CHEMWATCH

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ASIA PACIFIC

Safe Work Australia has published Occupational lung diseases in Australia 2006-2019

2020-08-07

The report provides an overview of occupational lung diseases in Australia and identifies industries and occupations where workers may be at risk of developing an occupational lung disease, such as the construction, mining and quarrying industries, and those working with engineered stone.

The report highlights several significant trends, including:

- a substantial increase in:
- pneumoconiosis, especially coal workers pneumoconiosis, and
- silicosis from working with engineered stone
- a decline in workers' compensation claims for asbestos-related occupational lung diseases, such as asbestosis
- an increase in the understanding of the role of occupational exposure and the risk of developing coal workers pneumoconiosis, and
- an apparent decline in work-related asthma cases as evidenced by fewer compensation claims.

Overall, the report shows that occupational lung diseases continue to be a health concern in Australia and substantially contribute to the burden of lung disease. In particular, the report found:

- Further research is needed to identify incidence patterns of occupational lung diseases in high risk industries and occupations.
- Additional data collection and the thorough application of all data sources will assist in providing a comprehensive national perspective on occupational lung diseases.

Findings from the report will inform the implementation of Safe Work Australia's occupational lung diseases work plan.

Occupational lung diseases in Australia 2006–2019 was prepared by the Monash Centre for Occupational and Environmental Health and presents

The report provides an overview of occupational lung diseases in Australia and identifies industries and occupations where workers may be at risk of developing an occupational lung disease, such as the construction, mining and quarrying industries, and those working with engineered stone.

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changes in the extent and incidence of occupational lung diseases since the 2006 report Occupational respiratory disease in Australia.

Safe Work Australia, 7 August 2020

https://www.safeworkaustralia.gov.au/occupational-lung-diseases

22 October 2019

'Super-pollutant' emitted by 11 Chinese chemical plants could equal a climate catastrophe—Emission controls worked perfectly at Chinese plants, until a foreign subsidy dried up

2020-08-06

In December 2007, Charles Perilloux, an American chemical engineer, traveled to China to help install inexpensive and game-changing technology at a Chinese chemical plant that was spewing a climate "super-pollutant" into the atmosphere. The emissions quickly fell to near zero.

The state-owned Henan Shenma Nylon Chemical Company manufactures adipic acid, a key ingredient in nylon and polyurethane, which is used in everything from car parts to running shoes. While producing adipic acid, the factory emitted thousands of tons of nitrous oxide, a greenhouse gas nearly 300 times more potent than carbon dioxide in warming the planet.

Shenma's emission reductions had a greenhouse gas impact equivalent to taking one million cars off the road, records from the United Nations' Clean Development Mechanism (CDM) show. Through the program, Shenma reduced its emissions in exchange for lucrative carbon credits.

Full Article

Inside Climate News, 6 August 2020

https://insideclimatenews.org/news/04082020/china-n2o-super-pollutant-nylon-emissions-climate-change

The emissions quickly fell to near zero.

Australia want chemical plant moved after deadly Beirut blast

2020-08-06

SYDNEY (Reuters) - Some Australian residents of the city of Newcastle, 163 km (101 miles) north of Sydney, have called for a large ammonium nitrate

plant, stockpiling up to four times the amount reportedly detonated in Lebanon, to be relocated.

Lebanese authorities have blamed a huge stockpile of the highly explosive material stored for years in unsafe conditions at Beirut's port for Tuesday's explosion, which killed at least 145 people and injured more than 5,000.

It is estimated about 2,750 tonnes of ammonium nitrate were involved in the explosion.

By comparison, Australia's Orica stores between 6,000 to 12,000 tonnes of ammonium nitrate (AN), on average, at its Kooragang Island plant in the port of Newcastle, the company said in a statement.

The stockpile has led resident groups to campaign for the plant to be relocated, according to several local media reports.

