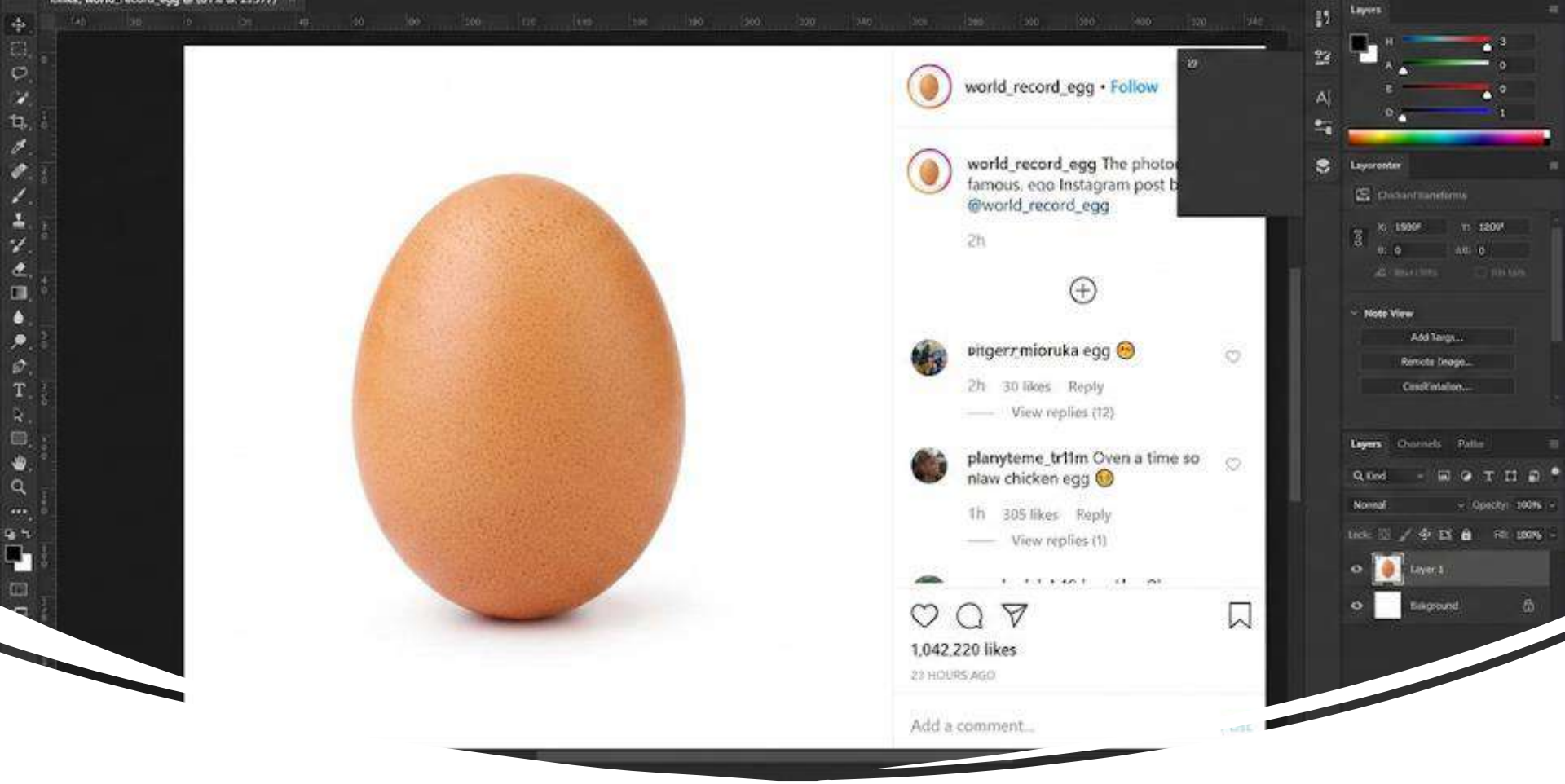
HER ŞEY VİRALDİR

esin şenol



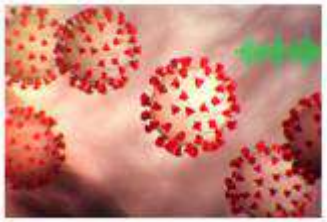
- "Her şey viraldir" demek, aslında internet çağının en büyük gerçeğine parmak basmak demek: Her nesne, her an ve her kelime doğru bağlamla buluştuğunda bir dünya olayına dönüşebilir.

VİRAL OLANLAR!!!

Videodaki doktorun koronavirüs nedeniyle yere yığıldığı iddiası

EZGİ TOPRAK ✖ 01/02/2020 ✖ 17:42

Koronavirüs ile ilgili seri iddialar içeren tweet zincirinde yer alan bir videonun, koronavirüs nedeniyle yere yığılan bir doktoru gösterdiği öne sürüldü. Ancak bu videodaki doktor hasta yakınlarının



Koronavirüsle ilgili yayılan ses kaydına ilişkin iddialar

EZGİ TOPRAK ✖ 30/01/2020 ✖ 00:00

Çin'in Wuhan kentinden yayılmaya başlayan koronavirüs salgınıyla ilgili kapalı mesajlaşma platformlarında yayılan bir ses kaydı, Teyit'e birçok farklı kanaldan ihbar olarak geldi. Ancak iddialar gerçeği yansıtmıyor.

Koronavirüs salgınının arkasında Gates Vakfı'nın olduğu iddiası

EMRE SAKLICA ✖ 01/02/2020 ✖ 03:00

Yayılan bir iddiada koronavirüs çıkmadan üç ay önce Gates Vakfı'nın virüsün ilacının patentini alıp yayılma simülasyonu hazırladığı iddia edildi. Ancak iddia doğru değil.

9 Şubat 2020 Düzenle

teyit.org

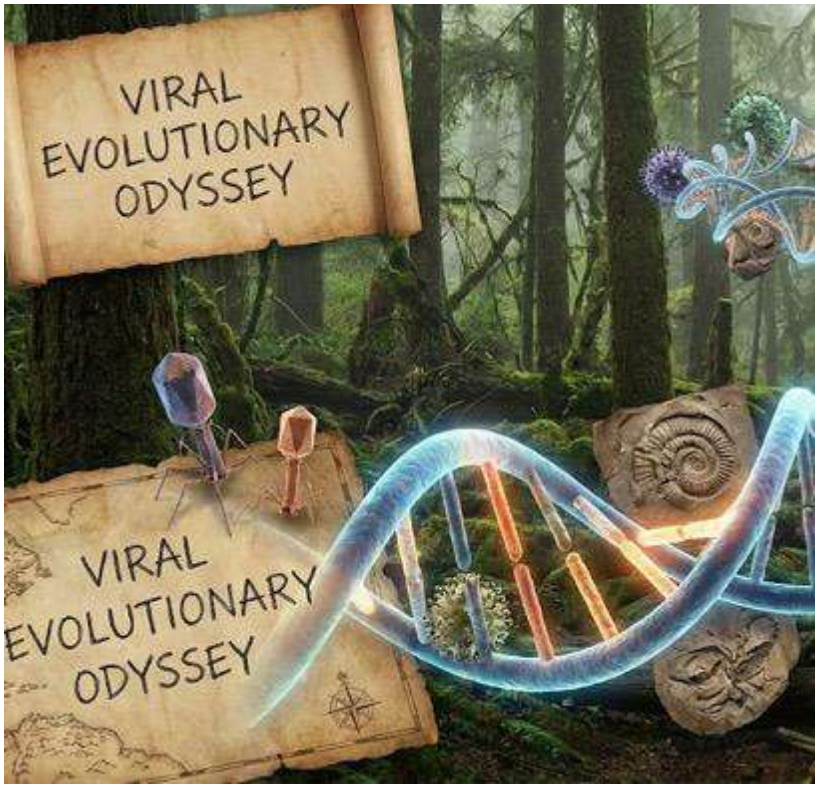
Başlarken: Yarasa çorbası videosu Çin'den değil

İddiaların yayılmasında bir videonun ayrıcalıklı bir yeri vardı. Dünya çapındaki sosyal medya kullanıcılarının Çin'de yarasa çorbası yendiğini gösterdiği iddiasıyla paylaştığı video, Çin halkının yemek alışkanlıklarıyla ilgili yargıları pekiştirdi.

Halbuki, Les Observateurs, South China Morning Post, News, Metro gibi internet sitelerinin de ortaya koyduğu gibi, video ünlü bir gezi bloggeri olan Wang Mengyun'a aitti ve Çin'de değil, bir Pasifik adası olan Palau'da 2016'da kaydedilmişti. Mengyun bölgeyi tanıtırken yarasa çorbası da dahil olmak üzere yerel lezzetleri de tatmıştı. 2016'da böyle bir salgın yoktu. Mengyun'un konu hakkında açıklama yaptığı Weibo gönderisi kaldırılsa da, açıklamanın ekran

VİRAL BİR YOLCULUK

- Virüsler konak genomlarını ve konaklar virüs genomlarını şekillendiriyor
- Salgınlar bağışıklık sistemlerimizi etkiliyor
- Yaşam ağacındaki savunma sistemlerinin korunması ve hızlı evriminin karşılaştırılması; silahlanma yarışları otoimmünite veya kanser gibi hastalıklara katkıda bulunuyor
- Rezervuar konaklar çeşitli virüs enfeksiyonlarıyla başa çıkıyor
- Geçmişteki çatışmalar günümüzdeki virüs ortaya çıkışına yatkınlığa katkıda bulunuyor



News & views

Immunogenetics

https://doi.org/10.1038/s41588-025-02490-x

Written in our genes, epigenetically edited by infection

Luis B. Barreiro & Musa M. Mhlanga

Three layers shaping infection-driven epigenetic remodeling

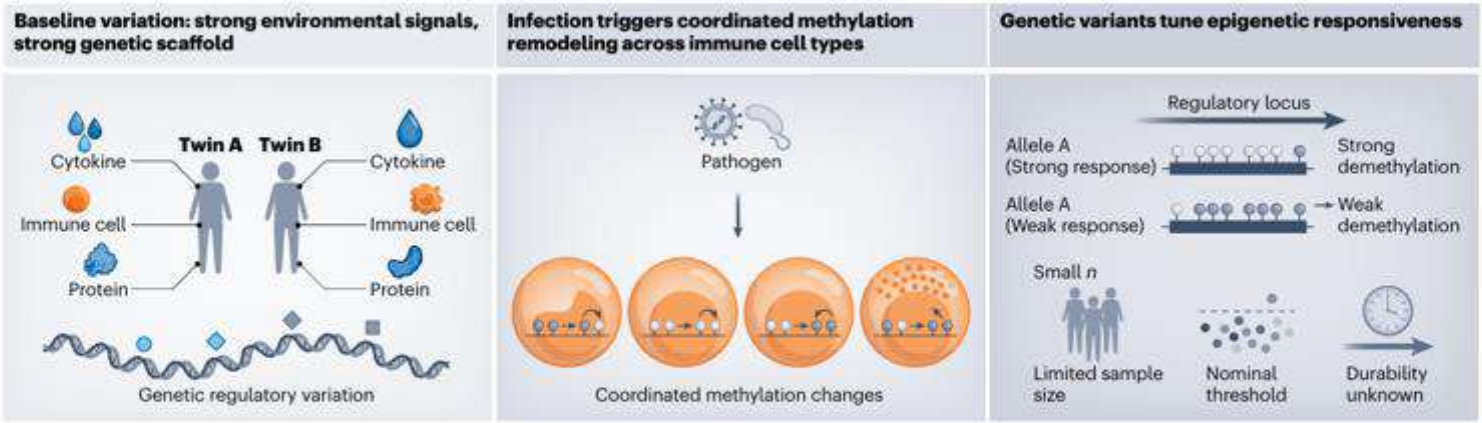
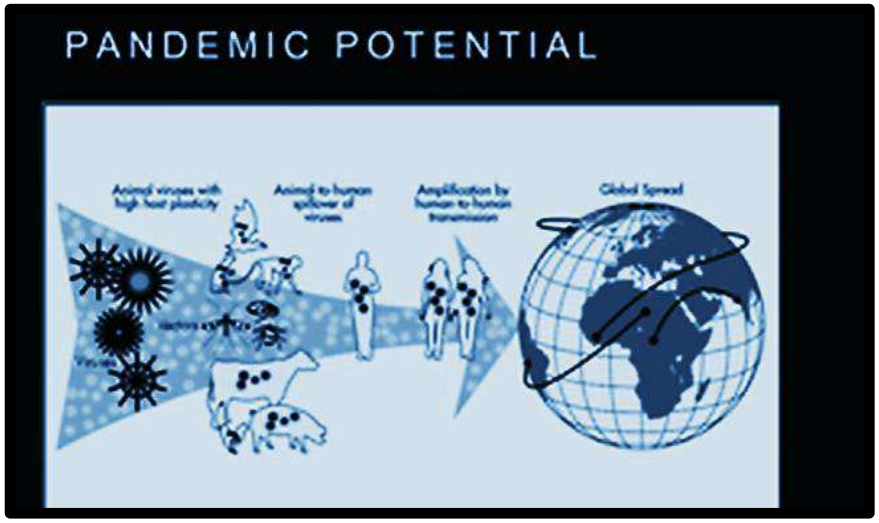


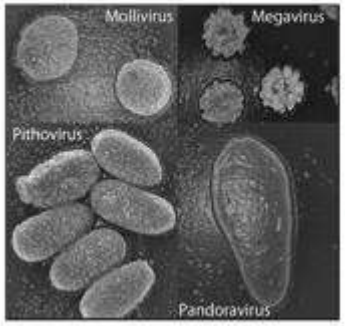
Fig. 1 | Genetic and non-heritable factors shape infection-driven epigenetic remodeling. Environmental stimuli induce DNA methylation changes across immune cells, and regulatory variants modulate their magnitude and direction. Limited cohort sizes and nominal thresholds reduce resolution. Together, these findings show how inherited predisposition and environmental exposures collectively sculpt the immune epigenome during infection.

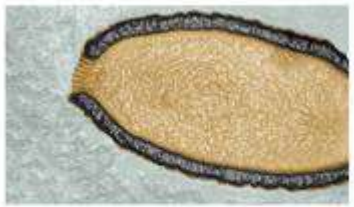
- 50 BİN VERTEBRALI TÜRÜ VAR
- 20 VİRUS/TÜR:1 MİLYON VİRUS
- 1500 İNSAN PATOJENİNİN: %60 ZOONOZ
- 10^{31} VİRÜS VAR≈100 MİLYON IŞIK YILI



HENÜZ KEŞFEDİLMEMİŞ AMA ARAMIZDAKİ VİRÜSLER

30,000-YEAR OLD GIANT VIRUS IN SIBERIAN SOIL





Pithovirus sibericum

MICROBIOLOGY

NEWS

Computer scan uncovers 100,000 new viruses

Clues to future outbreaks may be hidden in existing genomic databases

By Elizabeth Pennisi

It took just one virus to cripple the world's economy and kill millions of people; yet virologists estimate that trillions of still-unknown viruses exist, many of which might be lethal or have the potential to spark the next pandemic. Now, they have a new—and very long—list of possible suspects to interrogate. By sifting through unprecedented amounts of existing genomic data, scientists have uncovered more than 100,000 novel viruses, including nine coronaviruses and more than 300 related to the hepatitis Delta virus, which can cause liver failure.

"It's a foundational piece of work," says J. Rodney Brister, a bioinformatician at the National Library of Medicine. The study, published last week in *Nature*, expands the number of known viruses that use RNA instead of DNA for their genes by an order of magnitude. It "demonstrates our outrageous lack of knowledge about this group of organisms," says disease ecologist Peter Daszak, president of the EcoHealth Alliance, a nonprofit research group in New York City that is raising money to launch a global survey of viruses.

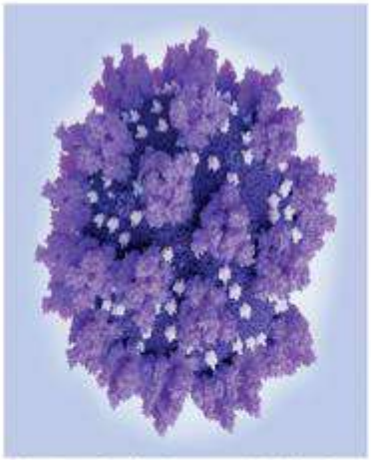
Scientists predict the study will also help launch so-called petabyte genomics—the analyses of previously unfathomable quantities of DNA and RNA data. (One petabyte is 10^{15} bytes.) That wasn't exactly what computational biologist Artem Babaian had in mind when he came up with the project while in between jobs in early 2020. Instead, he was simply curious about how many coronaviruses—aside from the virus that had just launched the COVID-19 pandemic—could be found in sequences in existing genomic databases.

So, he and independent supercomputing expert Jeff Taylor scoured cloud-based genomic data that had been deposited to a global sequence database and uploaded by the U.S. National Institutes of Health. As of now, the database contains 16 petabytes of archived sequences, which come from genetic surveys of everything from fugu fish, the risky Japanese delicacy, to farm soils to human guts. (A database with a 5-megabase digital photo of every person in the United States would take up about the same amount of space.) The sequences also capture the genomes of viruses infecting different organisms in samples, but the viruses usually go undetected.

To sift through the reams of data, Babaian and Taylor devised a set of computer search tools specialized for cloud-based data. With the help of several bioinformaticians, some whom became collaborators on the project, they tweaked the new software to make their analysis "way faster than anyone thought possible," recalls Babaian, who is now at the University of Cambridge.

They soon expanded the viral hunt beyond coronaviruses and looked at all the data in the cloud. Babaian and his colleagues' programs hunted among the cloud's sequences for matches to the central core of the gene for RNA-dependent RNA polymerase, which is key to the replication of all RNA viruses. Such viruses include not only coronaviruses, but also those that cause flu, polio, measles, and hepatitis.

Babaian's approach was fast enough to work through 1 million data sets a day—at a com-

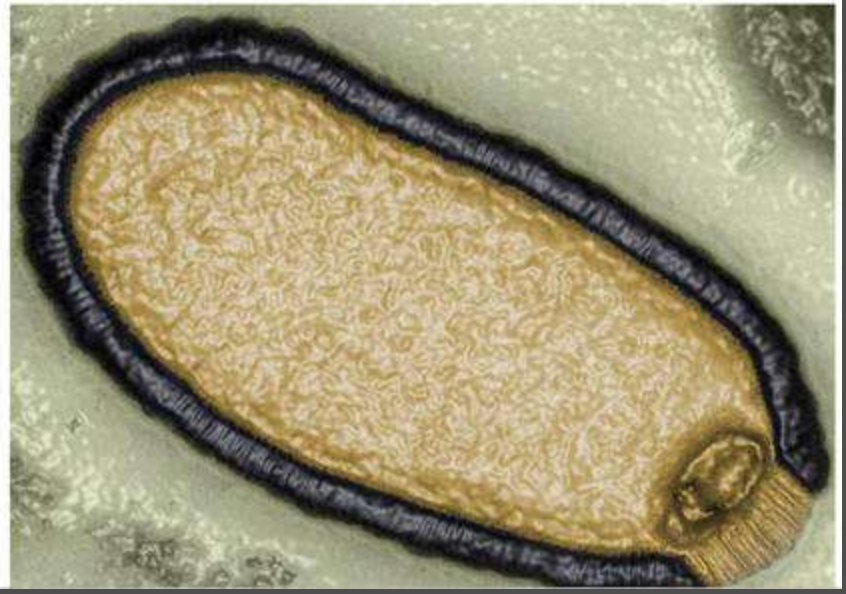


In a vast repository of genetic sequences, scientists found nine unknown coronaviruses, relatives of SARS-CoV-2 (computer model).

