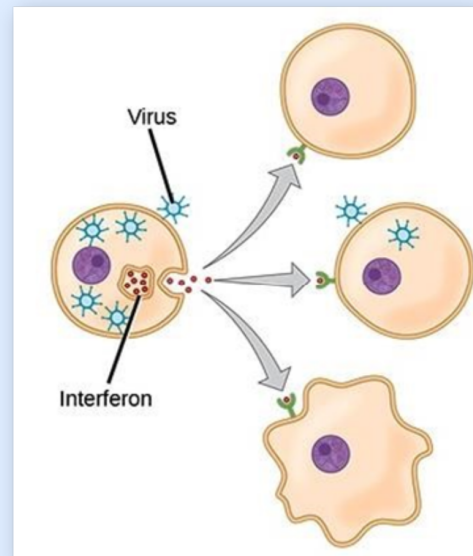
We have three mechanisms for fighting with viruses. Since viruses are very “clever” they can mutate[12] so that one of the mechanisms doesn’t work and the virus still survives.

The first method is via cytotoxic cells. When a host cell is infected, our immune system is unable to detect the virus, for this reason the host cells use a molecule called class I major histocompatibility complex proteins (MHC class I), this molecule basically exposes the viral proteins on the outside and then virus can be detected by T cells[13]. The T cells are equipped with T cell receptors that can detect MHC and they release cytotoxic factors[14] which kill the cell and virus with it (Laing).



(Laing)

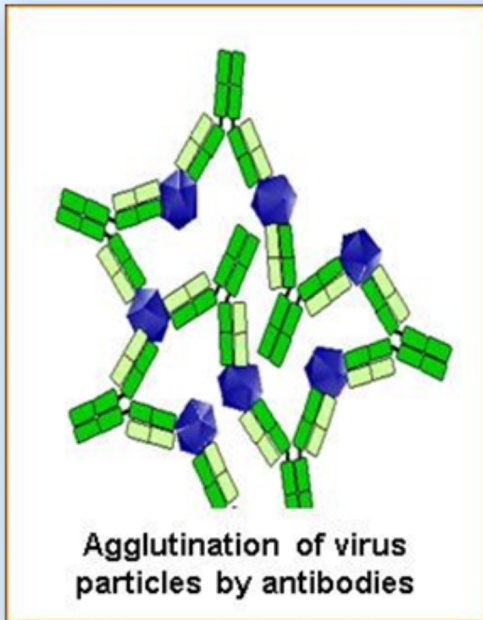
The second method for eliminating viruses is via interferons. Infected cells produce small proteins called interferons, these interferons prevent DNA replication of the virus by interfering with the ability of DNA replication. Interferons also act as signalling proteins since they signal other cells to increase MHC molecules so that TCR’s will spot infected cells more quickly and the virus won’t spread to other healthy cells (Laing).



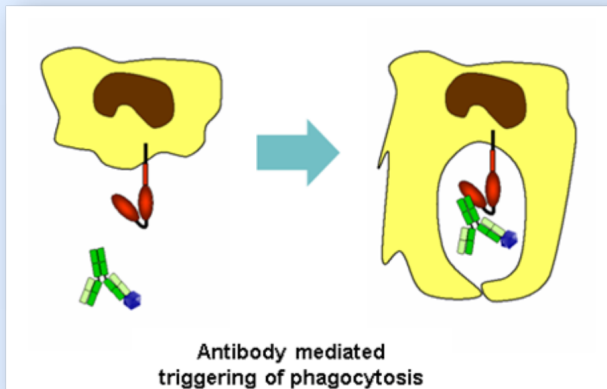
(Laing)

The third method for fighting viruses off is via antibodies. Using antibodies is removing viruses before they infect cells. Antibodies are proteins organised to demolish the virus via sticking to them and destroying them. Eliminating viruses by antibodies have three main steps. The first step is neutralising the virus, once virus is neutralised it can’t infect cells anymore. The second step is agglutination[15] of the viruses, because it’s easier for immune cells to work on viruses in a bunch rather than single virus.

After, these antibodies activate phagocytes[16] which literally digest deactivated viruses and eject them as a waste (Laing).