Full Article

WTVB, 6 August 2020

https://wtvbam.com/news/articles/2020/aug/06/australians-want-chemical-plant-moved-after-deadly-beirut-blast/1046816/?refer-section=world

AMERICA

Proposed regulation would significantly change the warning requirements for Acrylamide and other chemicals formed during the cooking of foods under California's Proposition 65

2020-08-05

On August 4, 2020, the Office of Environmental Health Hazard Assessment (OEHHA), the lead agency that implements California's Proposition 65 ("Prop 65"), proposed to adopt a new regulation that would significantly change the warning requirements for listed chemicals formed by the cooking or heat processing of foods. 1/ The proposed regulation would provide that intake of such chemicals does not represent an exposure for the purposes of Prop 65 if the concentrations are reduced to the lowest level currently feasible using appropriate quality control measures. The proposal would establish maximum concentration levels for acrylamide in specific foods that are deemed by OEHHA to be the lowest levels currently

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feasible. Concentrations of the chemical at or below the level identified for the specified products would not require a warning.

It is noteworthy the proposed regulation is published at a time when a new wave of Prop 65 challenges are targeting acrylamide in food. In the past three months alone, private litigants have filed close to one hundred 60-day notices indicating their intent to sue food companies for acrylamide. This translates to about one Prop 65 notice filed every day in California for acrylamide in food. In its Initial Statement of Reasons, OEHHA stated the proposal is intended to (1) reduce exposures to listed chemicals present in food due to the human activities of cooking or heat processing, (2) provide warnings for avoidable exposures to acrylamide, and (3) safeguard the effectiveness of those warnings. 2/

The public can request a hearing on the proposed changes and the request must be received no later than September 21, 2020. Written comments to the proposed regulation are due on October 6, 2020.

Concentrations of the chemical at or below the level identified for the specified products would not require a warning.

Background on Acrylamide Prop 65 Litigations

For brief background, Prop 65 requires the Governor of California to publish, at least annually, a list of chemicals known to the state to cause cancer or reproductive toxicity. 3/ Businesses are required to provide a "clear and reasonable" warning before knowingly and intentionally exposing anyone in California to a listed chemical. Acrylamide has been a listed Prop 65 carcinogen since 1990. 4/ The California Attorney General and plaintiff's lawyers have filed numerous lawsuits in California seeking to impose Prop 65 warning requirements on food products for acrylamide.

Prop 65 permits private litigants or "bounty hunters" to bring private lawsuits to enforce the warning requirements. 5/ A "bounty hunter" seeking to sue for failure to warn as required by Prop 65 must notify the potential defendant and state prosecutors of the alleged violation and its intent to sue 60 days before a suit may be filed. 6/ These 60-day notices are publicly posted on the State's Office of the Attorney General's website. 7/ Over the past ten years, there have been multiple consent decrees for various foods that have set the level of acrylamide that does not require a Prop 65 warning. Food companies who are parties to these settlements and produce products within the negotiated levels do not have to provide the Prop 65 warning. The settlement also will identify the dollar damages the company must pay. Based on the most recent data available (i.e., 2018), the average settlement payment is around USD 42,424. 8/

Acrylamide in certain foods are already "exempt" from warning requirements. For example, on June 7, 2019, OEHHA adopted a regulation clarifying that exposures to Prop 65 substances in coffee do not pose a significant cancer risk. 9/ In essence, the regulation exempts coffee products from Prop 65 carcinogen warning requirements for chemicals such as acrylamide, to the extent that these carcinogens are created by and inherent in the process of roasting coffee beans or brewing coffee.

The Proposed Rulemaking

OEHHA is proposing to amend Title 27 of the California Code of Regulations, by adopting a new Section 25505, to address listed chemicals formed by cooking or heat processing foods. OEHHA noted that some degree of formation of listed chemicals in many foods (such as acrylamide) is unavoidable when the foods are cooked or otherwise processed with heat. As such, OEHHA reasoned, exposures to these chemicals in food are not necessarily the type of "knowing and intentional" exposures that require a warning under Prop 65. The proposed regulation provides that a person otherwise responsible for an exposure to a listed chemical in a food does not "expose" an individual to the extent the chemical was created by cooking or other heat processing, if the quality control measures that reduce the chemical to the lowest level currently feasible are utilized.