IMAGE: JUAN GAERTNER/SCIENCE SOURCE

Arctic zombie viruses in Siberia could spark terrifying new pandemic, scientists warn

Threat of outbreak from microbes trapped in permafrost for millennia raised by increased Siberian shipping activity



Researchers from Aix-Marseille University were the first to isolate viruses from ancient permafrost, reporting in 2014 and 2015 that samples of Siberian soil frozen for 30,000 years harbored two large DNA viruses that could infect amoebae but posed no threat to humans. In the 18 February issue of *Viruses*, the same team, led by genomicist Jean-Michel Claverie and materials scientist Chantal Abergel, revealed 13 more permafrost megaviruses that infect amoebae, one dating back 48,500 years.

- Yeni araştırmalar, insanların on binlerce yıldır HPV'den muzdarip olduğunu öne sürüyor. Bilim insanları yakın zamanda, 5300 yıllık **Ötzi Buz Adamı** ve **45000 yıllık Ust'-Ishim** adamının her ikisinin de günümüzde insanlarda kansere neden olduğu bilinen yüksek riskli bir tür olan **HPV16** ile enfekte olmuş olabileceğini keşfetti

bioRxiv
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Oncogenic HPV types identified in Paleolithic and Chalcolithic human genome sequencing data from Ust'-Ishim and Ötzi

Juliana B. Yazigi[1], Caio O. Cyrino[1,3], Cristina M. Peter[1,2], Renata C. Ferreira[1,4], Juliana T. Maricato[1], Luiz M. Janini[1,3], Marcelo R.S. Briones[1*]

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Abstract

Human papillomaviruses (HPVs) are ancient viruses with diverse lineages infecting epithelial tissues in primates and humans. Although contemporary distribution and clinical importance are well understood, there is limited knowledge about their occurrence among prehistoric human populations. We investigated the presence of HPV in ancient anatomically modern humans (AMHs) by analyzing genome sequencing data from two exceptionally preserved individuals: Ust'-Ishim (~45,000 years BP) and Ötzi the Iceman (~5,300 years BP). Using a combination of reference-guided mapping and ancient DNA authentication criteria, we searched for HPV sequences in these ancient genomes. We detected high-confidence papillomavirus fragments in both individuals. Further phylogenetic and comparative analyses revealed that the reconstructed sequences belong to HPV16, the most oncogenic HPV lineage. Our study presents the earliest molecular evidence of HPV16 in anatomically modern humans (AMHs), pushing back its evolutionary history and challenging the idea that HPV16 entered *Homo sapiens* through Neanderthal interbreeding. Our results suggest that HPV16 was already present in modern humans during the Upper Paleolithic, indicating a long-standing host–virus association independent of Neanderthal transmission.

Keywords: Paleovirology, Ancient DNA, HPV, Oncogenesis, Human evolution

- "Homo sapiens, Neandertallerle melezleşme yoluyla bu virüsü yaymıştır. Sonuçlarımız, HPV16'nın Üst Paleolitik dönemde modern insanlarda zaten mevcut olduğunu ve Neandertal bulaşmasından bağımsız olarak uzun süreli bir konak-virüs ilişkisine işaret ettiğini göstermektedir."

Smithsonian *magazine*

A Shrew-Borne Virus Is Responsible for Deadly Brain Infections in Humans

First discovered in livestock hundreds of years ago, Borna disease virus has apparently been claiming human lives for decades

Now, after years of fruitless searching for Borna in people, it's clear that the virus indeed infects humans—and has likely been killing them for decades, reports Kai Kupferschmidt for *Science* magazine. In a study published this week in *Lancet Infectious Diseases*, researchers identified eight instances of lethal Borna disease in humans, roughly doubling the number of known infections in our species.

"Borna disease virus infection has to be considered a severe and potentially lethal human disease," says study author Barbara Schmidt, a microbiologist at Regensburg University Hospital in Germany, in a statement.

To better understand these patterns of infection, Beer and his colleagues searched for the genetic evidence of the virus in 56 samples of brain tissue collected in Germany between 1995 and 2018. All the patients had died from some kind of brain inflammation, which can result from autoimmune disease, cancer, infection and a variety of other conditions. Half the specimens had been logged without a known cause for the inflammation. In seven of these, the researchers discovered traces of Borna disease virus. An additional search at another German medical center turned up yet another case, bringing them to a total of eight patients, two of whom had been recipients of organ transplants.

Crocidura leucodon

Article

The spatiotemporal distribution of human pathogens in ancient Eurasia

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Open access
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Martin Sikora[1,2], Elisabetta Canteri[3], Antonio Fernandez-Guerra[1], Nikolay Oskolkov[3], Rasmus Ågren[4], Lena Hansson[5], Evan K. Irving-Pease[1], Barbara Mühlemann[6,7], Sofie Holtsmark Nielsen[8], Gabriele Scorrano[1,9], Morten E. Allentoft[1,10], Frederik Valeur Seersholm[1], Hannes Schroeder[2], Charleen Gaunitz[1], Jesper Stenderup[1], Lasse Vinner[1], Terry C. Jones[6,11], Björn Nystedt[12], Karl-Göran Sjögren[13], Julian Parkhill[14], Lars Fugger[15,16,17], Fernando Racimo[2], Kristian Kristiansen[1,18], Astrid K. N. Iversen[1,17] & Eske Willerslev[1,18,19]

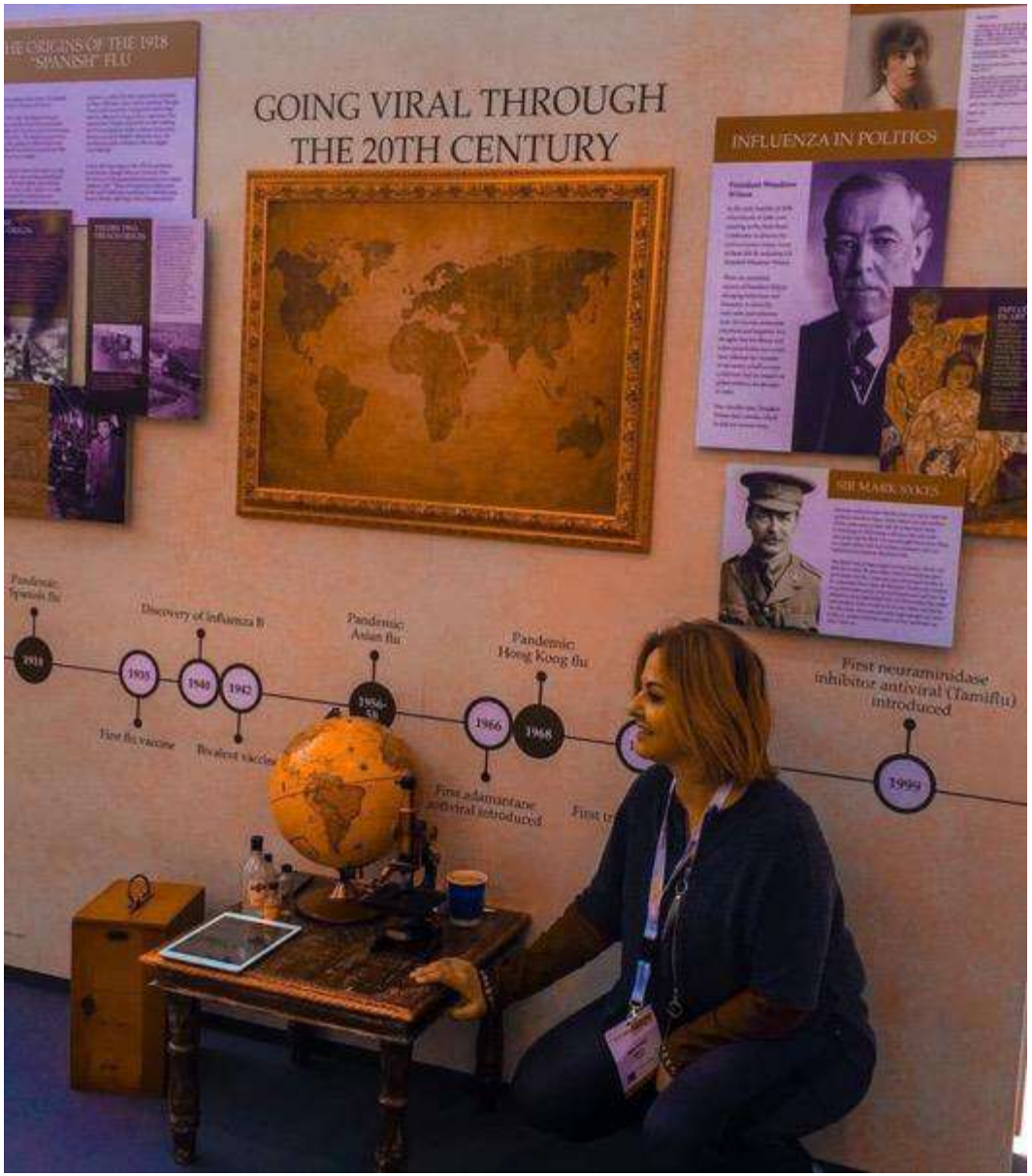
Infectious diseases have had devastating effects on human populations throughout history, but important questions about their origins and past dynamics remain[1]. To create an archaeogenetic-based spatiotemporal map of human pathogens, we screened shotgun-sequencing data from 1,313 ancient humans covering 37,000 years of Eurasian history. We demonstrate the widespread presence of ancient bacterial, viral and parasite DNA, identifying 5,486 individual hits against 492 species from 136 genera. Among those hits, 3,384 involve known human pathogens[2], many of which had not previously been identified in ancient human remains. Grouping the ancient microbial species according to their likely reservoir and type of transmission, we find that most groups are identified throughout the entire sampling period. Zoonotic pathogens are only detected from around 6,500 years ago, peaking roughly 5,000 years ago, coinciding with the widespread domestication of livestock[3]. Our findings provide direct evidence that this lifestyle change resulted in an increased infectious disease burden. They also indicate that the spread of these pathogens increased substantially during subsequent millennia, coinciding with the pastoralist migrations from the Eurasian Steppe[4,5].

Nature dergisinde yayınlanan yeni bir çalışmada, bir grup bilim insanı son 37.000 yıl boyunca Avrupa ve Asya'daki 214 hastalığın genetik tarihini inceledi.
Bulguları, hayvancılığın yükselişinin insanlığın hastalıkla ilişkisini sonsuza dek şekillendirdiğine dair güçlü kanıtlar sunuyor, ancak zamanlama beklentilerinin aksine oldu.

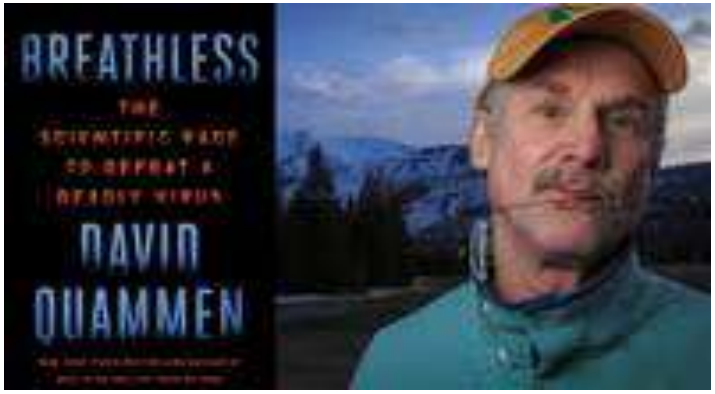
Zoonotik patojenler yaklaşık 6500 yıl öncesinden itibaren tespit edilmeye başlanmış ve yaklaşık 5000 yıl önce, hayvanların yaygın evcilleştirilmesiyle aynı zamana denk gelecek şekilde zirve yapmıştır[3]. Bulgularımız, bu yaşam tarzı değişikliğinin bulaşıcı hastalık yükünde artışa yol açtığına dair doğrudan kanıt sağlamaktadır. Ayrıca, bu patojenlerin yayılımının, Avrasya Bozkırlarından gelen pastoralist göçlerle[4,5] aynı zamana denk gelecek şekilde, sonraki bin yıllar boyunca önemli ölçüde arttığını göstermektedir.

.Sana attığım email'i almadın mı?

İnfeksiyon Hastalıklarının , seyahatlerimiz ile sınırlar aştığını, 20.yy da olduğu gibi politikanın ve bilimin gündeminde ilk sırada kalacağını göreceğiz..
Geleceğin Tıp" ı , kişiselleştirilmiş tıp , 4P kuralı, mikrobiyom ,vektörler , direnç ve tabii mikroplar ile şahane yolculuklarımız ve maceralarımızı konuşacağız..
Tabii, inanca meyilli kişilerin ,infeksiyoncu olmayan cemaatlerin yalan- dolan rüzgarlarına kapılmaması zor olacak..
Ben bloğumda çok güzel



2012-DAVİD QUAMMEN,SIÇRAMALAR;HAYVAN İNFEKSİYONLARI VE GELECEK İNSAN PANDEMİSİ



the scientific race to defeat a deadly virus

In 2012, author David Quammen wrote a book, *Spillover: Animal Infections and the Next Human Pandemic*, that was the result of five years of research on scientists who were looking into the possibility of another Ebola-type disease emerging. The consensus: There would indeed be a new disease, likely from the coronavirus family, coming out of a bat, and it would likely emerge in or around a wet market in China.

But what was not predictable was how unprepared we would be. In this interview, the *Bulletin*'s Dan Drollette Jr talks with the author, who lives in Bozeman, Montana, about what drew him to this topic, the nature of new viruses, why more are expected to emerge, and what makes some viruses more likely to infect humans than others. Quammen also talks a little about his next book (still untitled, but about the coronavirus). He cautions against being overly optimistic about the development of a vaccine, saying the coronavirus that causes COVID-19 will likely be around in some form for generations: "This virus is never going to be gone."

SARS-like viruses may jump from animals to people hundreds of thousands of times a year

NEWS

Study pinpoints Asian regions that could spark the next coronavirus pandemic

By Kai Kupferschmidt

most likely to emerge," Daszak says. The maps could guide efforts to reduce the likelihood of spillover by changing behaviors in high-risk communities and targeting surveillance to detect new outbreaks earlier, he says. Daszak, a vocal advocate of the hypothesis that SARS-CoV-2 came from the wild instead of a research lab, says the maps could also guide efforts to find the virus' natural origin. (Several studies are underway or being planned to look for SARS-CoV-2 and its relatives in *Rhinolophus* [horseshoe] bats and other animals.)

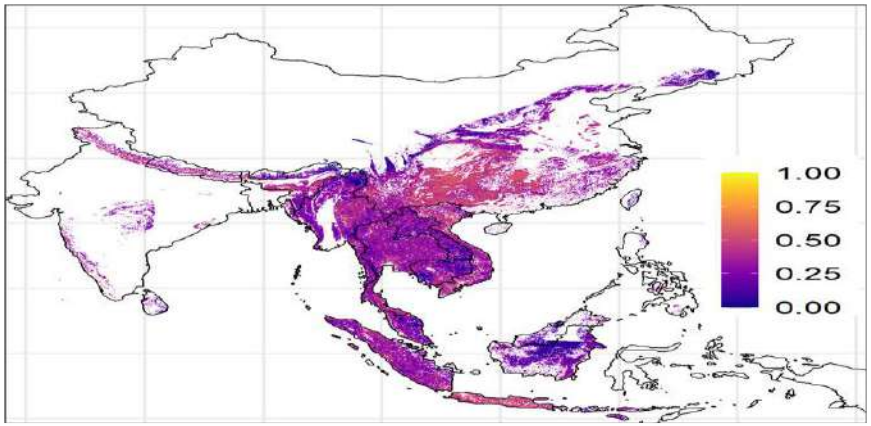
But the researchers went one step further. Small surveys done before COVID-19 erupted

"I think if the seroprevalence estimate is way off, the whole thing collapses," says David Fisman, an epidemiologist at the University of Toronto, who calls the modeling "shaky." The high number of hidden infections "doesn't ring true," Fisman says, because you would expect regular spillovers to be recognized, as they are for rabies and the Nipah virus.

But Rasmussen says many infections could remain hidden if they are short-lived and don't lead to onward transmission because the viruses are not well adapted to humans. They might not infect enough cells—or cells of the right type—to be transmitted to another person, or they

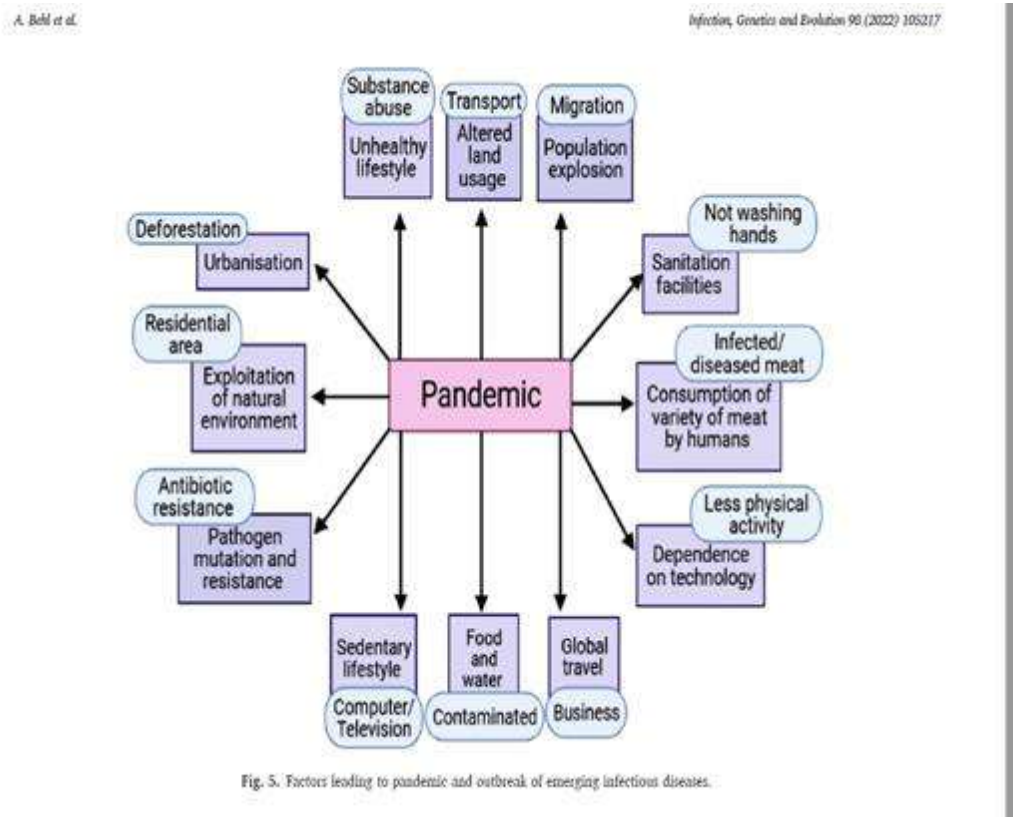
23 yarasa türü
500 milyon insana sıçrama

calculated that some 400,000 undetected human infections with these viruses occur each year across the region.