(Laing)



(Laing)

Scientific terms:

- 1 **virology**-subject that studies viruses,
 - 2 **infects**- viral invasion,
 - 3 **hostcell**-the cell that is infected/hosting the virus,
 - 4 **phospholipid bilayer**- two layers of fat molecules,
 - 5 **membrane**-thin layer separating two things (in this case separating virus insides from outside),
-

- 6 **genome**-complete set of DNA/RNA/genetical material,
- 7 **pore**-a small well,
- 8 **peptide**-part of a protein,
- 9 **cytoskeleton**-cellular skeleton,
- 10 **endocytic**- in this case, a cell ingesting something through the membrane,
- 11 **receptor**-in this case protein responsible for the recognition of viral proteins ,
- 12 **mutate**-change DNA sequence/change how the virus acts and appears,
- 13 **T cells**- part of immune system, produced in bone marrow, found in blood,
- 14 **cytotoxic factor**-substance that destroys the cell,
- 15 **agglutination**-bunching up, in this case deactivated viruses and antibodies,
- 16 **phagocytes**-white blood cells responsible for digestion of deactivated viruses/bacteria

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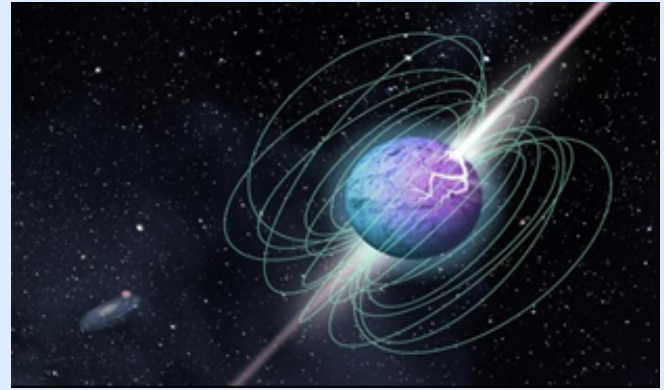
A galactic discovery; fast radio burst!

BY ANAMARIA SHAMUGIA

Astronomers were drawn to a vivid, short “burst of radio waves” erupting from the Milky Way's periphery in 2007. Since their discovery in 2007, a lot of research has gone into figuring out where the extragalactic, millisecond-duration fast radio bursts (FRBs) come from. According to the observations at several wavelengths, the first example of an FRB identified in our star system “originated from a magnetic neutron star” (Berkowitz). Since then, radio telescopes have identified dozens of more fast radio bursts, some of which appear to originate from the same spot.

Magnetars, a type of neutron star, are a prominent potential source of FRBs. Surface magnetic fields in magnetars exceed 10^{14} gauss, and the decay of these fields fuels a variety of high-energy processes. Empirical evidence, however, “has not directly linked an FRB to a magnetar” or other particular stellar body until now (Berkowitz).

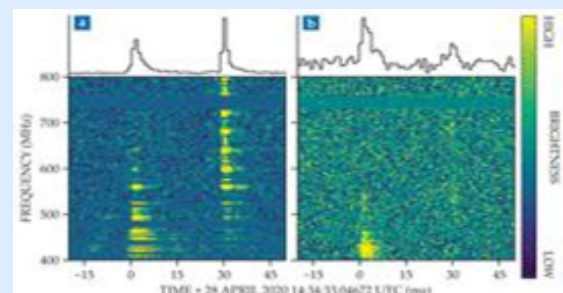
The strain generated by the high magnetic field develops until it is abruptly alleviated in a starquake, resulting in characteristic x-ray and gamma-ray bursts, which are shown in Picture 1.



Picture 1 (Berkowitz) A magnetar, as depicted by an artist

“Multiple gamma-ray bursts” from the same body were detected by Fermi Gamma-Ray Space Telescope. Repeating FRBs are thought to originate from extragalactic magnetars, according to one popular theory. Although, in order for the theory to succeed, several “extragalactic magnetars” should be able to produce “radio emissions” that are orders of magnitude greater than those found in the nearby galaxy.

The data presented in Picture 2 was included in a notice to the astronomical community by researchers, who reported the finding of a fast radio burst, named FRB 200428.