Full Article

Lexology, 5 August 2020

https://www.lexology.com/library/detail.aspx?g=6ee62f8b-abb8-4d69-a34e-4df0e69a37fa

Sweeping U.S. pesticide reform bill introduced, banning some chemical agents

2020-08-05

The Protect America's Children From Toxic Pesticides Act of 2020, introduced on Tuesday by Sen. Tom Udall, a New Mexico Democrat, and Rep. Joe Neguse, a Democrat of Colorado, would overhaul the nation's framework for regulating the sale and use of pesticides to safeguard public health and the environment, the legislators said in a press call with a panel of experts.

Current regulations are based on outdated science and contain loopholes that keep dangerous pesticides on the market despite clear evidence of harm to people and the environment, they said. "Our nation's system for regulating harmful pesticides is broken and badly outdated," said Sen. Udall. The system was meant to protect farmers and agricultural workers, consumers and the environment, he said. "But it's not. It's protecting the pesticide industry."

"Our nation's system for regulating harmful pesticides is broken and badly outdated," said Sen. Udall.

The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) governs how pesticides are sold and used through registration and labeling requirements. Pesticide manufacturers must show that their product, if used properly, will not cause "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide." The EPA is charged with ensuring that they do so.

Yet since pesticide regulations were last updated a quarter century ago, scientists have discovered that many pesticides on the market cause a range of neurodevelopmental effects and are likely contributing to the catastrophic decline of the bees that pollinate the nation's crops.

The EPA has wide latitude in deciding whether to ban a dangerous pesticide, Sen. Udall said. "And the EPA has been coming down on the side of industry profits and against the health of our children, farmworkers, families and the environment."

Rep. Neguse's district in Colorado includes top experts in organic and regenerative agriculture, he said. "And for years, these experts have been warning us of the dangers posed by the profit-driven pesticide regulation and oversight system as it exists today."

Once pesticides are approved in this system, they often remain on the market for decades, Rep. Neguse said, "even when scientific evidence overwhelmingly shows that a pesticide is causing harm to people or the environment."

Full Article

Successful Farming, 5 August 2020

https://www.agriculture.com/news/crops/sweeping-us-pesticide-reform-bill-introduced-banning-some-chemical-agents

Democratic bill banning toxic pesticides in US applaud as 'much-needed' step to protect kids and the planet

2020-08-04

"The Protect America's Children from Toxic Pesticides Act says what we all know to be true: that nerve agents have no place on our food, in our workplaces, and in our communities."

Democrats in the House and Senate on Tuesday introduced sweeping legislation that would ban some of the most toxic pesticides currently in use in the United States and institute stronger protections for farmworkers and communities that have been exposed to damaging chemicals by the agriculture industry.

Full Article

Common Dreams, 4 August 2020

https://www.commondreams.org/news/2020/08/04/democratic-bill-banning-toxic-pesticides-us-applauded-much-needed-step-protect-kids

EUROPE

Germany publishes overview of field of advanced materials

2020-08-06

In July 2020, the German Environment Agency (UBA) published a report entitled *Advanced materials: Overview of the field and screening criteria for relevance assessment*. The report describes activities within the project "Advanced materials — Thematic conferences: Assessment of needs to act on chemical safety" to provide discussion input on approaches to describe, cluster, and prioritize advanced materials. The aim of the report is to identify relevant advanced materials in regard to chemical safety. The report states that since the term advanced materials is not clearly defined, a major focus of the investigation is to characterize the use of the term to obtain a reasonable separation within the materials sector. According to the report, a set of criteria that could be applied to assess the relevance of advanced materials regarding chemical safety was developed

"The Protect America's Children from Toxic Pesticides Act says what we all know to be true: that nerve agents have no place on our food, in our workplaces, and in our communities."

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and is provided for further discussion and refinement. In addition, a first description of identified advanced material clusters was performed.

Bergeson & Campbell, P.C., 6 August 2020

https://nanotech.lawbc.com/2020/08/germany-publishes-overview-of-field-of-advanced-materials/?utm_source=Bergeson+%26+Campbell%2C+P.C.+-+Nano+and+Other+Emerging+Chemical+Technologies+Blog&utm_campaign=48029bc9d7-RSS_EMAIL_CAMPAIGN&utm_medium=email&utm_term=0_9a895e87b2-48029bc9d7-73807113

INTERNATIONAL

Chemical in Beirut explosion was also linked to 2 Texas disasters

2020-08-06

Chemical safety regulations were put in place after an explosion in the city of West, Texas, involving more than 80,000 pounds of ammonium nitrate. But those regulations were rolled back by the EPA in November.