MİKROP,KONAK,ÇEVRESEL DEĞİŞİM

- **BİZ VİRÜSLER OKYANUSUNDA BİR ADAYIZ**
- **«DÜNYA VİRÜSLERİN ARKA BAHÇESİDİR»**
- **VİRAL YÜZYIL**



VSE IMA
SVOJO URO,
VSAKO VESELJE
IMA SVOJ ČAS POD
NEBOM.
Prd 3,1

EVERYTHING ON
EARTH HAS ITS OWN
TIME AND ITS OWN
SEASON.


KAAN'CIM SÜPER KAHRAMAN MI OLDUN SEN? HANGİ KAHRAMAN OLDUN TEYZECİM?
ENFEKSİ-YON HAS-TALIKLARI UZMANI.

Bir enfeksiyon uzmanı hekim, bir kamu entelektüeli ve nesnel gerçekliğin dinamizmini her türden önyargı, boş inanç ve popülizmden bağımsız kavrama çabası gösteren Esin Davutoğlu Şenol, bu kez, salgınla karşılaşmasını hızla direnişe çevirmeyi başarmış bir yazar olarak karşımızda.

Bu kitap yalnızca bilgece tutulmuş bir "pande günlüğü" değil, gerçekliğin içinde ve aracılığıy , di tüm var olanlar arasında bir varlık olarak insan olmak, etkileşmek, dayanıklı bir varoluş için çabalamak, kavramak, diren t mül e üzerine özenle hazırlanmış e lebi nem de bü Şenol, salgının ilk gününden günümüze gelinceye dek deneyimlenenleri f sel, edebi, bilimsel, etik ve politik pasajlarla z ngin eşti yo Böyec çık azımızın haberdar ldu nfek yon ili deki "nesnel bilimsel perspektifi" pek çoğumuza tanıdık gelecek "amatör bir yaşama telaşıyla" bir arada ele alıyor. Her açıdan özel bir kitap.

– Çetin Balanuye, Yazar

Humanist

SALGININ SEYİR DEFTERİ

Prof. Dr. Esin Şenol

H

SALGININ SEYİR DEFTERİ

Bir Enfeksiyon Hekiminin Salgın Günlüğü

Prof. Dr.
Esin Şenol

Humanist

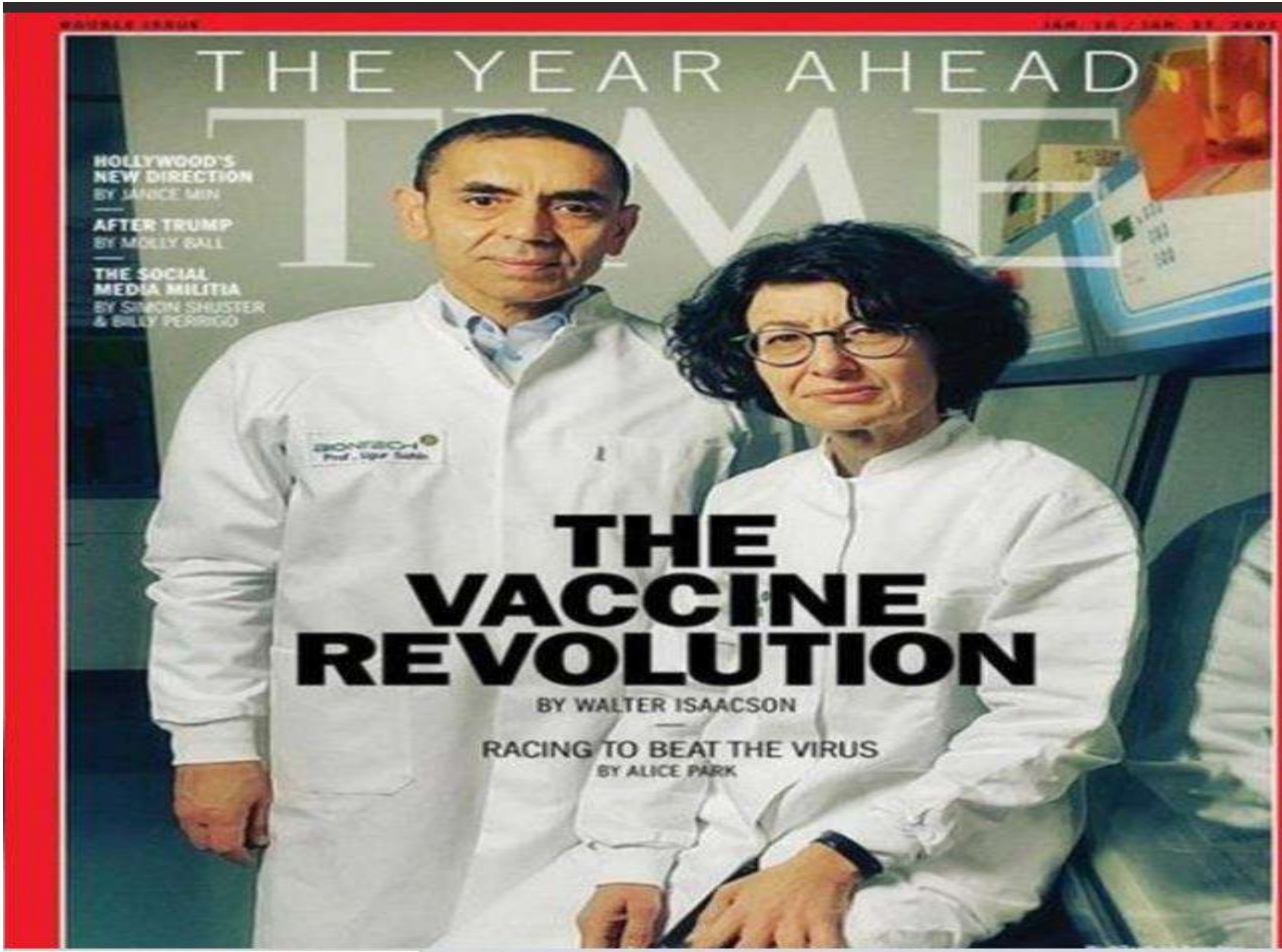
HİÇBİR ŞEY HATIRLANMAMAK ÜZERE YAŞANMAMALI

Artık her şeyin başka türlü yaşanma zamanıdır..


TUNAY @tunay_1071 · 1 gün
Bir enfeksiyoncu profesör düşünün maskesini koluna taksın :)) Sorular çalıntı tezler kopyala yapıştır olunca ortaya bir Türkiye fotoğrafı çıkmış :)))
1
8
21
Engn @Engn85... · 1 gün
Chp yandasi Doktora bakin hele

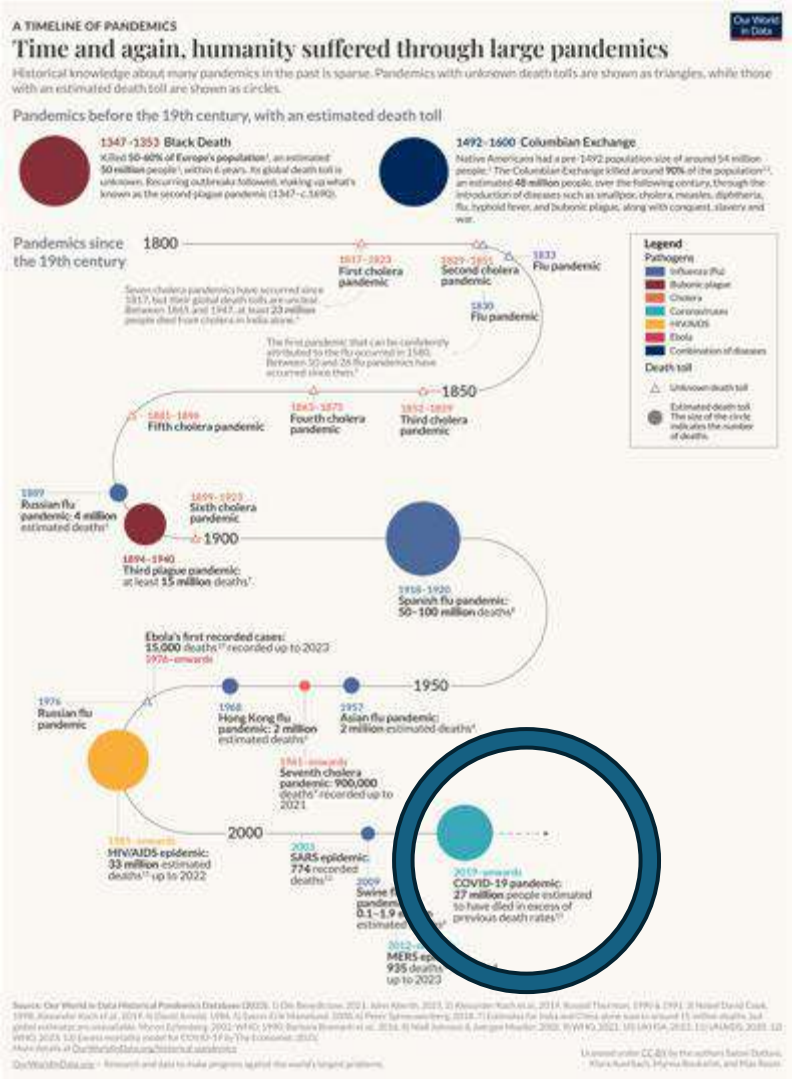

akit
ÇANAKKALE ZAFERİ'NİN 105. YILINDA
Hazımsızlığın böylesi görülmedi! Sözde 'Prof' Esin Şenol'dan Türkiye için küstah sözler
Son Haberler
HDP Grup Başkanvekili Meral Danış Beştaş karantinaya alındı mı?
Gündem
Hazımsızlığın böylesi görülmedi! Sözde 'Prof' Esin Şenol'dan Türkiye için küstah sözler
Çin'in Vuhan kentinde ortaya çıkan ve küresel bir salgına dönüşen koronavirüsle ilgili Türkiye'nin aldığı önlemler büyük takdir toplarken bazı kesimlerde ise hazımsızlığa neden oldu. Verilen başarılı mücadeleyi baltalamaya çalışan sözde profesör Esin Şenol, "Virüs Türkiye'de yayılmadıysa bu başarıdan ziyade tesadüf" ifadelerini kullandı.

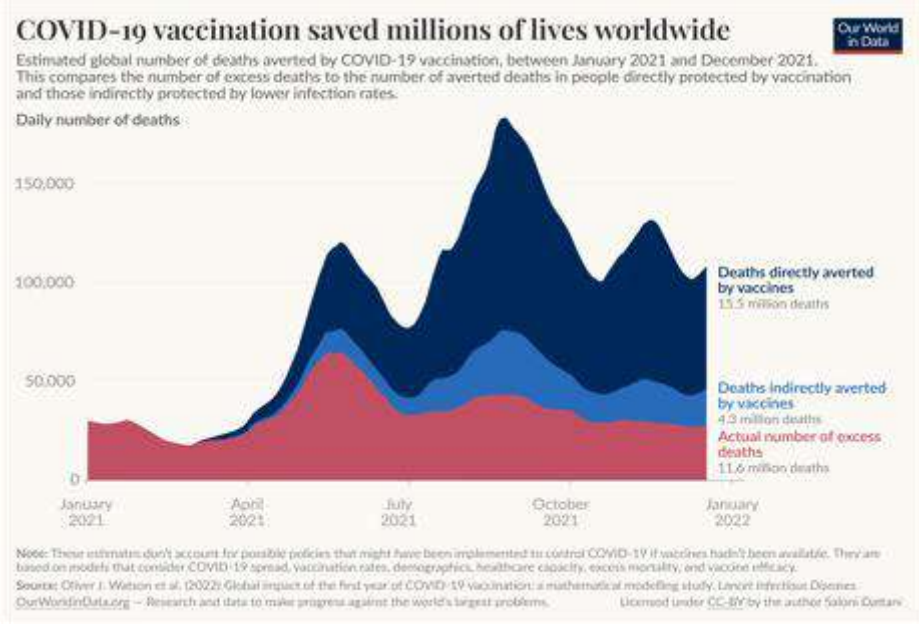

cezmikalorifer
Erkan Erten
@erkanerten
Twiter ı anlatan en iyi foto serisini buldum 😂😂
Coronavirus Report @Koron... · 3 sa
SON DAKİKA
Tarihin en ölümcül salgınlarından bubonik veba yeniden Çin'de ortaya çıktı ve 3 numaralı alarm verildi. —Euronews
477 2.550 4.653
Esin Davutoğlu Şenol @esenol · 2 sa
Onun tedavisi var ve kolay
16 13 626
Ömer iyi çocuktu
@niyeabiniye
den biliyosunuz ablacım bence
değildir o kadar
Takip et
Esin Davutoğlu Şenol
@esenol
Professor of Medicine, Infectious Diseases Specialist, works at Gazi University, Faculty of Medicine, married with one child
Takip et
Ömer iyi çocuktu
@niyeabiniye
Manav


THE YEAR AHEAD
TIME
HOLLYWOOD'S NEW DIRECTION
BY JANICE MIN
AFTER TRUMP
BY MOLLY BALL
THE SOCIAL MEDIA MILITIA
BY SIMON SHUSTER & BILLY PERRIGO
THE VACCINE REVOLUTION
BY WALTER ISAACSON
RACING TO BEAT THE VIRUS
BY ALICE PARK

COVID-19 aşıları

- Hastaneye yatışlar ve ölümler , ciddi hastalık olasılığını azaltmada oldukça etkili
- Sadece ilk yılında dünya çapında yaklaşık 20 milyon hayat kurtardığını tahmin edilmekte





The RNA sequence used in the COVID-19 vaccine developed by Pfizer and BioNTech (Ψ is a modified form of the uridine nucleotide, U).

THE TANGLED HISTORY OF MRNA VACCINES

Hundreds of scientists had worked on mRNA vaccines for decades before the coronavirus pandemic brought a breakthrough. **By Elie Dolgin**

INSIDE AN MRNA COVID VACCINE

COVID-19 vaccines made from messenger RNA use lipid nanoparticles — bubbles of fats — to carry the molecules into cells. The mRNA contains the code for cells to produce the 'spike' protein that the coronavirus SARS-CoV-2 uses to enter cells. Here are key innovations in the design of these vaccines.

mRNA

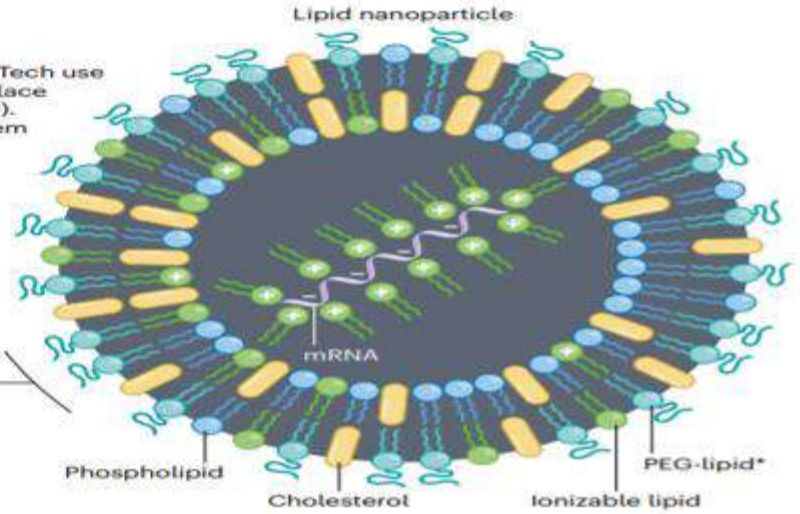
...CGAGΨΨCGΨGΨΨΨAA...

The vaccines made by Moderna and Pfizer–BioNTech use mRNA that has been chemically modified to replace the uridine (U) nucleotide with pseudouridine (Ψ). This change is thought to stop the immune system reacting to the introduced mRNA.