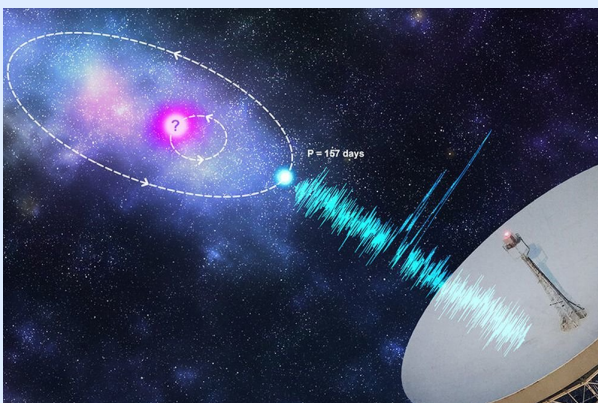


Picture 2 (Berkowitz) The data for FRB 200428

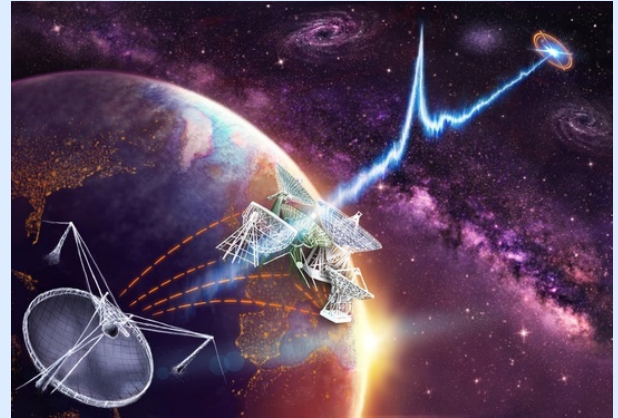
“The top panels depict the burst's total intensity in milliseconds, while the bottom panels depict the burst's intensity as a function of frequency” (Berkowitz).

Several satellite telescopes have been keeping a careful check on the same spot “because of the Swift telescope's gamma-ray alert”. (Berkowitz) “The three STARE2 telescopes” were built specifically to search for a rare but extraordinarily luminous FRB in the Milky Way.

“The nondetection is significant because it indicates that an FRB's association with a gamma-ray repeater is unique and rare,” says Bing Zhang. It's possible that this rarity is due to the complex “physical conditions required for FRB emission”, or because the beams of FRBs are weak and infrequently aim toward Earth (Berkowitz).



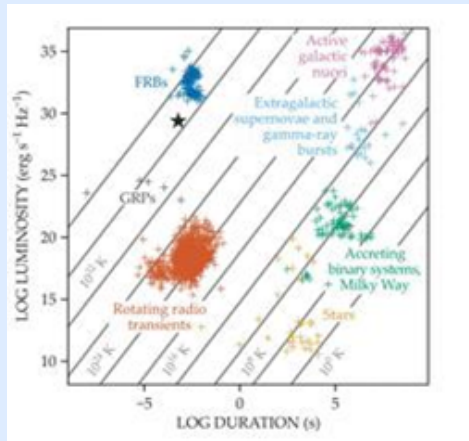
Picture 3 (Mickaliger) Orbital modulation model by artist's conception: FRB progenitor in an orbit with astronomical



Picture 4 (Futselaar) Fast radio burst, depicted by an artist

FRB 200428's finding suggests that “active magnetars” can create FRBs strong enough to be detected at “extragalactic distances”. Despite the fact that the measured signal was a factor smaller than other extragalactic FRBs when adjusted for distance, the researchers assessed that if the wave had arrived from the same position as “other known FRBs”, it would still be visible. The specific mechanism by which a magnetar generates FRBs is however still unknown. According to one hypothesis, the starquake causes “magnetic field lines near the magnetar's surface” to be disrupted.

These disruptions cause accelerated particles to escape the magnetosphere, resulting in radio emissions (Berkowitz).



Picture 5 (Berkowitz) The energy and duration of the FRB compared to “radio emissions from other known astronomical objects”

Repeating fast radio bursts are most likely made in a variety of ways. It's also conceivable that catastrophic occurrences like merging neutron stars might produce uncommon FRB events. As a result, determining the particular signals of various “physical mechanisms from various astrophysical objects” will be critical to the following studies. Physician Adam Bochenek explains that they knew that “magnetars and neutron stars could produce coherent radio emission”, but, until the discovery of fast radio bursts, no one predicted magnetars to produce such powerful “radio emission” (Berkowitz).