For some, Tuesday's deadly explosion in Beirut, which officials say was caused by thousands of tons of ammonium nitrate, was a grim reminder of a 2013 disaster in the city of West, Texas.

In April of that year, a fertilizer plant exploded after more than 80,000 pounds of ammonium nitrate caught fire, killing 15 people, injuring more than 200 and causing significant damage to nearby buildings. The explosion was so powerful that it was recorded as a 2.1-magnitude earthquake on the Richter scale.

In response to that tragedy, the EPA under the Obama administration finalized a new Chemical Disaster Rule that was meant to prevent similar disasters from occurring by strengthening chemical safety and storage procedures. But that rule was largely rolled back in November 2019, amid outcry from environmentalists and some local governments.

Now, the explosion in Beirut is bringing renewed scrutiny to chemical safety regulations in the U.S.

"We've gone backwards instead of forward in terms of addressing some of the underlying risks that exist with the storage of this kind of material," said Elena Craft, senior director of climate and health at the Environmental

The explosion was so powerful that it was recorded as a 2.1-magnitude earthquake on the Richter scale.

Defense Fund. "I think the bottom line is these incidents are preventable and we're not doing enough to prevent them."

Full Article

Houston Public Media, 6 August 2020

https://www.houstonpublicmedia.org/articles/news/energy-environment/2020/08/06/379330/chemical-in-beirut-explosion-was-also-behind-two-texas-disasters/

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REACH current consultations

2020-08-07

Restrictions

Consultations on SEAC draft opinion: 2

- Start date: 24/06/2020
- Deadline: 24/08/2020

Consultations on SEAC draft opinion: 1

- Start date: 01/07/2020
- Deadline: 01/09/2020

Restriction proposals: 1

- Start date: 25/03/2020
- Deadline: 25/09/2020

Testing proposals

Testing proposals: 39

- Start date: 07/07/2020
- Deadline: 21/08/2020

ECHA, 7 August 2020

https://echa.europa.eu/consultations/current

Janet's Corner

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Ferrari

2020-08-14

https://www.reddit.com/r/memes/comments/i7abzo/big_brain_meme/

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Hazard Alert

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Chromium

2020-08-14

Chromium is a lustrous, hard steel-grey metal. It has the symbol of Cr and the atomic number 24. The pure metal is brittle and magnetic, but when alloyed, it can be malleable. Chromium is also found in chromium compounds, chromates and in chromic acid. Most chromium compounds have been classified as either Category 1A or Category 1B carcinogens: presumed or shown to cause cancer in humans. [1,2,3,4]

USES [5]

Chromium is used across various industries. It is primarily used as an alloy (ferrochrome) in the production of stainless steel and chrome plating. Chromium is used as it has a high corrosion resistance and hardness. The metal is also used across the tanning, glassmaking, wood preservation and reflective paint industries.

ROUTES OF EXPOSURE [2]

- Chromium III naturally occurs in rocks, soil and plants, and foods high in the metal include green beans and broccoli.
- Occupations, such as chrome plating, stainless steel welding and cutting, or leather tanning, are potential routes of exposure for the metal.

Chromium is a lustrous, hard steel-grey metal.

HEALTH EFFECTS

Chromium poisoning affects a range of systems, including the respiratory and integumentary systems.

Acute Effects [2]

Severity of symptoms depend on the level and type of exposure.

- Acute exposure to the metal can result in chrome ulcers and/or perforation of the nasal septum
- Chromium exposure can result in allergic dermatitis, which can be very persistent once developed.
- Short-term poisoning can result in respiratory effects, including airway irritation and obstruction.
- Exposure can lead to hepatic necrosis.

Chronic Effects [2]

Chromium is toxic to multiple body systems. Long-term exposure to the metal can cause chronic obstructive pulmonary diseases, including bronchopneumonia, chronic bronchitis, chronic inflammation of the lung, and emphysema. Chromium is also a known carcinogen; inhalation of the metal has been shown to result in lung cancer. Chronic exposure effects to the metal also include gastrointestinal effects, nephrotoxicity and hepatotoxicity.