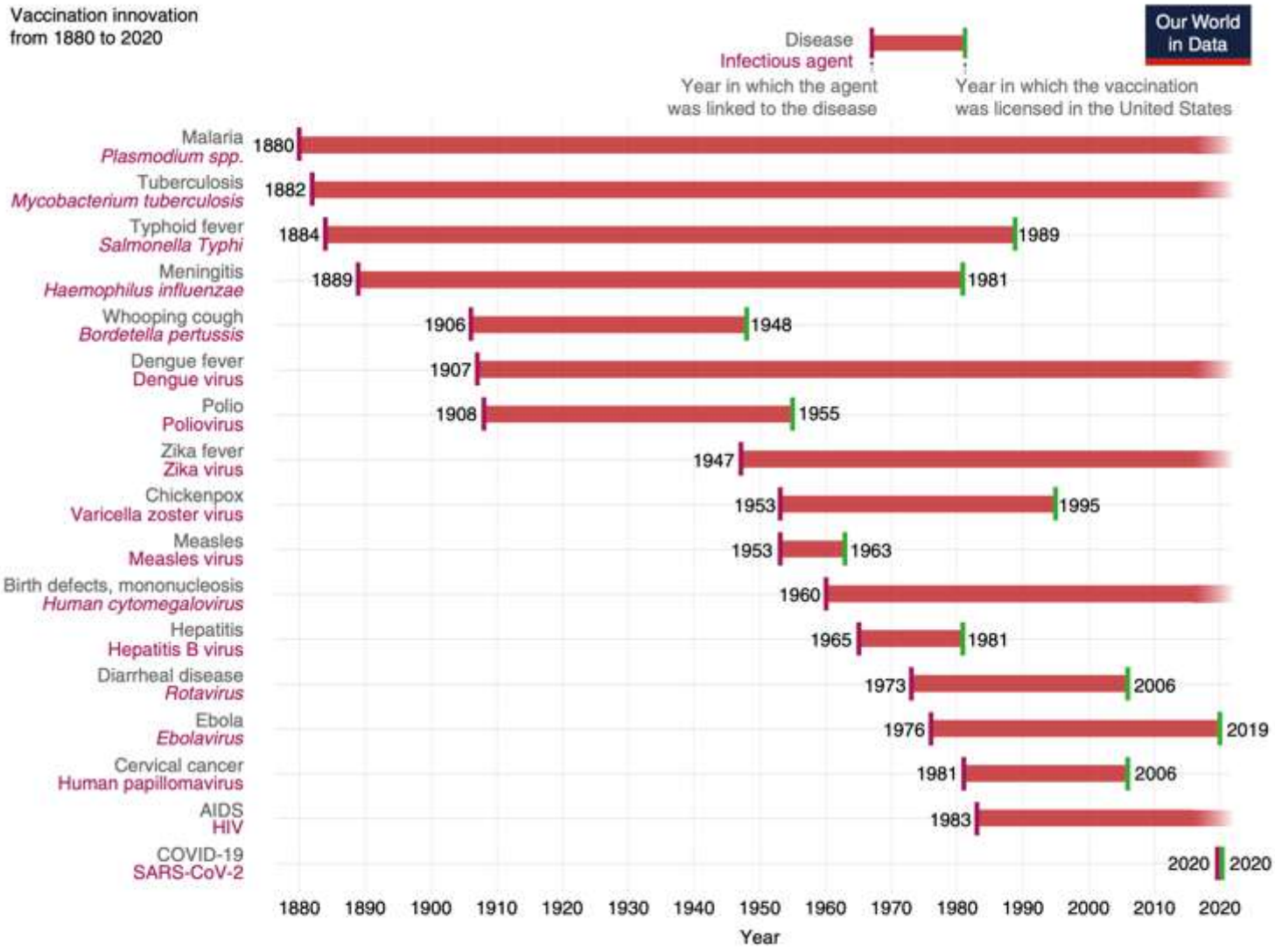
To help the body mount an effective immune response to later SARS-CoV-2 infections, the mRNA sequence is adapted to stabilize the spike protein in the shape it uses when fusing with human cells.

Lipids

The fatty nanoparticle around the mRNA is made of four types of lipid molecule. One of these is 'ionizable': in the vaccine, many of these molecules have a positive charge and cling to negatively charged mRNA, but they lose that charge in the more alkaline conditions of the bloodstream, reducing toxicity in the body.



*Lipid attached to polyethylene glycol



Timeline of innovation in the development of vaccines. Each bar begins in the year in which the pathogenic agent was first linked to the disease

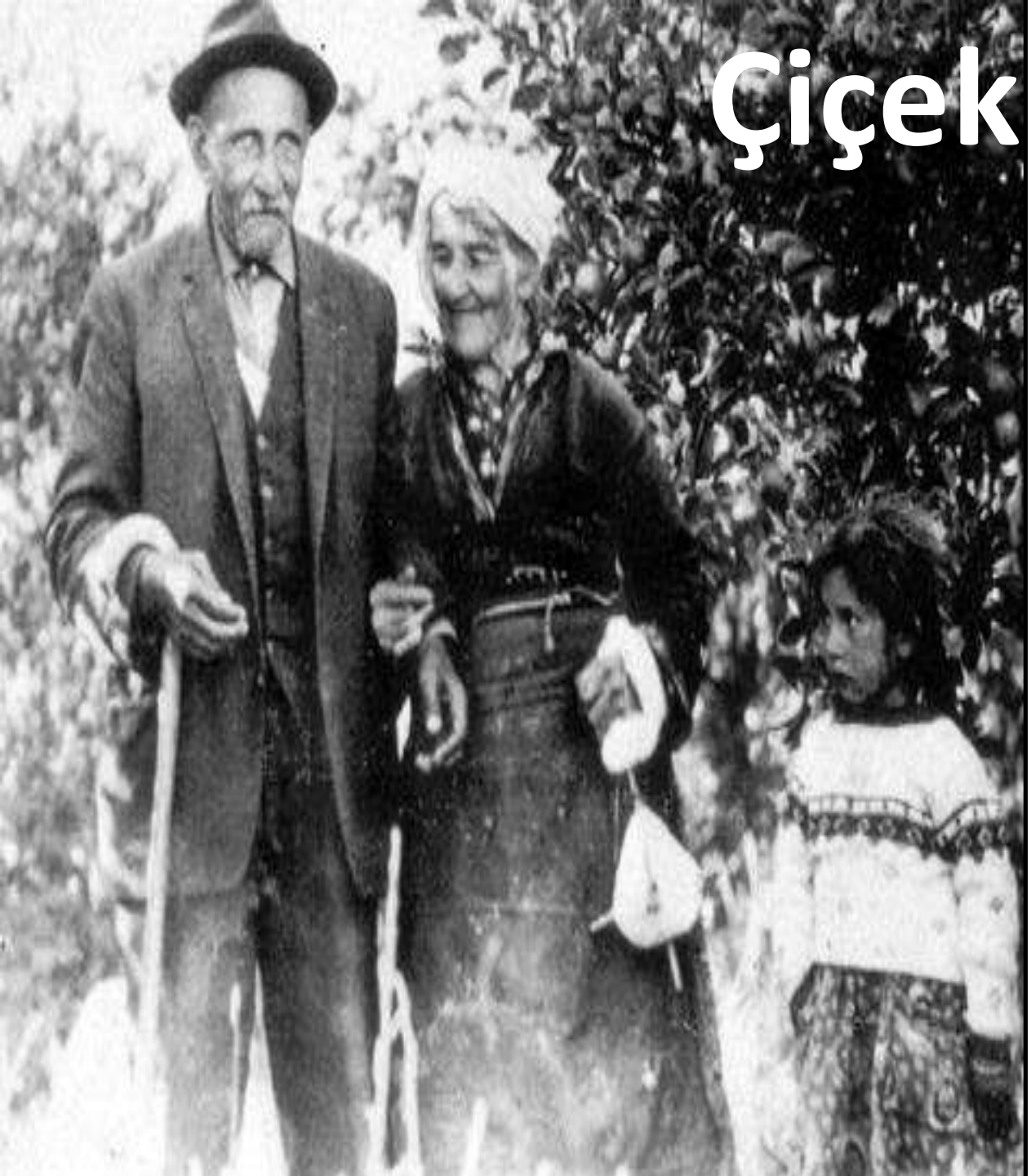
Salgın 20-40 yaş arasındakileri öldürdü

Alaska “Yetimleri”, 1919

PANDEMİNİN KIRILGANLAŞTIRDIĞI YENİ KIRILGAN BİR NÜFUS VE KIRILGANLAŞAN SAĞLIK SİSTEMLERİ

Tedros Adhanom Ghebreyesus
@DrTedros

The number of people in need of humanitarian relief has increased by 25% compared to last year. With funding & urgent action, we can:
-save lives
-support recovery
-prevent the spread of diseases
-give communities the opportunity to rebuild for the future



Çiçek

Elimde bir
göztaşı/gözlerim boş
gidiyordum
Ne bileyim bir türkünün
böyle Veysel olduğunu
Açıldım/çıkmaz bir sokak
gibi/kapanınca denizde

CAN YÜCEL

Mikroplara Karşı İnsanlık Tarihi

Başlıca Aşı Devrimleri:

Mayıs 1980'de Çiçek hastalığının eradikasyonu

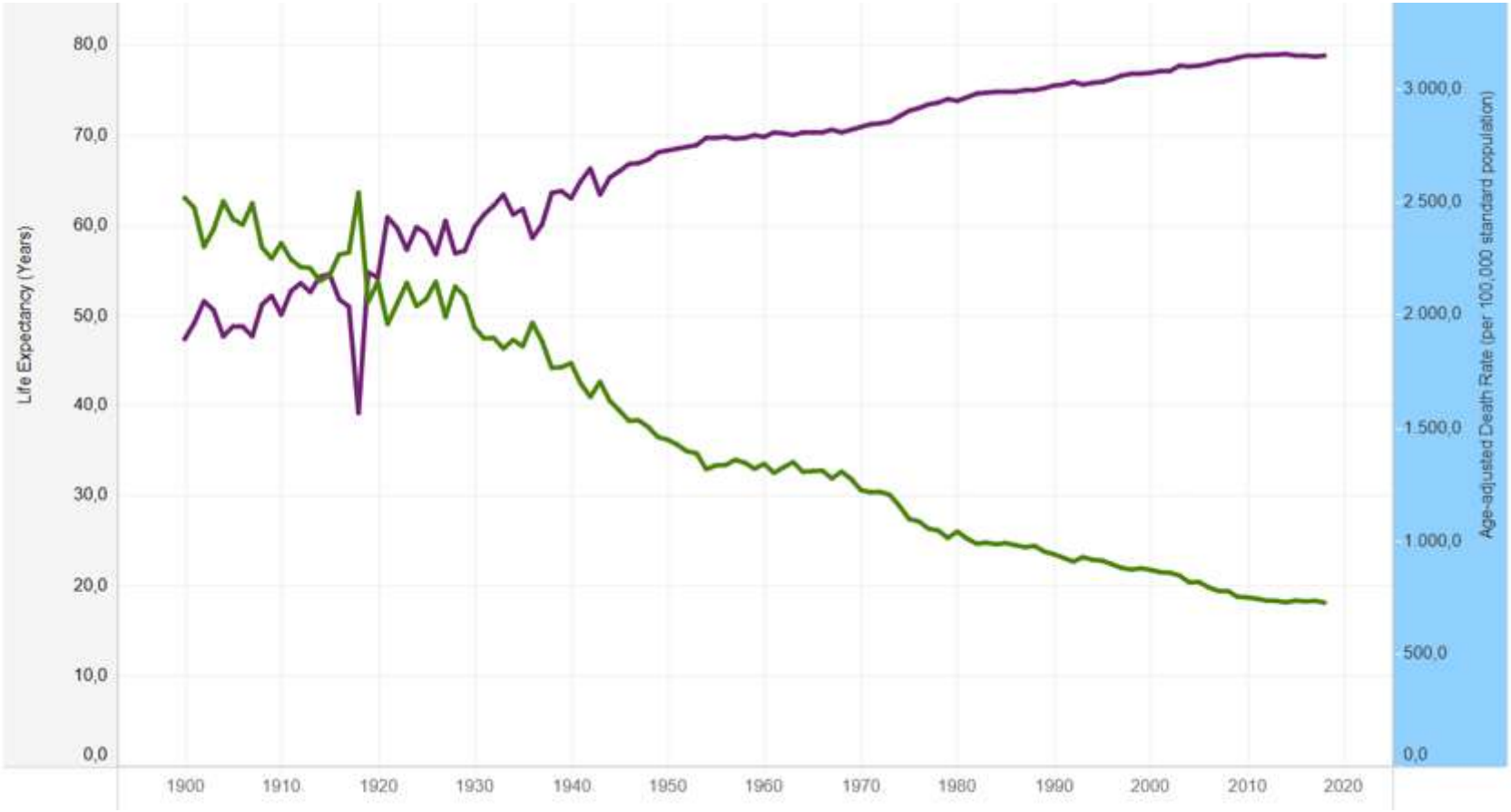
Çiçek hastalığına tutulmuş bir bebek. *(Centers for Disease and Prevention Public Heakh Image Library; by Dr. Noble, 1966.)*

Çiçek hastalığından ölen son olgu (1978), Janet Parker

M.Ö. 3000	18 y.
M.Ö. 275	26 y.
M.S. 1900	49 y.
M.S. 1980	76 y.
M.S. 2002	85 y.
M.S. 2020	90 y.


Erkek olanına baba, Ötekine de anne derim...
Amaan... Sağlıklı olsunlar da!..

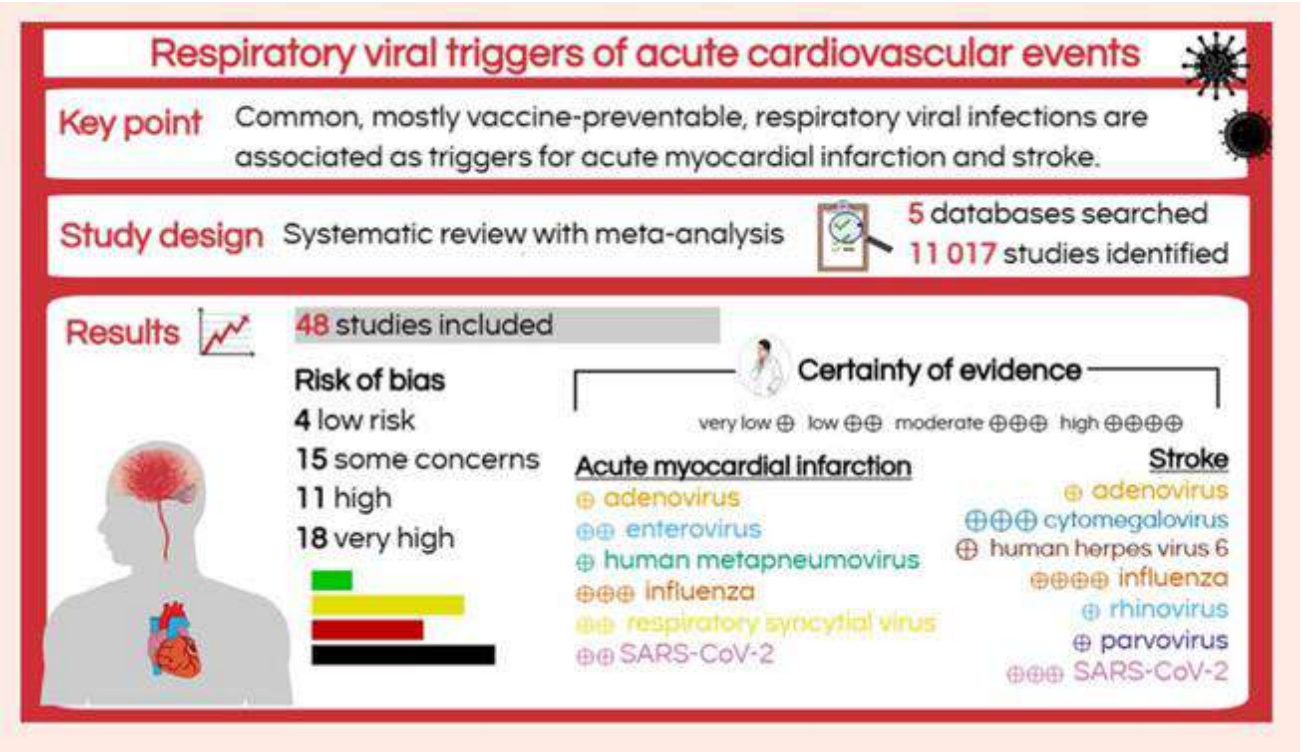
Age-adjusted Death Rates† and Life Expectancy at Birth (Both Sexes, All Races): United States, 1900 to 2018‡‡



https://www.cdc.gov/nchs/data-visualization/mortality-trends/index.htm

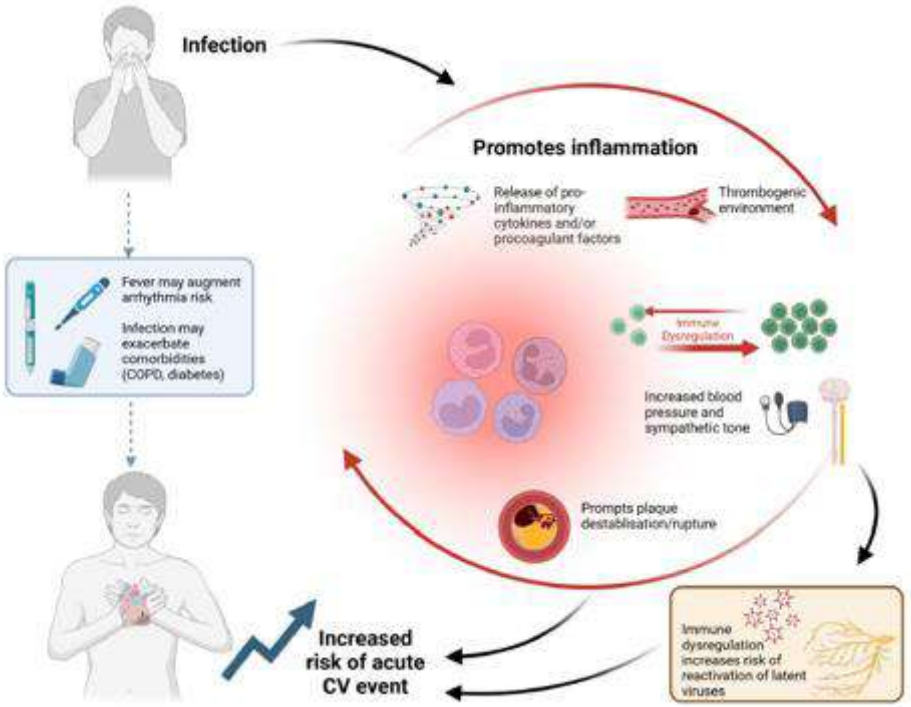
ESC European Society of Cardiology
Cardiovascular Research (2025) 121, 1330–1344
https://doi.org/10.1093/cvr/cvaf092
REVIEW