Picture 6 (ESO/L. Calçada) A magnetar, as depicted by an artist

The Milky Way's FRB source could therefore provide answers to open-ended concerns like “how often the signals repeat over years or decades, how far and how energetic the bursts are, and whether similar pulses can be used to distinguish specific objects in other galaxies” (Berkowitz).

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New research reveals how the lungs immune cells develop after birth

BY DIANA KOVZANADZE

Our lungs are exposed to germs such as bacteria and viruses from the moment we take our first breath. We are protected from most illnesses at a young age because of immune cells in the lungs known as macrophages. The researchers from Karolinska Institutet, a renowned Swedish science-based university, reveal how lung macrophages evolve in a new study published in the Journal of Experimental Medicine; new insights that can assist to prevent organ damage and are significant for the continuous development of essential lung disease therapies.

Humans create lung macrophages from birth, when the lungs are initially expanded with breathing air. Despite the importance of lung macrophages in the immune system, it was previously unknown how they grow in people due to the difficulty of doing in-vivo investigations in humans.

The scholars, however, have now been able to directly investigate the growth of human macrophages in a living lung using a model. The researchers revealed that lung macrophages grow in two ways.

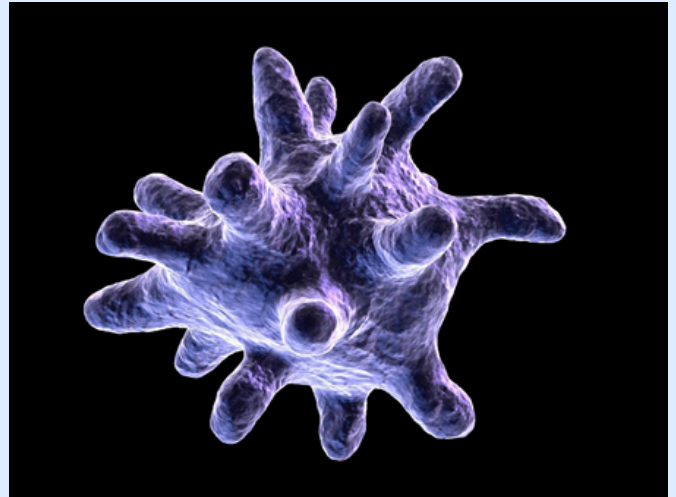
"In the first kind of development, lung macrophages arise from precursor cells that are already present in the fetus' liver," explains Tim Willinger, associate professor at Karolinska Institutet's Department of Medicine, who conducted the study. These precursor cells go through the circulation from the liver to the lungs where they can develop into mature macrophages. The second way involves the development of macrophages from monocytes, which are white blood cells that kill germs.

Similar gene expression but different functions

The researchers also looked at whether the origin of the lung macrophages influences their activity. They discovered that lung macrophages, regardless of origin, showed identical gene expression but performed diverse tasks. If macrophages are generated by precursor cells they are critical in early life in order to eliminate germs, however, if they come from the blood, they are responsible in defeating viral infections.

Limit lung damage and promote new treatments

The research is particularly important for human life as some lung macrophages might be used to produce tissue-protective macrophages, prevent organ damage, and enhance tissue regeneration in a damaged lung. These discoveries may potentially aid in the development of novel therapies for a variety of lung disorders.



Picture 1 : 3D macrophage

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Why Teapots Always Drip – Scientists Finally Explain the "Teapot Effect"