SAFETY

First Aid Measures [6]

- Ingestion: DO NOT INDUCE VOMITING. Loosen any tight clothing, and contact a medical professional.
- Skin contact: Immediately rinse affected areas with plenty of water, and cover contaminated areas with an emollient. Remove all contaminated clothing, footwear and accessories. Do not re-wear clothing until it has been thoroughly decontaminated. Contact a doctor immediately.
- Eye contact: Check for and remove contact lenses if easy to do so. Flush eyes with water for at least 15 minutes, and get medical attention immediately.
- Inhalation: Take victim to the nearest fresh air source and monitor their breathing. Keep the victim warm. If the victim is not breathing, and you are qualified, you may perform CPR with a one-way valve or protective mask. Immediately contact a medical professional.
- General: Never administer anything by mouth to an unconscious, exposed person.

Exposure Controls/Personal Protection [6]

- Engineering controls: Emergency eyewash fountains and safety showers should be accessible in the immediate area of the potential exposure. Ensure there is adequate ventilation. Use a local exhaust ventilation or process enclosure to limit the amount of chromium in the air.
- Personal protection: Safety glasses, protective and dustproof clothing, gloves, an apron and an appropriate mask or dusk respirator. For specifications regarding other PPE, Follow the guidelines set in your jurisdiction.

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REGULATION [4]

United States:

The Occupational Safety and Health Administration (OSHA) has set an 8-hour time weighted average (TWA) concentration limit for chromium of 1mg/m^3.

Australia [7]

Safe Work Australia has set an 8-hour time TWA for chromium compounds as follows:

Chromium compound	Eight hour time weighted average (TWA; mg/m^3)
Chromium (II) compounds (as Cr)	0.5
Chromium (III) compounds (as Cr)	0.5
Chromium (metal)	0.5
Chromium (VI) compounds (as Cr) certain water insoluble	0.05
Chromium (VI) compounds (as Cr), water soluble	0.05

Source is Reference 2, p. 7.

REFERENCES

1. https://en.wikipedia.org/wiki/Chromium
2. https://www.safeworkaustralia.gov.au/system/files/documents/2002/health_monitoring_guidance_-_chromium.pdf
3. https://www.thoughtco.com/metal-profile-chromium-2340130
4. https://www.cdc.gov/niosh/topics/chromium/default.html
5. https://www.osha.gov/chromium
6. http://louisville.edu/micronano/files/documents/safety-data-sheets-sds/chromium

From 'brain fog' to heart damage, COVID-19's lingering problems alarm scientists

2020-07-31

Athena Akrami's neuroscience lab reopened last month without her. Life for the 38-year-old is a pale shadow of what it was before 17 March, the day she first experienced symptoms of the novel coronavirus. At University College London (UCL), Akrami's students probe how the brain organizes memories to support learning, but at home, she struggles to think clearly and battles joint and muscle pain. "I used to go to the gym three times a week," Akrami says. Now, "My physical activity is bed to couch, maybe couch to kitchen."

Her early symptoms were textbook for COVID-19: a fever and cough, followed by shortness of breath, chest pain, and extreme fatigue. For weeks, she struggled to heal at home. But rather than ebb with time, Akrami's symptoms waxed and waned without ever going away. She's had just 3 weeks since March when her body temperature was normal.

"Everybody talks about a binary situation, you either get it mild and recover quickly, or you get really sick and wind up in the ICU," says Akrami, who falls into neither category. Thousands echo her story in online COVID-19 support groups. Outpatient clinics for survivors are springing up, and some are already overburdened. Akrami has been waiting more than 4 weeks to be seen at one of them, despite a referral from her general practitioner.

The list of lingering maladies from COVID-19 is longer and more varied than most doctors could have imagined. Ongoing problems include fatigue, a racing heartbeat, shortness of breath, achy joints, foggy thinking, a persistent loss of sense of smell, and damage to the heart, lungs, kidneys, and brain.