Systematic review and meta-analysis of respiratory viral triggers for acute myocardial infarction and stroke



doi: 10.1093/cvr/cvaf092. PMID: 40570137

ENFEKSİYONLAR SIRASINDA KARDİYOVASKÜLER OLAY RİSKİNİN ARTMA MEKANİZMALARI



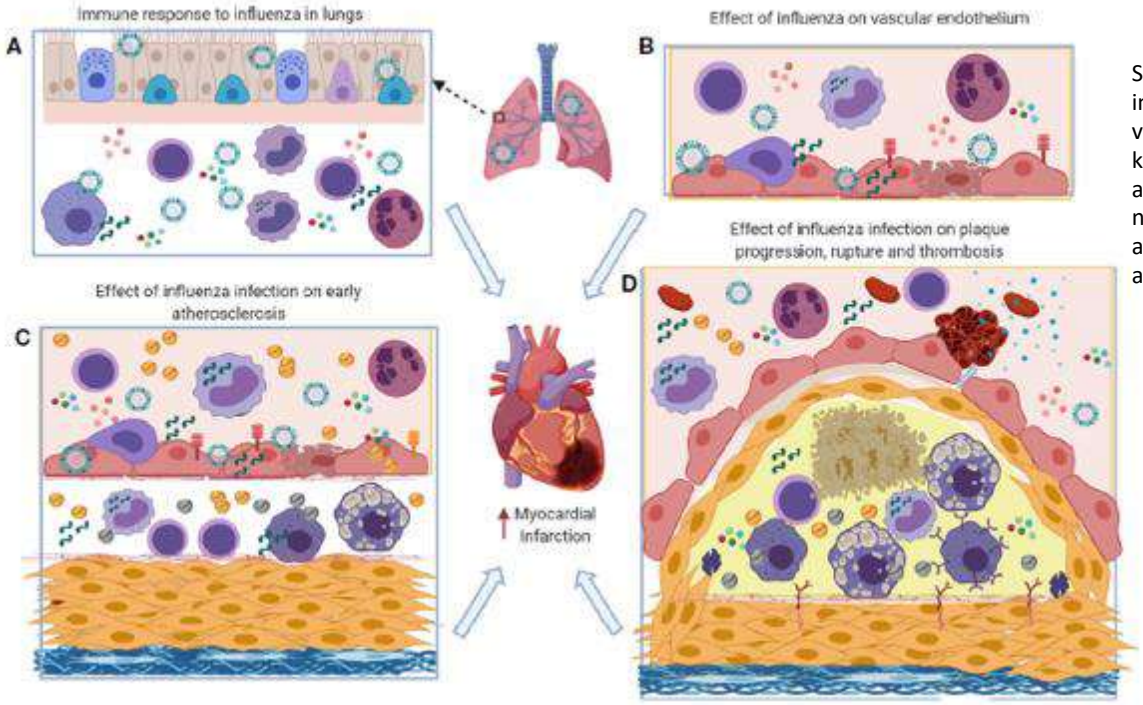
- Başta solunum yolları enfeksiyonları olmak üzere, akut enfeksiyonlar sırasında (ör. influenza, RSV, COVID-19, pnömoni) **immün sistem uyarılır**: pro-inflamatuvar sitokinlerin ve pro-koagülasyon faktörlerinin üretimi, endotel fonksiyonlarının bozulması, trombojenik ve hiperkoagubilite ortamın oluşumu gözlenir.
- Bu durum **sistemik inflamasyona yol açar**. Oluşacak immün aksama, yüksek tansiyon & sempatik tonusa neden olur.
- Bu gelişmeler inflamatuvar ortam ve plaklarda hasar ile kardiyovasküler sorunları beraberinde getirir.
- Ayrıca enfeksiyona bağlı ateş, aritmi riskini arttırır; KOAH, astım, diyabet gibi komorbiditeleri alevlendirir
- İmmün sistemdeki aksama, latent virüslerin reaktivasyon olasılığını arttırır

Aging Clin Exp Res 2025; 37:80 https://doi.org/10.1007/s40520-025-02968-y

INFLUENZA VİRÜSÜNÜN ATEROSKLEROZ OLUŞUMUNDA ETKİSİ

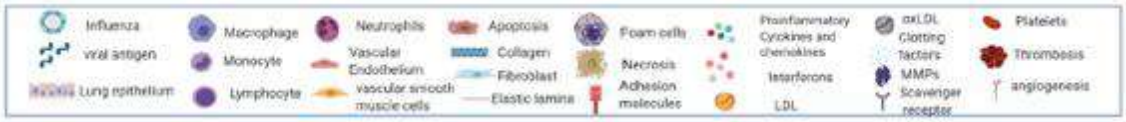
Influenza enfeksiyonu sırasında, immün yanıt sürecinde IFN ve diğer mediatörler salgılanır; makrofajlar, nötrofiller, NK'lar enfeksiyon bölgesine çekilirler.

Bu hücrelerin abartılı artışı & inflamatuvar sitokin üretiminin dengesiz seyretmesi enfeksiyon sürecinde patolojik yanıt oluşumuna yol açar



Sistemik ve lokal IFN ve pro-inflamatuvar sitokin artışı, vasküler endotel hücrelerinde kemotaktik faktörlerin ve adhezyon moleküllerinin artışına neden olur; bu durum aterosklerozda inflamatuvar hücre akışını arttırır

Influenza virüsünün uyardığı inflamatuvar mediatörler: köpüksü hücre artışına, düz kas hücre aktivasyonuna, plak bozulmasına, ve tromboza yol açarak aterosklerozu uyarır, akut MI'ne neden olur

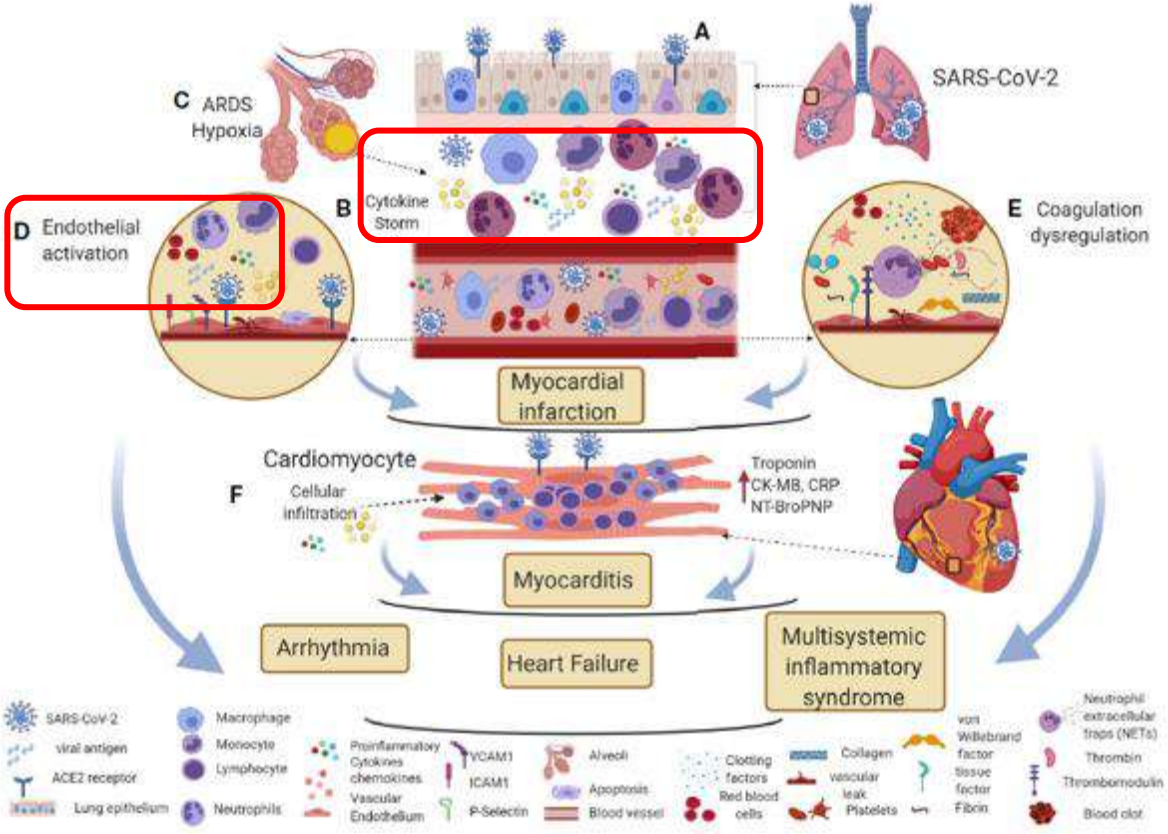


Front Immunol 2020;11: article 570681

SARS-CoV-2'ye bağlı kardiyovasküler sorunların olası immün mekanizmaları

Sitokin fırtınası vasküler geçirgenliği arttırır; pro-koagülasyon yollarını uyarır; solunum yetmezliğine neden olur

Virüs ve uyardığı mediyatörler MI riskini arttırır; tüm bu gelişmeler aritmiye, kalp yetmezliğine, miyokard inflamasyonuna neden olur

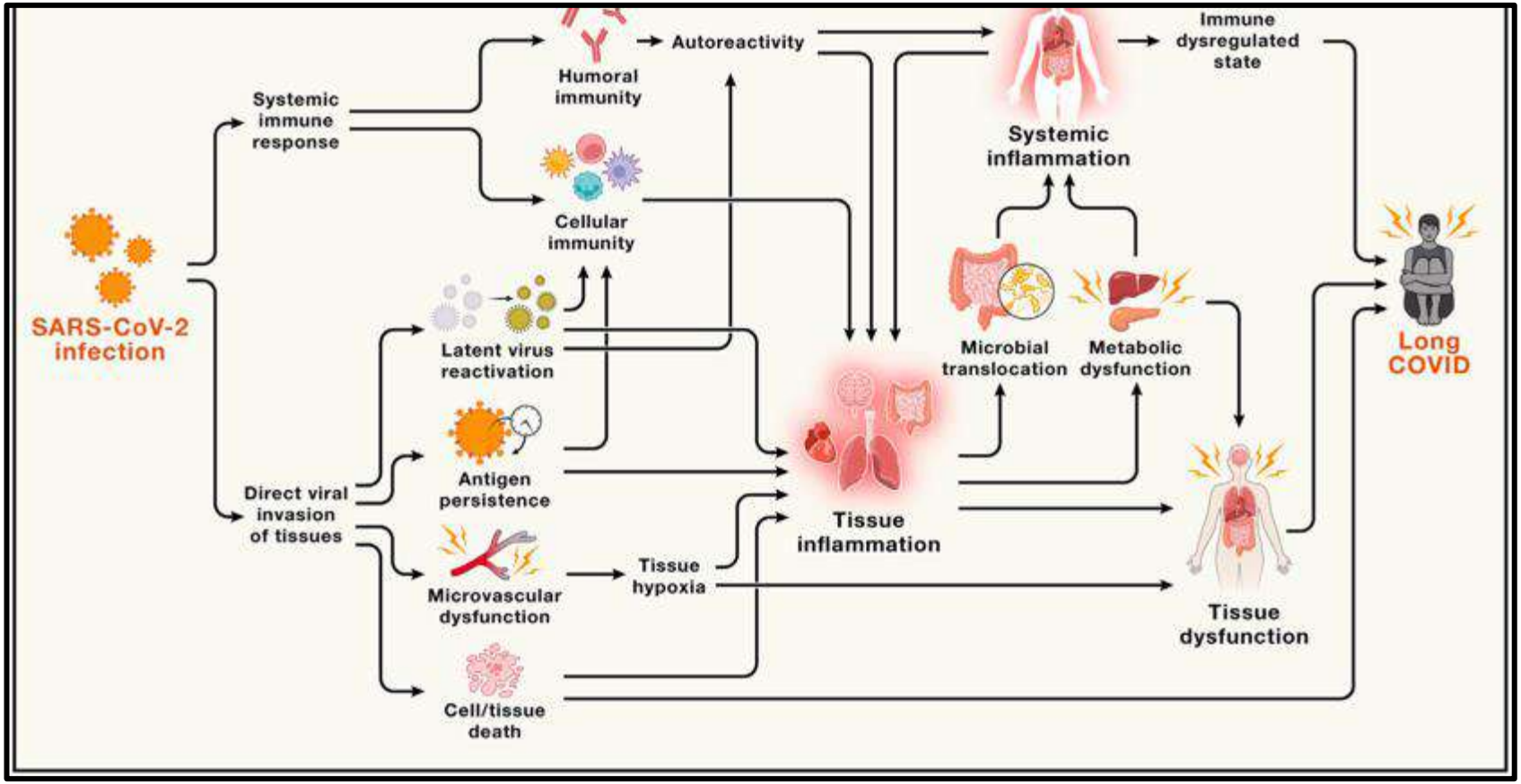


Virüsün ve mediatörlerin etkisi ile endotel adhezyonunun aktivasyonu olur

Sitokinler, hipoksi, endotel aktivasyonu COVID-19 hastalarında MI' ye neden olur

Front Immunol 2020;11: article 570681

- İnfluenza, SARS-CoV-2 (COVID-19), pneumokok ve herpes zoster (shingles) aşılarının kalp krizi ve inme riskini anlamlı olarak azaltmaktadır
- 18 yaş üzeri bireylerde herpes zoster aşılaması KVO riskini %18 azaltır
- Influenza ve pnömokok aşıları özellikle KAH olan hastalara önerilir, KV komplikasyonları ve mortaliteyi azaltır
- Aşılara atfedilen koruma infeksiyonları önleyerek inflamasyonu ve akut kardiyovasküler olayları (MI ve inme) önlemesiyle ilişkilidir
- Bu nedenle, aşılar, enfeksiyon kaynaklı kardiyovasküler stres ve inflamasyonu önleyerek kalp krizi sıklığını azaltmak için kritik bir değiştirilebilir faktör olarak giderek daha fazla kabul görmektedir.



Michael J. Peluso, and Steven G. Deeks, Cell 187, October 2024

Risk faktörleri


Kadın cinsiyet


Pre-omicron varyantlar


40-65 yaş


Re-infeksiyon


Komorbiditeler


Ağır hastalık

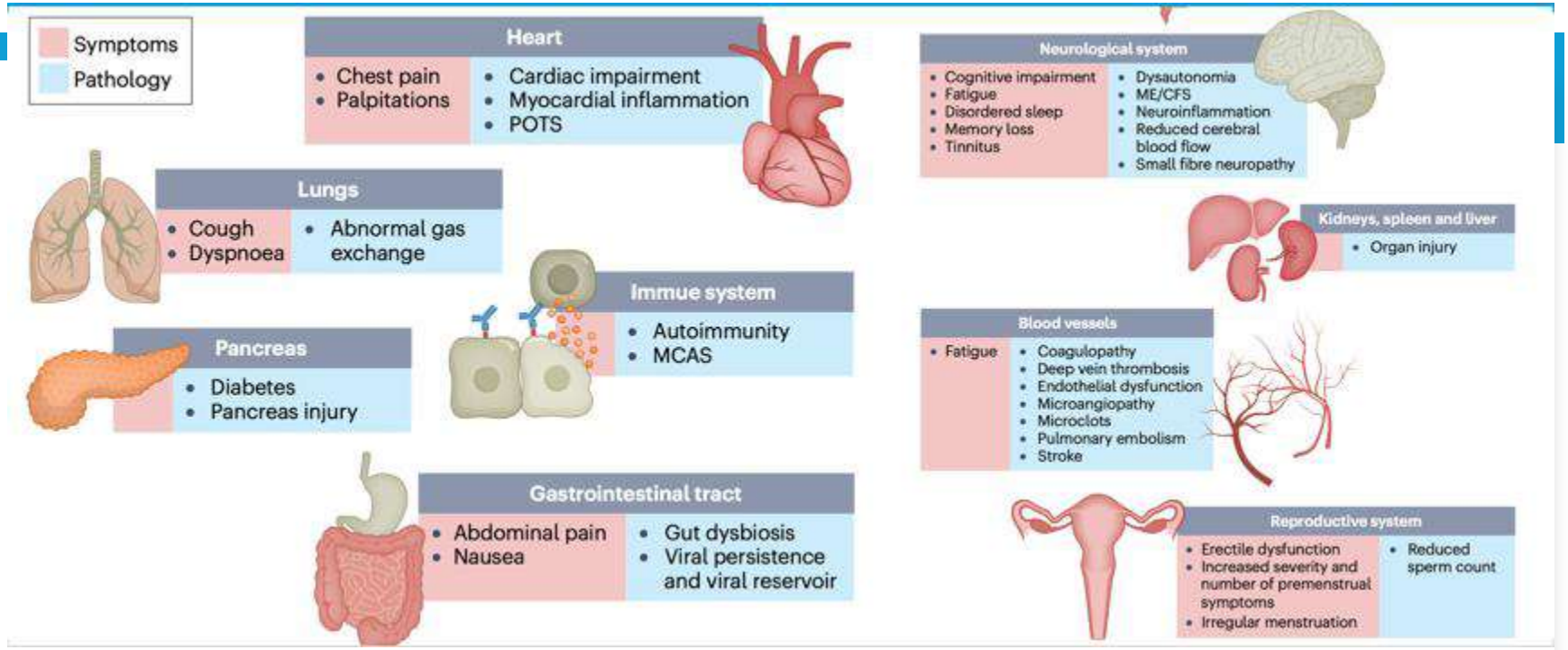

Düşük sosyoekonomik durum


Genetik faktörler

- Pandemi öncesi genel ruh sağlığının kötü olması
- Aşılamama veya eksik aşılanma
- Erken dönem antiviral tedavi kullanılmaması

Michael J. Peluso, and Steven G. Deeks, Cell, October 2024 Trisha Greenhalgh et al.Lancet,2024 Thompson, E.J., et al.Nat. Commun.2022 Tsampasian, V. Et al.JAMA Intern. Med.,2023

Klinik bulgular



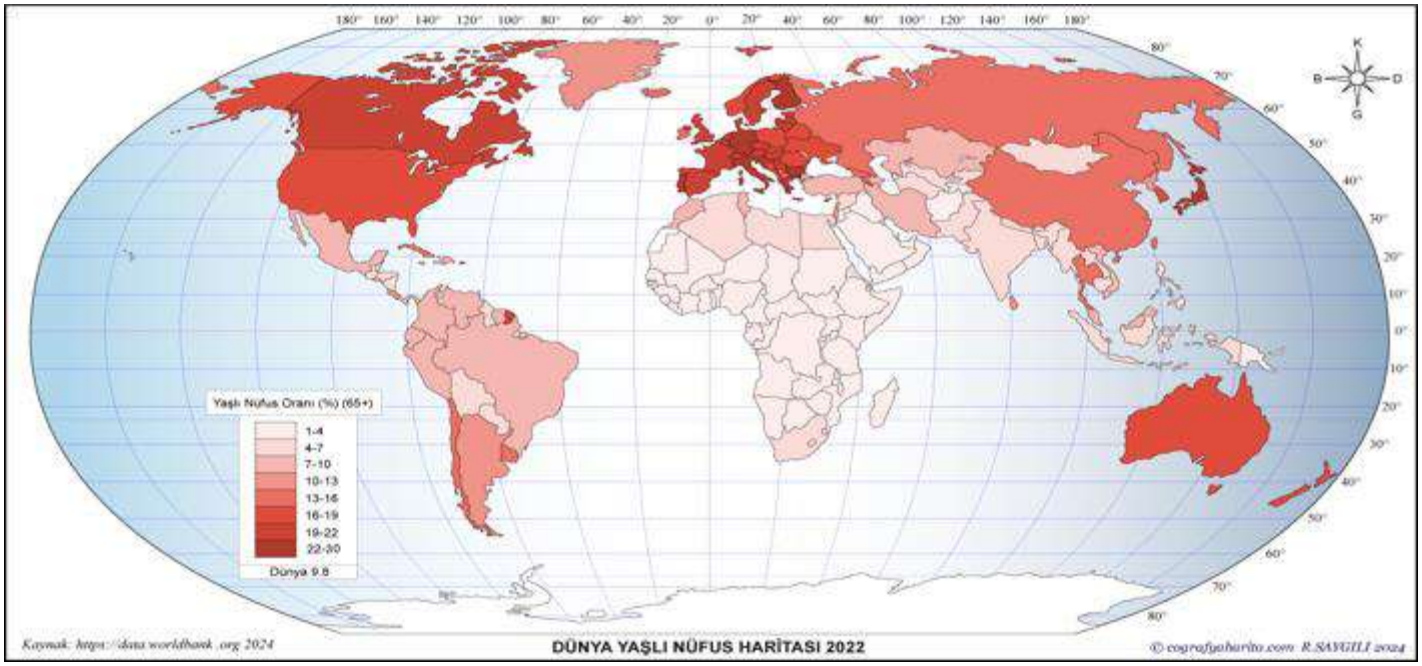
Hannah E. Davis et al. Nature Reviews Microbiology,2023; 133–146

ÇAĞIMIZIN, UYGARLIĞIN VE BELKİ DE İNSAN TÜRÜNÜN GELECEĞİ AÇISINDAN BİR YOL AYRIMINDA BULUNMAKTAYIZ...
İZLEYECEĞİMİZ YOL HANGİSİ OLURSA OLSUN,ALIN YAZIMIZ KAÇINILMAZ BİR ŞEKİLDE BİLİME BAĞLIDIR...