BY TEKLA KINTSURASHVILI

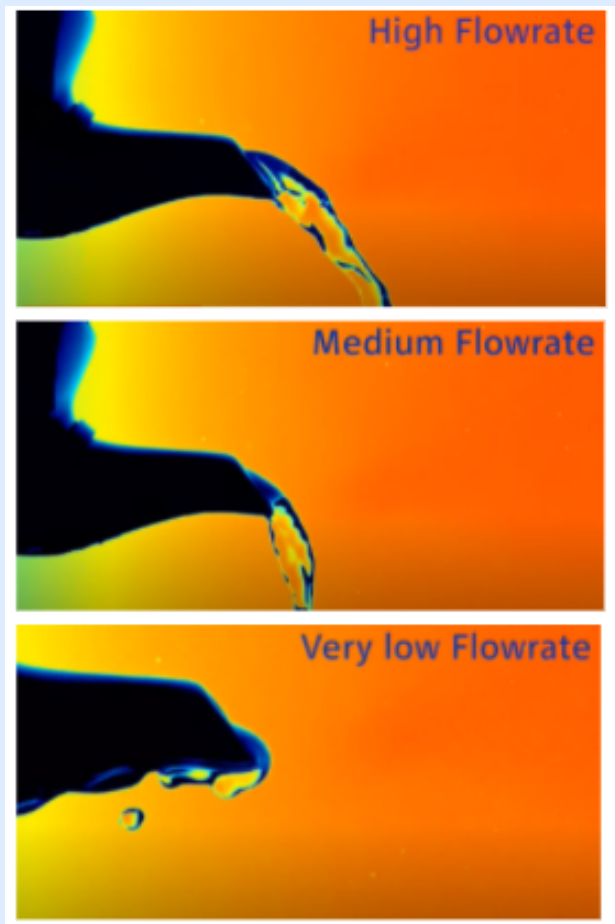
The so-called ‘Teapot effect’ has a long history of spoiling tablecloths. When liquid in the teapot is poured down in a cup at a relatively slower pace, as the liquid does not detach itself from the tip of the teapot, it fills the cup, but some of it still dribbles outside of the cup and teapot. After many decades of scientific study, through various experiments and theoretical work, the research team at TU Wien, has been able to completely describe the ‘teapot effect’: “An interplay of different forces keeps a tiny amount of liquid directly at the edge, and this is sufficient to redirect the flow of liquid under certain conditions”(Vienna University of Technology).

An effect with a long history

The ‘teapot effect’ was first outlined by Markus Reiner in 1956. Recently at TU Wien, research was led by Dr. Bernhard Scheichl, lecturer at the Institute of Fluid Mechanics and Heat Transfer and Key Scientist at the Austrian Center of

Excellence for Tribology, with help from the department of mathematics at the university college London.

According to Dr Scheichl, even though the effect seems not complex, it is rather difficult to explain within fluid mechanics. The beak of the teapot plays a very important role, as its underside always remains wet when forming a drop. Furthermore, the size of the drop depends on the speed of the liquid’s flow. If the speed is low and below critical threshold, liquid directs its flow under the edge, underside of the beak and liquid dribbles down. The pictures show different flow rates and its effect on the direction of water.



(Scitech Daily)

The research team has succeeded in completely explaining why the underside is always wet and why the drop forms this way. The basis behind the ‘teapot effect’ is interplay of inertia (state of rest), viscous force (a measure of a fluid's resistance to flow) and capillary action (the flow of a liquid in a narrow space without the assistance of external forces).

Inertial force is responsible for fluid maintaining its original direction. Meanwhile capillary forces slow liquid down at the edge of a beak, at a very specific angle between the wall of a teapot and liquid surface. Therefore, if the angle is small or if the material of the teapot is hydrophilic (attracted to water) then less liquid is detached from the teapot and the detachment process is effectively slowed down.

Tea in space

It is quite interesting that, strength of gravity is not decisive for the teapot effect. It only determines the direction of the liquid's flow. Therefore, the teapot effect can be seen on the moon base when drinking tea. However, the teapot effect does not work at a place with no gravity at all.

In September 2021, the research team published the theoretical calculations of the teapot effect and conducted an

experiment which was filmed by high speed cameras where water at different flow rates was poured down from the teapot. The experiment confirmed the theory behind the teapot effect.

When searching I was interested in some simple aspects of our daily lives that could be explained by complex science. It is truly fascinating how complex physics can be behind some simple task such as pouring water out of a teapot.

When making our teas or coffees I don't think we ever stop to wonder what goes beyond the process. With the help of this article we get to know the interplay of forces that goes on while pouring liquid from teapots.