VAR OLMAK, HAYATTA KALABİLMEK İÇİN BİLİM VAZGEÇİLEMEYECEK BİR TEMEL GEREKSİNİMDİR

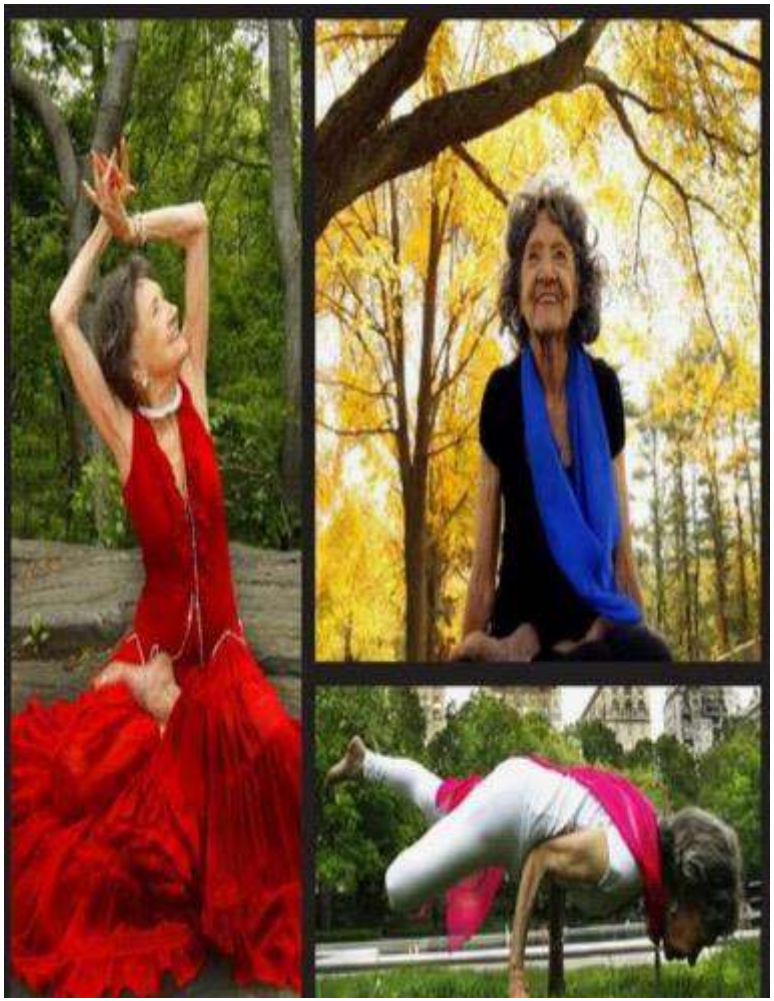
CARL SAGAN /KOZMOZ

Dünya Yaşlı Nüfus Haritası ve Türkiye



- Türkiye’de nüfusun %11’i 65 yaş ve üzerinde. Bu sayı hızlanarak artacak.

Tao Porchon-Lynch:101 yaş



Box 1. Immunosenescence*.

In contrast to younger individuals, hematopoietic stem cells (HSCs) in older subjects show a greater differentiation into myeloid progenitors at the expense of lymphoid progenitors than in younger individuals [5].
This results in fewer circulating antigen presenting cells (APCs) and a relative shift in the proportions of naïve and memory cells within both B and T-cell populations (fewer naïve cells and a corresponding increase in memory T and B cells) [5,6].
As a result, overall antibody production and specificity is diminished. It is this shift which leads to impaired immunity against infection in older people, and also to reduced vaccine efficacy and shorter duration of vaccine response [5].
A general increase in pro-inflammatory cytokine levels is also seen (in particular IL-6 and TNF-α), secreted by aging HSCs, and by other tissue cells as part of a more generalized cellular senescence [4,5].

*It is important to realize that substantial variation exists in the relative contribution and extent of these mechanisms in individuals, and no direct correlation with specific chronological ages can be made.

1 October 2022

Key facts

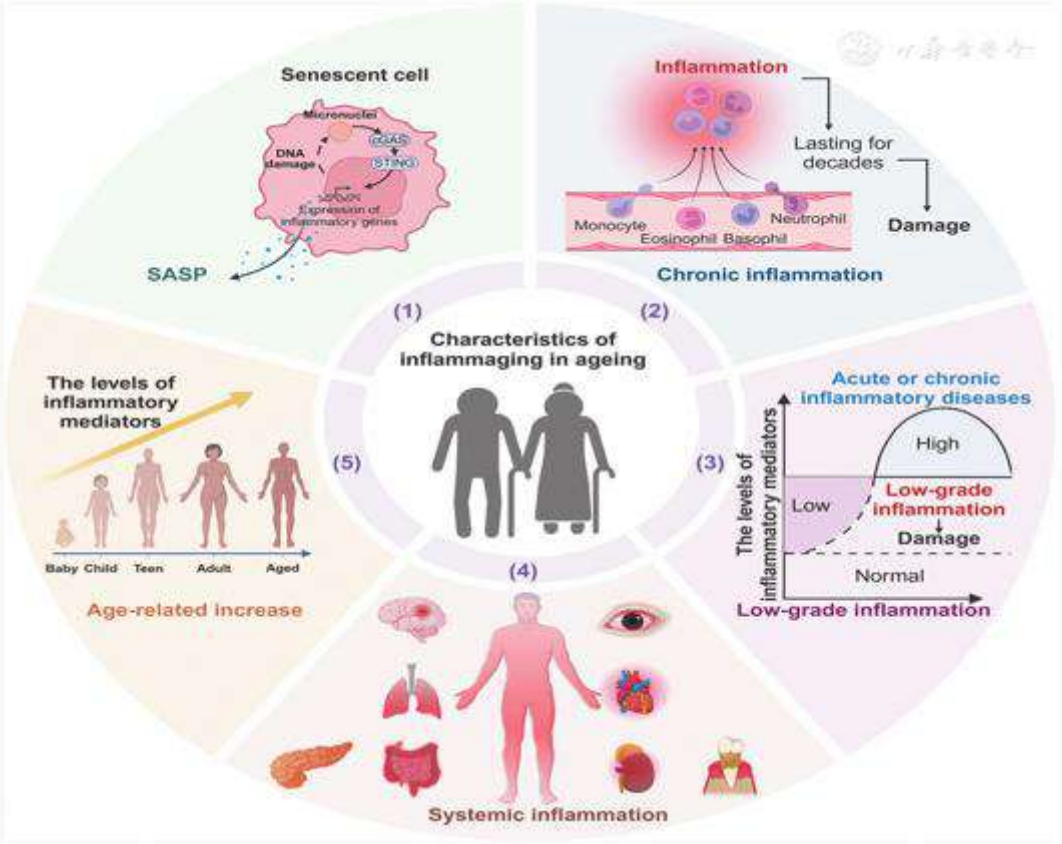
- All countries face major challenges to ensure that their health and social systems are ready to make the most of this demographic shift.
- In 2050, 80% of older people will be living in low- and middle-income countries.
- The pace of population ageing is much faster than in the past.
- In 2020, the number of people aged 60 years and older outnumbered children younger than 5 years.
- Between 2015 and 2050, the proportion of the world's population over 60 years will nearly double from 12% to 22%.

MMUNE AGING: CAN WE SLOW DOWN THE PROCESS?

- İnfluenza,
- Pnömokok infeksiyonları,
- RSV (Respiratuvar Sinsityal Virüs),
- Herpes zoster (zona)
- Covid-19

Yaşlılık ve İnfeksiyon Yükü:

- Bağışıklık Yaşlanması
- İnfeksiyon sıklığı
- “inflamm-aging”
- Sistemik ve nörolojik etkiler



Chronic viral infections and senescence

Viral infections can trigger a wide range of host cellula
esponses that mirror key hallmarks of cellular senes
ence. In particular, persistent viral infections have bee
hown to promote cellular aging through mechanism
uch as the induction of cell cycle arrest, accumulatio
f DNA damage, and sustained secretion of proinflam
natory cytokines [43]. These processes not only impai
ormal cellular function but also contribute to the deve
pment of a senescent phenotype. Viruses manipulat
ost cellular pathways to support their persistence, but a
consequence of this, they induce senescence-associate
henotypes, marked by DNA damage, mitochondria
lysfunction, and secretion of proinflammatory SAS
actors, which can accelerate tissue degeneration an
ystemic aging. This persistent senescent state not onl
acilitates viral survival but also exacerbates age-relate
omorbidities and immune exhaustion in infected ind
iduals. In this context, we explore how certain viruses
articularly those capable of evading the immune syster
nd establishing chronic infections, can drive senescenc
n host cells.

Compared with healthy individuals, chronic hepat
is C virus (HCV) infection gradually damages the live
nd promotes hepatocellular senescence through th
pregulation of key cell cycle inhibitors, including p1
21, and p27 in patients chronically infected with HC
44]. Mechanistically it is not clear how chronic HC
nfection leads to upregulation of cell cycle inhibitor
luring chronic infection. An in vitro study using huma
epatoma cells showed that HCV core protein can down
egulate p16 expression via promoter hypermethylatio
nediated by the upregulation of DNA methyltransferas
(DNMT1) and DNMT3B, ultimately enhancing ce

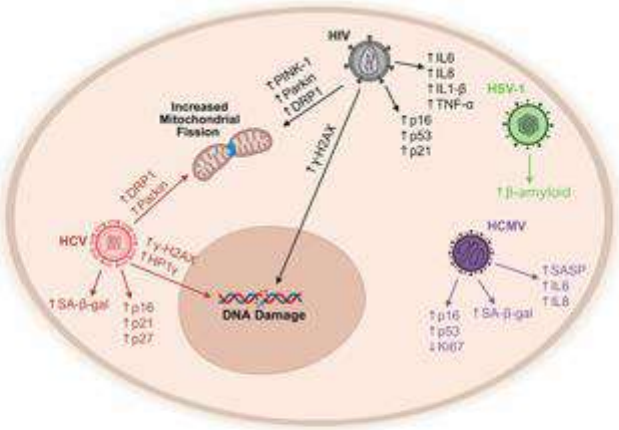


Fig. 1 Senescent phenotypes in viral infections. HCV increases the levels of the senescence marker SA-β-Gal, cell cycle inhibitors (p16, p21, p27), and DNA damage, resulting in increased γ-H2AX and HP1γ and mitochondrial damage via increased DRP1 and Parkin. HIV increases the levels of proinflammatory cytokines (IL-6, IL-8, IL-1β, and TNF-α) and DNA damage, resulting in increased γ-H2AX, increases in the levels of cell cycle inhibitors (p16, p53, and p21), and mitochondrial damage resulting from increased DRP1, PINK-1, and Parkin. HSV-1 results in increased accumulation of β-amyloid in neuronal cells, leading to Alzheimer's disease pathology. HCMV leads to increased senescence markers (p16, p53, Ki67, and SA-β-Gal) and increased SASP factors (IL-6 and IL-8). Figure created by BioRender

REVIEW Open Access

The impacts of chronic infections on shaping cellular senescence

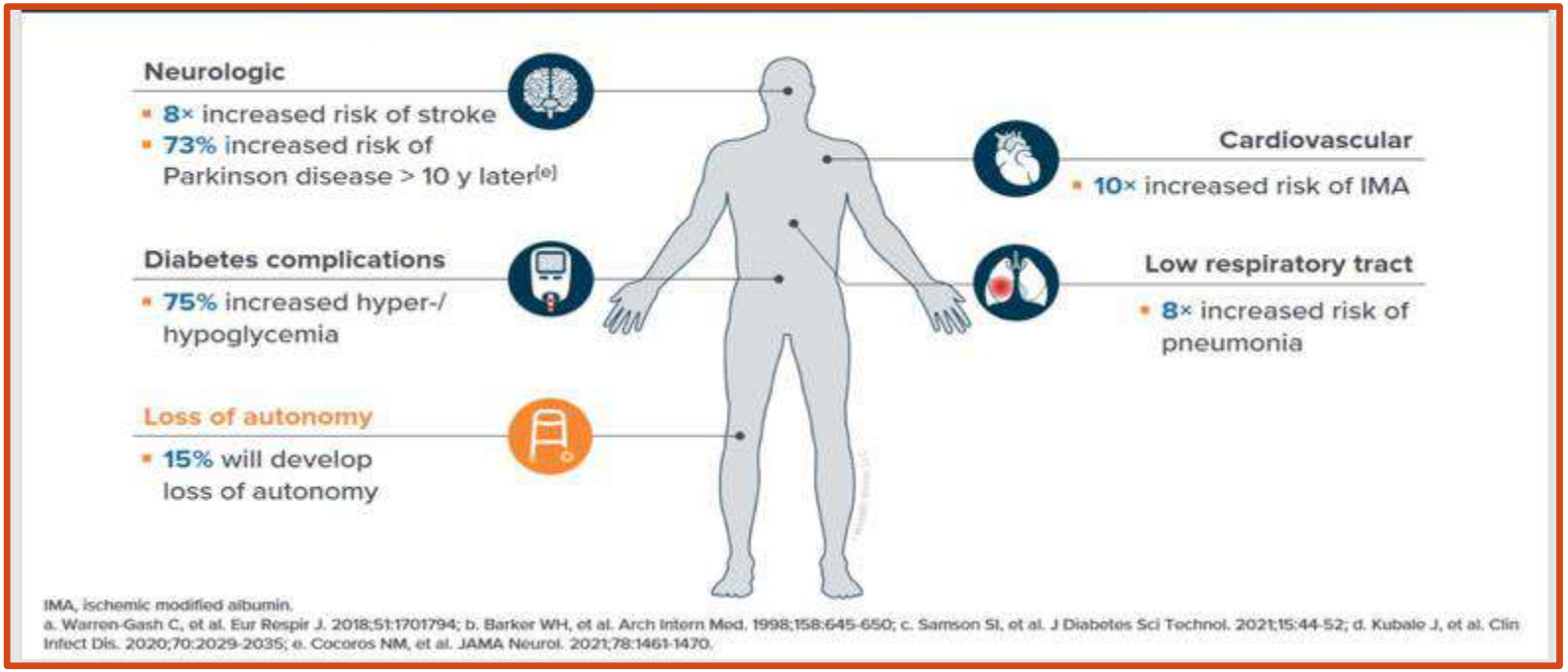
Ashley Johnson[1†], Troy Rought[1†], Jacob Aronov[2], Princess Pokharel[2], Angelina Chiu[2] and Azadeh Nasuhidehnavi[1*]

Abstract

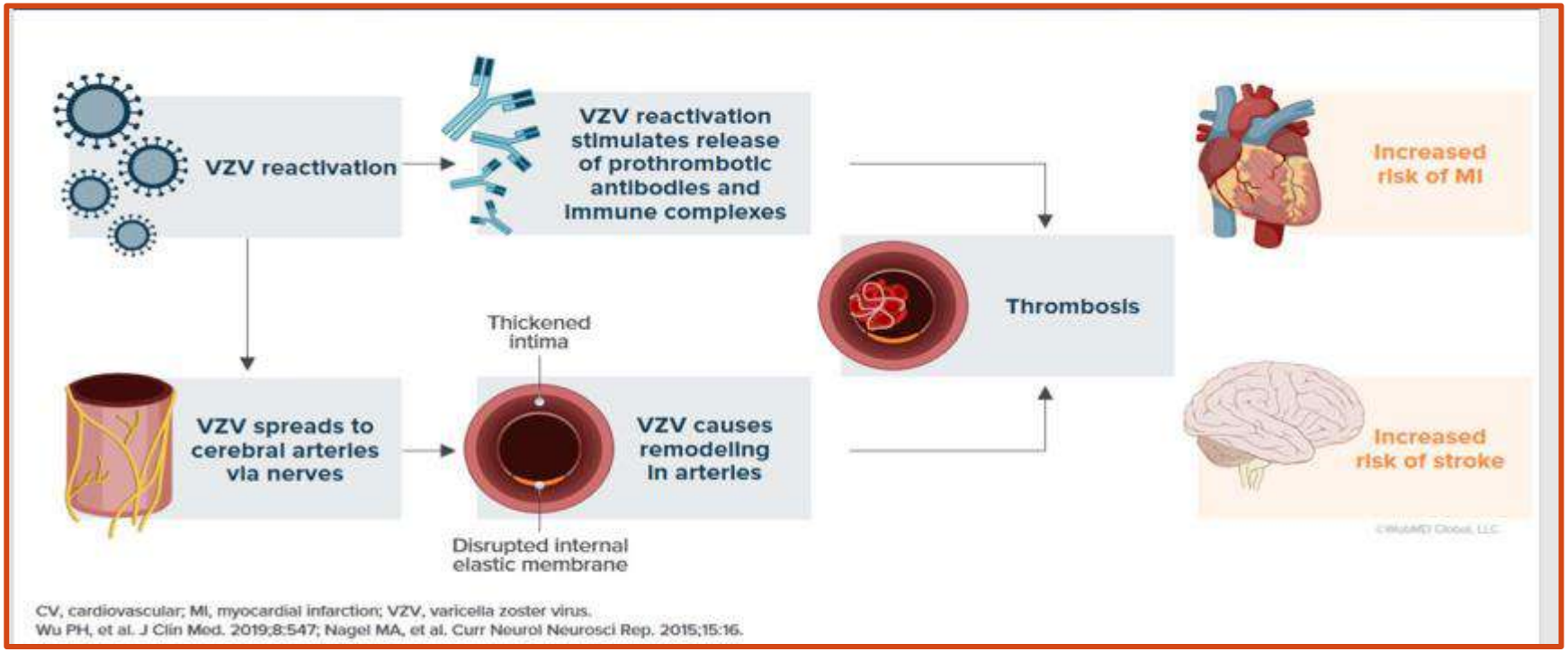
Cellular senescence is a fundamental biological process characterized by stable cell cycle arrest, genomic instability, and the acquisition of a proinflammatory secretory phenotype. While senescence is traditionally associated with aging, growing evidence reveals that chronic infections such as viral, bacterial, and protozoan parasites can serve as powerful inducers of senescence, contributing to premature aging and long-term tissue damage. This review explores the diverse mechanisms by which persistent pathogens trigger or sustain senescence in host cells. We highlight how these chronic infections manipulate host DNA repair, mitochondrial dynamics, telomere maintenance, oxidative stress, and immune function to promote senescence and immunosenescence. Emerging findings also reveal how pathogens hijack the host cellular machinery to induce senescence across various tissue types. In many cases, senescence not only enables pathogen persistence but also drives pathological outcomes such as fibrosis, neurodegeneration, cardiomyopathy, and immune exhaustion. Collectively, this emerging evidence highlights a unifying strategy among diverse pathogens: the exploitation of cellular senescence to support chronic infection and promote disease. Understanding how infectious agents drive senescence offers new insights into age-related pathologies and highlights potential therapeutic targets, such as senolytic and senomorphic agents, to mitigate the long-term impacts of chronic infections.

Keywords Senescence, Chronic infection, Viral infection, Bacterial infection, Protozoan parasites

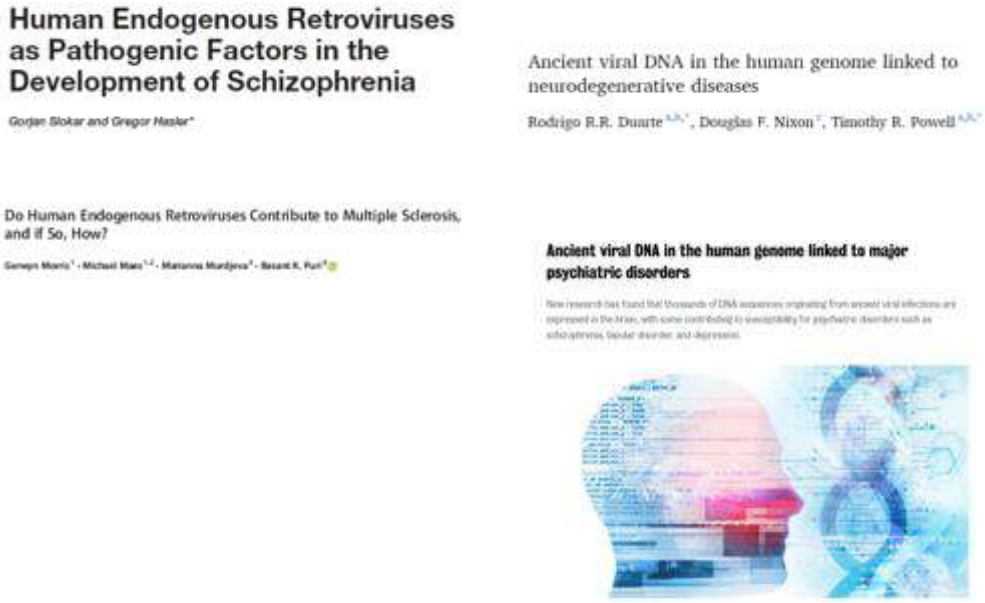
İNFLUENZANIN SOLUNUM SİSTEMİ DIŞINDAKİ BULGULARI



HERPES ZOSTER REAKTİVASYONU-VASKULOPATİ



CV, cardiovascular; MI, myocardial infarction; VZV, varicella zoster virus.
Wu PH, et al. J Clin Med. 2019;8:547; Nagel MA, et al. Curr Neurol Neurosci Rep. 2015;15:16.



Human Endogenous Retroviruses as Pathogenic Factors in the Development of Schizophrenia

Gorjan Stokar and Gregor Hasler*

Ancient viral DNA in the human genome linked to neurodegenerative diseases

Rodrigo R.R. Duarte [a,b,*], Douglas F. Nixon [c], Timothy R. Powell [a,b,*]

Do Human Endogenous Retroviruses Contribute to Multiple Sclerosis, and if So, How?

Ancient viral DNA in the human genome linked to major psychiatric disorders

- Front Psychiatry 2016;6: 183; Brain, Behavior, and Immunity 2025;123: 765–770; Mol Neurol 2019;56: 2590

Esin Davutoğlu Şenol

Beynimize hasar veren virüsler

Virüsler, canlılık çeşitliliğinde ekosisteme en büyük etkiyi yapan insan türüne eşlik ederken evcilleşmiyor çünkü daima evriliyor...

Bilim

ZONA VE DEMANS

Yeni bir çalışma, İtalya'da 10 milyonluk bir nüfusta 20 yıl boyunca yapılan eşleştirilmiş kohort çalışması Zona'nın, demans için bir risk faktörü olduğunu gösterdi. Heyecan verici bir buluş da, Galler'de bir halk sağlığı politikası olarak zona aşısının uygulanması sonucunda, zona aşısı olanların sonraki yedi yıl içinde demans olma olasılığının, aşı olmayanlara göre %20 daha az olduğunun güçlü bir yöntemsel çalışma ile gösterilmiş olmasıydı. Bu aynı zamanda bir aşının ve aşı ile önleyici halk sağlığı yaklaşımlarının, uzun ve bedensel, zihinsel, ruhsal bakımdan sağlıklı bir yaşam için önemini ortaya koyan güçlü bir kanıttı.

Beyinde küçülme, erken beyin yaşlanması, zeka kalsiyumun azalması daha beyinde önemli etkileri gösterilen COVID-19' un pandemik fırtınasını yaşıyoruz aşılar, uzamış COVID ve beyindeki etkilerini önlemede yeterli mi sorusunun net bir cevabı yok. Çünkü maalesef COVID-19 etkeni olan virüs, sonekli evrilerek aramızda ve aşı bir stratejik yaklaşım olmaktan çok bireysel tercihlere indirgendi. Benim" tam bir biyolojik kaçıcı" dediğim virüs, nörolojik hastalıklarla ilgili öngörülerden daha fazla bir yük getirebilir.

'Virüsler, canlılık çeşitliliğinde ekosisteme en büyük etkiyi yapan insan türüne eşlik ederken evcilleşmiyor çünkü daima evriliyor. ...Ünlü bir virolog olan Vincent Racaniello nun deyimiyle "yok edilemeyecek kadar sekiziler"

Bir asır kadar önce yaşanılan "İspanyol Gribi" pandemisiyle bağlantılı olan "ensefalit letharjik" ve sonrasındaki parkinsonizm* ile ilgili gözlemler, beynimizi hastalandıran virüslerle ilişkili tarihsel kökleri oluşturan bulgulardır. Parkinson en hızla artan nörokognitif hastalıklardan ve son zamanlarda, grip ve herpes simpleks (HSV), Epstein-Barr Virus (EBV), Varicella-Zoster Virus (VZV) gibi virüslerle karşılaşmış olmanın riski artırdığına ilişkin, kanıt oluşturma gücü daha yüksek çalışmalar yayımlanıyor. Tıp disiplini dışı okuyucular için, sözü geçen virüslerin, çok yaygın olan, uçuk, zona ve mononükleoz (öpüşme hastalığı diye de bilinir) hastalık etkenleri olduklarını, dipnot olarak düşelim. Parkinson'un ve Alzheimer hastalığının, COVID-19'un nörokognitif uzun dönem etkileri arasında olabileceği düşünülmekte ve çalışmalar yürütülmektedir.

Eldeki veriler virüslerin nörolojik hastalıkların tetikleyicisi ya da potansiyel nedenleri arasında olabileceğini işaret edince, tıp disiplinini destekleyen çok daha sofistike yeni tanısal yöntemler, yapay zeka destekli yaklaşımlar ile yapılan çalışmalardan heyecan verici sonuçlar gelmektedir. Henüz yayımlanmış bir çalışmada "Human pegivirus" (HPgV) adlı bir virüsün önemli bir çevresel faktör olarak Parkinson'u tetikleyebileceği veya gelişiminde, ilerlemesinde rolü olabileceğini gösteren veriler kuşkusuz daha detaylı çalışılacak, sonuç verirse tedavinin hedefleri bakımından çok önemli olacaktır. MS hastalığında önemli rolü olduğu gösterilen EBV infeksiyonu, demans ilişkisi artan veri ile desteklenen zona hastalığı, uzamış COVID ve bilişsel, zihinsel fonksiyonlardaki kayıp ilişkisi, nörolojik hastalıklarda tedavi hedeflerinin belirlenmesindeki yeni araştırmalara da yön vermektedir.

The potential for dementia prevention through vaccination

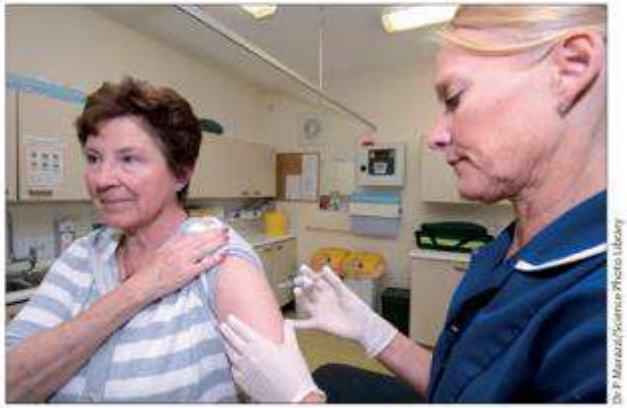
See Articles page 170

The need to prevent dementia is compelling, with 150 million people projected to be living with dementia by 2050.[1] Efforts to prevent dementia have focused on potentially modifiable risk factors that can occur throughout the life course, in particular lifestyle measures such as smoking, obesity, and physical inactivity.[2]

Growing research efforts have focused on the potential to target viral contributions to diseases that cause dementia, in particular the neurotropic herpes viruses such as varicella zoster virus and herpes simplex virus (HSV).[3] Evidence of links between these viruses and dementia have long been postulated but have been difficult to prove beyond associative studies.[3] The emerging ability to perform large-scale, quasi-experimental analyses of national vaccination programmes has facilitated insights into the effects of preventing reactivation of latent herpes zoster infection. In *The Lancet Neurology*, Michael Pomirchy and colleagues[4] provide further evidence from Canada's live attenuated single-dose vaccination programme for varicella zoster virus that targeting varicella zoster infection could prevent dementia.[4]

Canada is a nation with strong health-data linkage, which provides the foundation for a natural experiment based on the distinct age cutoffs for zoster vaccine eligibility. For example, in Ontario, individuals with their 71st birthday after Jan 1, 2017, were eligible for publicly funded vaccination, but those with birthdays before were not. By studying the incidence of dementia in groups either side of the cutoff, controlling for several inherent biases in observational studies is possible given the allocation for eligibility is effectively random.

Analysing a cohort of over 250 000 individuals in Ontario, Pomirchy and colleagues[4] compared dementia incidence in the population born immediately before the threshold for eligibility with the population born immediately after. They found that being born in the month immediately before the cutoff decreased the probability of being diagnosed with dementia by 2·0 percentage points (95% CI 0·4–3·5; p=0·012) compared with being born immediately after. The only date-of-birth thresholds that were associated with a significant decrease in incident dementia were



Dr P Marazzi/Science Photo Library

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IOS Press

Short Communication

Association of COVID-19 with New-Onset Alzheimer's Disease

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Abstract. An infectious etiology of Alzheimer's disease has been postulated for decades. It remains unknown whether SARS-CoV-2 viral infection is associated with increased risk for Alzheimer's disease. In this retrospective cohort study of 6,245,282 older adults (age ≥65 years) who had medical encounters between 2/2020–5/2021, we show that people with COVID-19 were at significantly increased risk for new diagnosis of Alzheimer's disease within 360 days after the initial COVID-19 diagnosis (hazard ratio or HR:1.69, 95% CI: 1.53–1.72), especially in people age ≥85 years and in women. Our findings call for research to understand the underlying mechanisms and for continuous surveillance of long-term impacts of COVID-19 on Alzheimer's disease.

Keywords: Alzheimer's disease, COVID-19, electronic health records, viral etiology

scientific reports

OPEN

Cognitive impairment 2 years after mild to severe SARS-CoV-2 infection in a population-based study with matched-comparison groups

Natália Araújo[1,2], Isa Silva[1], Patrícia Campos[1,3], Adriana Costa[1,2], Catarina Lopes[1,2], Mariana Seco[4], Ana Rute Costa[1,2], Maria Margarida Calejo[4], Maria Joana Pais[1], Susana Pereira[1,5], Samantha Morais[1], João Firmino Machado[1,6], Luís Ruano[1,7], Nuno Lunet[1,2] & Vítor Tedim Cruz[1,4]

COVID-19 may have long lasting cognitive consequences. Studies with a follow-up longer than 1 year after infection are lacking. This study presents the prevalence of cognitive impairment 2 years after SARS-CoV-2 infection in survivors of the first year of the pandemic and comparison groups matched 1:1 for sex, age, and level of care. Users of the Local Health Unit of Matosinhos (comprising almost all citizens of the municipality) were retrospectively selected according to hospitalization and SARS-CoV-2 infection between March 2020 and February 2021: group #1, hospitalized for COVID-19 (n = 101); group #2, hospitalized, uninfected (n = 87); group #3, non-hospitalized, infected (n = 252); group #4, non-hospitalized, uninfected (n = 258). Between July 2022 and October 2023, all participants completed the Montreal Cognitive Assessment. Those with a score below 1.5 SD of age- and education-specific norms (n = 279) were invited for a comprehensive neuropsychological assessment to identify cognitive impairment. The prevalence of cognitive impairment was higher in group #1 than #2 (19.1% vs. 6.8%; adjusted OR 5.41, 95% CI 1.54, 19.03) and in group #3 than #4 (10.7% vs. 3.2%; adjusted OR 3.27, 95% CI 1.23, 8.67). These results suggest that specific care to timely diagnose and treat cognitive impairment is needed for COVID-19 survivors of the first year of the pandemic.

Keywords Cognitive impairment, SARS-CoV-2 infection, COVID-19, Neuropsychological assessment

COVID-19 no longer constitutes a public health emergency of international concern[1]. However, a significant proportion of the population was exposed to COVID-19 in the first year of the pandemic. In this period, individual and group immunities to the disease were low and SARS-CoV-2 infection caused severe disease more frequently[2].

Previous studies have reported higher risks of neurodegenerative diseases following the acute phase of SARS-CoV-2 infection compared to SARS-CoV-2-uninfected individuals[3]. These risks were even higher in the most severe cases of COVID-19 and in unvaccinated individuals[4]. Conversely, deficits in cognitive tests were reported to be higher in individuals with COVID-19 post-acute sequelae persistent for more than 12 weeks than in SARS-CoV-2-uninfected individuals. Moreover, larger deficits were noticed in survivors of COVID-19 who had been hospitalized and whose infection occurred in periods when original and alpha variants were dominant[5]. However, methodological issues have limited our understanding of the effect of COVID-19 on cognitive function, specifically after a period of more than 1 year following infection. Indeed, the validity of previous results is

Neuropsychiatric Complications of COVID-19

- Anosmia
- Dysgeusia
- Headache
- Myalgias
- Seizure
- Stroke
- Encephalopathy/Encephalitis
- Guillain-Barré syndrome (and variants)
- Transverse myelitis
- Acute disseminated encephalomyelitis (ADEM)
- Primary psychiatric disorders

- Zubair AS, et al. JAMA Neurol 2020
- Koralnik IJ, Tyler KL. Ann Neurol. 2020
- Solomon IH, et al. N Engl J Med. 2020
- A. Varatharaj et al., Lancet Psychiatry 2020

REVIEW OPEN ACCESS

Epstein-Barr Virus in Multiple Sclerosis

Past, Present, and Future

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Neurol Neuroimmunol Neuroinflamm 2025;12:e200460. doi:10.1212/NXI.0000000000200460

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Supplementary Material

Abstract

Epstein-Barr virus (EBV) is a very common herpesvirus that infects more than 90% of the general population. Epidemiologic data indicate that EBV is a requisite risk factor for the development of multiple sclerosis (MS); however, the mechanisms by which EBV contributes to MS pathogenesis are unclear. In this review, we discuss how EBV alters the functions of B cells, its primary cellular reservoir, and the associated dysregulation of anti-EBV immunity in patients with MS. We comprehensively explore the evidence for different potential mechanisms by which EBV may lead to the development of MS, including the so-called driver and hit-and-run models. Finally, we discuss key outstanding scientific questions that must be addressed to advance not only our understanding of the role of EBV in MS pathology but also the development of novel disease therapies.

Introduction

Epstein-Barr virus (EBV), also known as human herpesvirus 4, is a ubiquitous gamma herpesvirus that infects most of the global population. Although primary infection is often asymptomatic, EBV has been linked to a broad range of diseases, including infectious mononucleosis, lymphoproliferative disorders, lymphomas, and certain autoimmune diseases such as multiple sclerosis (MS).[1]

Global burden of cancer attributable to infections: the critical role of implementation science

See Articles page e180

Despite significant advances in methodology and data quality since the first estimation of the global burden of cancer attributable to infections, one key fact remains: infections are responsible for at least a sixth of all cancer cases worldwide.[1–4] In the latest set of estimates in this issue of *The Lancet Global Health*, Catherine de Martel and colleagues[5] use newly released 2018 GLOBOCAN cancer incidence data to estimate for the first time age-standardised incidence rates of infection-attributable cancers at the country, regional, and global level. While many sources of uncertainty remain (including global variations in data completeness, quality of case detection and inclusion in cancer registries, and challenges in estimation of population denominators), incidence rates, as opposed to attributable fractions, provide policy makers with a more actionable metric of the burden of infection-associated cancers within and between populations over time. De Martel and colleagues report an astounding 25 cases of infection-attributable cancer per 100 000 people in 2018, with global variation ranging from 38 and 33 cases per 100 000 in eastern Asia and sub-Saharan Africa, respectively, to 14 cases per 100 000 in northern Europe and western Asia.

Equally as important as the magnitude of the public health burden highlighted in the Article by de Martel and colleagues is the emphasis they place on the ever-improving landscape of evidence-based interventions to combat this persistent, yet preventable, fraction of the global burden of cancer. The four main oncogenic agents responsible for approximately 90% of the infection-attributable cancer cases worldwide—*Helicobacter pylori*, human papillomavirus (HPV), hepatitis B virus (HBV), and hepatitis C virus (HCV)—are either vaccine-preventable (HPV, HBV) or treatable (*H pylori*, HCV) infections, and all are amenable to some level of behavioural intervention focused on reducing infection transmission. Consideration of the role of HIV as a co-factor in the incidence (and mortality) of many infection-associated cancers—currently not taken into account in the estimates by de Martel and colleagues—further expands our opportunities to reduce the burden of infection-associated cancers through coordinated HIV prevention and early treatment strategies.