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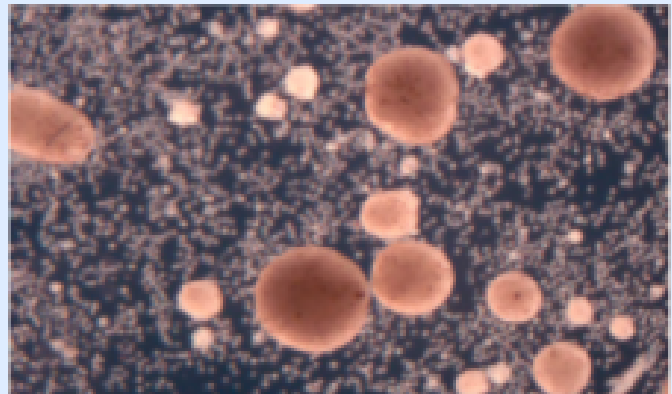
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RESEARCHERS CREATE SELF-REPLICATING LIVING ROBOTS

BY SIMONA FEJEROVA

Researchers from the University of Vermont, Tufts University, and Harvard University's Wyss Institute for Biologically Inspired Engineering constructed living robots in 2020 called Xenobots and have now discovered that these handmade organisms can swim around, find and gather single cells and assemble baby Xenobots.

Professor Michael Levin, director of the Allen Discovery Center at Tufts University explains that Xenobots were assembled using cells from the frog species *Xenopus laevis*. In a regular *Xenopus laevis*, these embryonic cells would naturally develop into skin – their job would be to protect the organism by keeping out pathogens as well as the redistribution of mucus. However, with Xenobots, these cells are given a chance to reimagine their multicellularity.



“Kriegman et al. show that clusters of cells, can similarly find and combine loose cells into clusters that look and move like they do” (“Researchers Create Self-Replicating Living Robots | Sci-News.com”)

In the image above we can observe a cluster of cells, which, when extracted from a developing organism, have the ability to discover and combine other loose cells into clusters such as themselves. These newly developed clusters look and move around exactly like the original ones. This proved that “this ability does not have to be specifically evolved or introduced by genetic manipulation” (Kriegman et al.) Professor Levin says that the full, unaltered frog genome is present in the Xenobot, but his team had no idea that

the cells could work collaboratively to gather and then compress separated cells into working self-copies. Dr. Douglas Blackiston, the senior scientist at Tufts University adds that this is an innovative way of replication very different from the way of any animal or plant known to man and it has never been observed before.

In the image below we can observe Xenobots gathering the cells around them into clusters as a form of replication.



“AI-designed organisms push loose stem cells into piles as they move through their environment” (Brown Joshua at Wyss Institute)

When left on its own, the Xenobot, which is made out of roughly 3000 cells, forms together in a sphere shape. However, as Dr. Sam Kriegman, a postdoctoral researcher at Tuft’s Allen Center and Harvard University’s Wyss Institute

for Biologically Inspired Engineering explains, the organism’s reproduction capabilities are limited when it is shaped like a sphere. In other words, the system can replicate once but dies after that. To solve this issue, the researchers used “an evolutionary algorithm to test billions of body shapes in simulation to find ones that allowed the cells to be more effective at the motion-based ‘kinematic replication’ reported in the new research” (“Researchers Create Self-Replicating Living Robots | Sci-News.com”) Kinematic replication has never before been seen with whole cells or organisms – only at the level of molecules. The group of researchers discovered a vast space inside of organisms that was previously unknown to science. Further research into this space could lead to even bigger discoveries concerning Xenobots.

The discoveries made by these researchers could also lead to advancements in regenerative medicine in general. Professor Levin explains that being able to control a group of cells and tell them what to do, which is the basis of regenerative medicine, could lead to solutions for aging, birth defects, traumatic injury, and even cancer. These issues are present because humans aren’t able to control or predict which groups of cells will form. Xenobots could be the solution we need.

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IT

LUKA TURMANIDZE
TORNICA KADEISHVILI
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TRANSLATORS

NINO BEROSHVILI
SERGI GETSADZE
NIA DOLIDZE
NUTSA MAMULASHVILI
TEKLA KINTSURASHVILI

DESIGNERS

BILURA ALAKBARLI
MARIAM MOTSIKULASHVILI
VATO BOSTOGANASHVILI
ELIZAVETI METREVELI
MARIYA DUBESHKO

WRITERS

SABA AKHALADZE
ANAMARIA SHAMUGIA
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