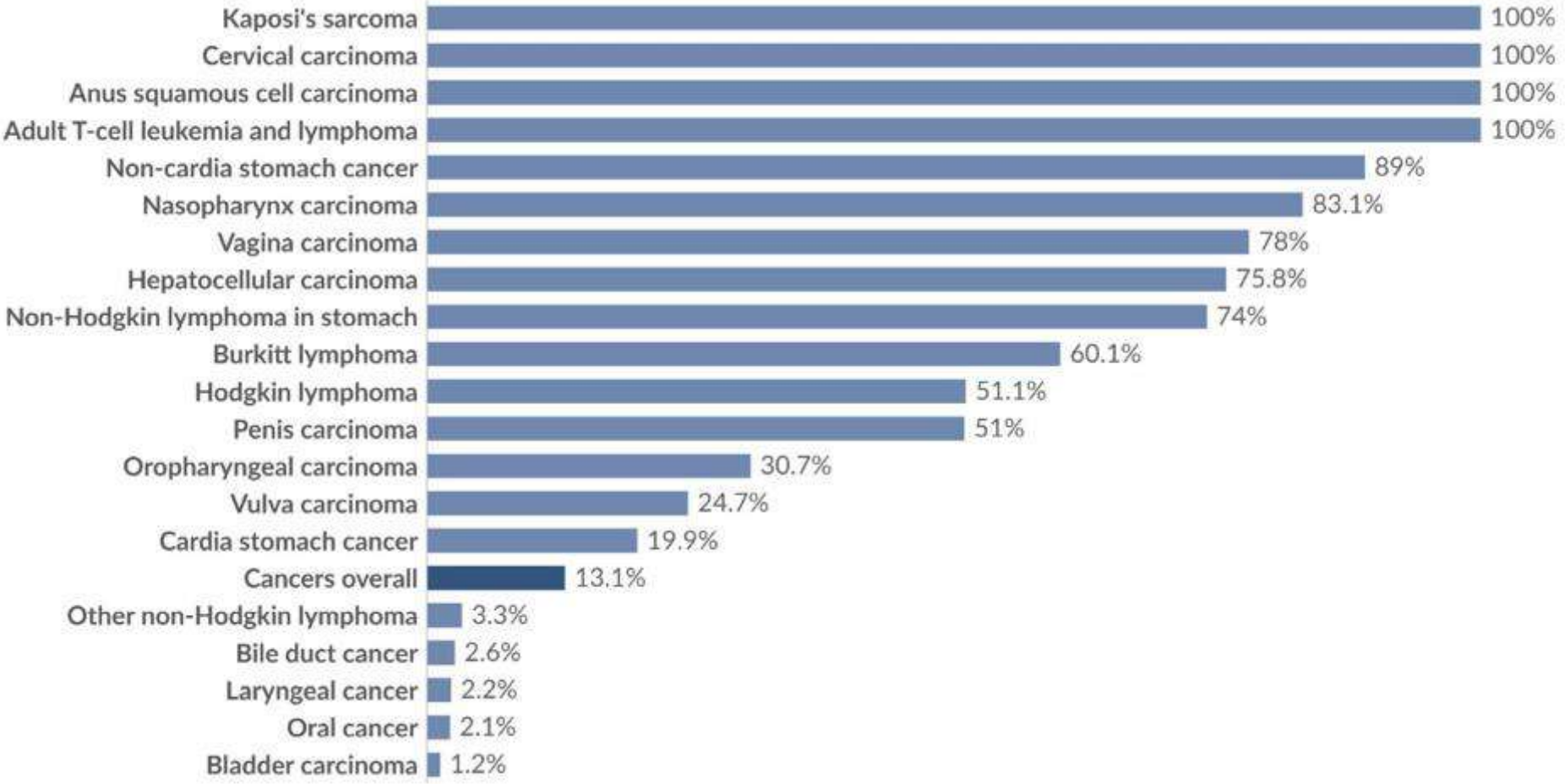
The pressing question to the global community of researchers, policy makers, health-care delivery specialists, public health programmers, and clinicians now is how can we accelerate sustainable implementation of these evidence-based interventions such that the sixth version of the global burden of infection-associated cancer estimates will reflect achievable progress? WHO recently released its plans towards ending viral hepatitis and eliminating cervical cancer.[6,7] If achieved, these combined efforts will have an enormous impact on the incidence rates of cancers attributable to HPV, HBV, and HCV. To date, great strides have been made in hepatitis B vaccination, but progress to achieve widespread coverage of HPV vaccination has been slow in many world regions[8] and more research is needed on the use of antibiotic treatment for *H pylori* in the reduction of cancer. Global disparities in cancer incidence and mortality persist not due to lack of evidence-based options for the prevention and control of these cancers, but in large part due to measurable gaps between evidence and practice in cancer control, including management of infectious diseases. It is clear, then, that meeting these calls to action will necessarily require a fundamental change in prioritisation of research efforts to foster the development of "practice-based evidence" for successful adoption and scale-up of coordinated programmes of cancer control.

Dissemination and implementation science, or implementation research, fill this critical gap by providing a formal framework to adapt evidence-based interventions for effective implementation in diverse settings and to evaluate the implementation process in order to estimate, understand, and respond to successes and failures in programme adoption, scale-up, and sustainability. Dissemination and implementation science is formally defined as the scientific study of methods to promote the systematic uptake of evidence-based practices into real-world contexts to prevent disease and improve the quality and effectiveness of health care services.[9] If our research is going to make an impact on policy and practice or inform the scale-up and sustainability of programmes—critical to vaccination and widespread treatment for infectious

Share of new cancers caused by infections, World, 2020

Our World in Data

Estimated share of new cancer[1] cases attributed to all known cancer-causing pathogens. This includes H. pylori[2], human papillomavirus[3], hepatitis B & C virus[4], Epstein-Barr virus, human herpesvirus type 8, schistosoma haematobium, human T-cell lymphotropic virus, O. viverrini, and C. sinensis.



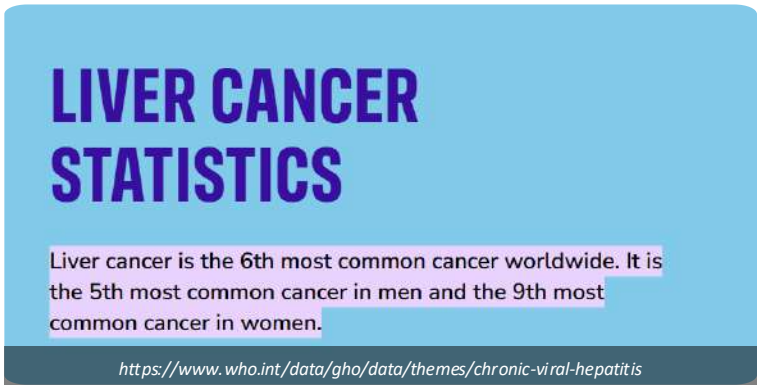
Data source: International Agency for Research on Cancer (2020)

OurWorldinData.org/cancer | CC BY

Note: Non-melanoma skin cancers[5] are excluded due to potentially incomplete records and inconsistent registry practices.

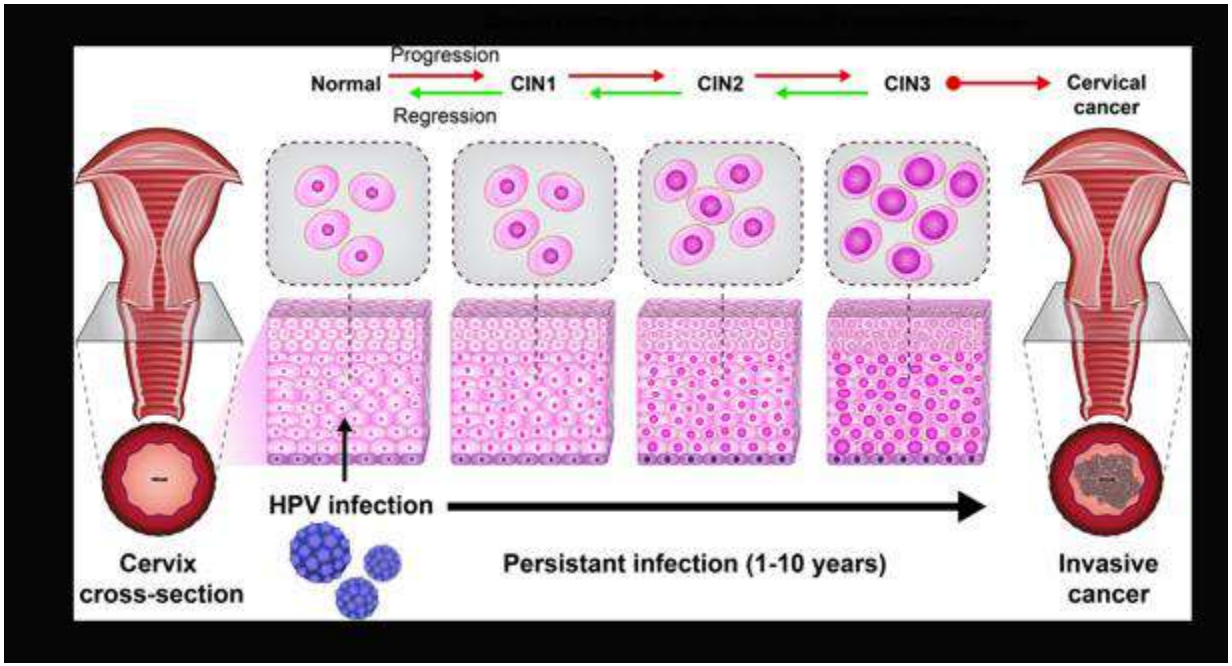
1. Cancer Cancer describes a group of diseases in which abnormal cells in the body begin to grow and multiply uncontrollably. These cells can form

HBV İnfeksiyonları- Durum Tespiti



Hepatit B virüsü (HBV), dünya çapında en yaygın görülen viral enfeksiyonlardan biridir.	Dünya Sağlık Örgütü (WHO) verilerine göre 2022 yılı itibarıyla:254 milyon kişi kronik HBV taşıyıcısı var.
Yıllık yaklaşık 1.2 milyon yeni HBV enfeksiyonu meydana gelir.	HBV enfeksiyonu her yıl en az 880.000 ölüme neden olur, bunların büyük kısmı siroz ve hepatoselüler karsinom (HCC) nedeniyle olur.
Kür yok!	Etkin antiviralleriniz var ama HCC antiviral tedavi altında dahi gelişebiliyor.

HPV.....



Dünyada en sık görülen kanserlerde 4. sırada, her yıl 600.000 yeni vaka, 300.000'i aşkın ölüm…

https://gco.iarc.fr/en

Table 1
Reported extrahepatic disease associations with HCV infection [2–69]

Antiphospholipid syndrome	MALToma
Aplastic anemia	Membranoproliferative glomerulonephritis
Autoimmune hemolytic anemia	Membranous glomerulonephritis
Autoimmune thyroiditis	Mixed cryoglobulinemia
Chronic fatigue syndrome	Mooren corneal ulcers
Behcet's syndrome	Multiple myeloma
Carotid atherosclerosis	Non-Hodgkin's lymphoma
CRST syndrome	Neurocognitive Impairment
Dermatomyositis	Pancreatitis
Diabetes mellitus	Polyarteritis nodosa
Fibromyalgia	Polymyositis
Guillain-Barré syndrome	Porphyria cutanea tarda
Hypertrophic cardiomyopathy	Rheumatoid arthritis
Hypocholesterolemia	Sialadenitis
Idiopathic pulmonary fibrosis	Sjögren's syndrome
Idiopathic thrombocytopenia purpura	Systemic lupus erythematosus
IgA deficiency	Uveitis
Lichen planus	Waldenstrom's macroglobulinemia

Helping bring phage medicines to UK patients – guidance for industry

Bacteriophages – viruses that selectively fight bacteria – may offer new hope in fighting infections and tackling antimicrobial resistance.

- https://theconversation.com/how-ancient-viruses-could-help-fight-antibiotic-resistance-261970

TABLE S-1 Possible Infectious Etiologies for Chronic Diseases Discussed at the Workshop

Infectious Agent	Chronic Disease/Condition	Chapter
Human papillomavirus	Cervical cancer	1
Hepatitis B virus	• Liver cancer	
	• Cirrhosis	1
Chlamydia pneumoniae	Atherosclerosis	1
Vaccinia virus	Postinfectious encephalomyelitis or acute disseminated encephalomyelitis (ADEM)	1
JC virus	Progressive multifocal leucoencephalopathy (PML)	1
Various viruses	Multiple sclerosis	1
Enteroviruses	Type I diabetes mellitus	1
Toxoplasma gondii	Schizophrenia	1
Herpes Simplex virus Type 2	Schizophrenia	1
Jaagsiekte sheep retrovirus (JSRV)	Ovine pulmonary adenocarcinoma	1
Propionibacterium acnes	• Chronic inflammatory acne	
	• Other chronic diseases	1
Cryptosporidiosis and intestinal helminthic infections	Disability consequences including growth shortfalls, fitness and cognitive impairment	2
Helminthic infections	Epilepsy	2
Plasmodium falciparum	Epilepsy	2
Treponema pallidum	Congenital syphilis	2
Toxoplasma gondii	Congenital toxoplasmosis	2
Maternal rubella virus	Congenital rubella	2
Perinatal HIV	Developmental disabilities	2
Perinatal herpes viruses	Neurodevelopmental disabilities	2
Plasmodium falciparum	• Cognitive development	
	• Childhood anemia	2
Haemophilus influenzae Type B meningitis	Nervous system impairment	2
Japanese encephalitis virus	Neuropsychiatric sequelae	2
Measles virus	Developmental disabilities	2
Poliovirus	Paralysis	
Chlamydia trachomatis	Trachoma	
Human T-cell lymphotropic virus Type 1	• Adult T-cell leukemia/lymphoma	2
	• Autoimmune disorders	
	• Infections associated with immunosuppression	
Human herpes virus Type 8	Kaposi's sarcoma	3
Borna disease virus	Neurodevelopmental disorders	3
Hepatitis C virus and *Schistosoma mansoni* interaction		2
HIV and *Plasmodium falciparum* interaction		2

Institute of Medicine. 2004. The Infectious Etiology of Chronic Diseases:Defining the Relationship, Enhancing the Research, and Mitigating the Effects:Workshop Summary. Washington, DC: The National Academies Press.
https://doi.org/10.17226/11026

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Epstein-Barr virus reprograms autoreactive B cells as antigen presenting cells in systemic lupus erythematosus

Shady Younis[1,2,*], **Salvinaz I. Moutusy**[1,2], **Sajede Rasouli**[1,2], **Shaghayegh Jahanbani**[1,2], **Mahesh Pandit**[1,2], **Xiaohao Wu**[1,2], **Suman Acharya**[1,2], **Orr Sharpe**[1,2], **Tilini U. Wijeratne**[1,2], **Marlayna L. Harris**[1], **Emily Y. Yang**[1], **Yashaar Chaichian**[1], **Shima Parsafar**[1], **Matthew C. Baker**[1], **John B. Harley**[3,4], **Eric Meffre**[1], **Lawrence Steinman**[5,6], **Ann Marshak-Rothstein**[7], **Judith A. James**[8,9], **Olivia M. Martinez**[10], **Paul J. Utz**[1,11], **Dana E. Orange**[12,13], **Tobias V. Lanz**[1,11], **William H. Robinson**[1,2,11,*]

Abstract

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease characterized by anti-nuclear antibodies (ANA). Epstein-Barr virus (EBV) infection has been epidemiologically associated with SLE, yet its role in pathogenesis remains incompletely defined. Here, we developed an EBV-specific single-cell RNA-sequencing platform and used it to demonstrate that EBV infection reprograms autoreactive anti-nuclear antigen B cells to drive autoimmunity in SLE. We demonstrated that, in SLE, EBV$^+$ B cells are predominantly CD27$^+$CD21^{low} memory B cells that are present at increased frequencies and express *ZEB2*, *TBX21*, and antigen presenting cell transcriptional pathways. Integrative analysis of chromatin immunoprecipitation sequencing (ChIP-seq), assay for transposase-accessible chromatin (ATAC-seq), and RNA Polymerase II occupancy data revealed EBNA2 binding at the transcriptional start sites and regulatory regions of *CD27*, *ZEB2*, and *TBX21* (Tbet), as well as the antigen presenting cell genes demonstrated to be up-regulated in SLE EBV$^+$ B cells. We expressed recombinant antibodies from SLE EBV$^+$ B cells and demonstrated that they bind prototypical SLE nuclear autoantigens, whereas those from healthy individuals do not. We further found that SLE EBV$^+$ B cells can serve as antigen presenting cells to activate T peripheral helper cells with concomitant activation of related EBV$^-$ anti-nuclear double negative 2 B cells and plasmablasts. Our results provide a potential mechanistic basis for EBV being a driver of SLE through infecting and reprograming nuclear antigen reactive B cells to become activated antigen presenting cells with the potential to promote systemic disease-driving autoimmune responses.

One sentence summary:

Epstein-Barr virus reprograms autoreactive B cells as antigen presenting cells to promote pathogenic anti-nuclear T and B cell responses in lupus.

Editor's Summary:

A Viral Driver of Lupus. Epstein-Barr virus (EBV) has been making waves as a candidate driver of diseases like multiple sclerosis and Long Covid. It has also long been linked to systemic lupus erythematosus (SLE), although the "why" behind this link has not been defined. Here, Younis *et al.* provide evidence for a link between EBV infection and disease development. The authors found, using a new strategy to identify EBV-infected cells by RNA-sequencing, that infected B cells were transcriptionally distinct from their uninfected counterparts. EBV-infected B cells exhibited features associated with antigen presentation, and this programming seemed likely to be directly driven by the EBV protein, EBNA2. These EBV-infected B cells with antigen presenting abilities had the capacity to activate autoreactive helper T cells, setting off a chain reaction where those T cells could activate other autoreactive B cells, including uninfected ones. In vitro studies in B cell lines provided functional support for this hypothesis. These data suggest that EBV infects and reprograms autoreactive B cells that in turn drive the systemic autoimmune response in SLE.

Esin Davutoğlu Şenol

Pandeminin izindeki çağ

Bu yüzyılın ilk çeyreği biterken olanca dehşetiyle yaşanmış bir salgının gölgesi bu sarılı yüzyılın gidişatını da belirliyor. Pandemi gelecek yıllara da ışık tutuyor.

Bilim

Chief Seattle

"...Whatever happens to the beasts also happens to man. All things are connected. *Whatever befalls the earth befalls the children of the earth."*

Prof. Dr. Esin Şenol

SİLAHLAR SAHİPSİZ ŞATOLAR
ŞÖVALYESİZ KALDI ŞİMDİ
MIGUEL DE CERVANTES

SAVAŞIYORUZ ÇÜNKÜ KENDİ
GERÇEKLİĞİMİZİ YİTİRDİĞİMÜZ BİR
GERÇEĞİN SAHNESİNDE ROLÜMÜZÜ
ANLAMAYA ÇALIŞIYORUZ
ESİN ŞENOL-ŞUBAT 2018