The likelihood of a patient developing persistent symptoms is hard to pin down because different studies track different outcomes and follow survivors for different lengths of time. One group in Italy found that 87% of a patient **cohort hospitalized for acute COVID-19 was still struggling 2 months later**. Data from **the COVID Symptom Study**, which uses an app into which millions of people in the United States, United Kingdom, and Sweden have tapped their symptoms, suggest 10% to 15% of people—including some "mild" cases—don't quickly recover. But with the crisis just months old, no one knows how far into the future symptoms will endure, and whether COVID-19 will prompt the onset of chronic diseases.

"I used to go to the gym three times a week," Akrami says. Now, "My physical activity is bed to couch, maybe couch to kitchen."

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Researchers are now facing a familiar COVID-19 narrative: trying to make sense of a mystifying illness. Distinct features of the virus, including its propensity to cause widespread inflammation and blood clotting, could play a role in the assortment of concerns now surfacing. "We're seeing a really complex group of ongoing symptoms," says Rachael Evans, a pulmonologist at the University of Leicester.

Survivor studies are starting to probe them. This month, researchers across the United Kingdom including Evans launched a study that will follow 10,000 survivors for 1 year to start, and up to 25 years. Ultimately, researchers hope not just to understand the disease's long shadow, but also to predict who's at highest risk of lingering symptoms and learn whether treatments in the acute phase of illness can head them off.

For Götz Martin Richter, a radiologist at the Klinikum Stuttgart in Germany, what's especially striking is that just as the illness' acute symptoms vary unpredictably, so, too, do those that linger. Richter thinks of two patients he has treated: a middle-aged man who experienced mild pneumonia from COVID-19, and an elderly woman already suffering from chronic leukemia and arterial disease, who almost died from the virus and had to be resuscitated. Three months later, the man with the mild case "falls asleep all day long and cannot work," Richter says. The woman has minimal lung damage and feels fine.

EARLY IN the pandemic, doctors learned that SARS-CoV-2, the virus that causes COVID-19, can disrupt a **breathtaking array of tissues in the body**. Like a key fitting neatly into a lock, SARS-CoV-2 uses a spike protein on its surface to latch onto cells' ACE2 receptors. The lungs, heart, gut, kidneys, blood vessels, and nervous system, among other tissues, carry ACE2 on their cells' surfaces—and thus, are vulnerable to COVID-19. The virus can also induce a dramatic inflammatory reaction, including in the brain. Often, "The danger comes when the body responds out of proportion to the infection," says Adrija Hajra, a physician at Albert Einstein College of Medicine in New York City. She continues to care for those who were infected in the spring and are still recovering.

Despite the novelty of SARS-CoV-2, its long-term effects have precedents: Infections with other pathogens are associated with lasting impacts ranging from heart problems to chronic fatigue. "Medicine has been used to dealing with this problem" of acute viral illness followed by ongoing symptoms, says Michael Zandi, a neurologist at UCL. Even common illnesses such as pneumonia can mean a monthslong recovery. "I see a lot of people who had [the brain inflammation] encephalitis 3, 4 years ago,

and still can't think, or are tired," Zandi says. Infections with certain bacteria and Zika virus, among others, are linked to Guillain-Barre syndrome, in which the immune system attacks nerve tissue, causing tingling, weakness, and paralysis. (Some cases of Guillain-Barre after COVID-19 have been reported, but "it's not definite [there's] a spike," says Rachel Brown, a UCL neurologist who works with Zandi.)

Pain that lingersA subset of COVID-19 patients experiences ongoing symptoms and complications such as organ damage, and researchers are proposing reasons for some of them (bottom). Scientists are trying to identify suchsymptoms, how common they are, how long they last,who's at risk, and how to treat and prevent them.1Joint painChest painSkin rashCoughFatigueHeadacheInsomniaVertigo234Persistent fever1 Brain fogDifficulty thinking canoccur after acute COVID-19infection. The virus maydamage brain cells, andinflammation in the brainor body may also causeneurologic complications.Other viral infections canalso lead to brain fog.2 Shortness of breathDoctors are eyeing lungand heart complicationsincluding scarring.Patients who becomecritically ill with COVID-19seem more likely to havelingering shortness ofbreath, but those with mildcases are also at risk.3 Heart arrhythmiaThe virus can harmthe heart, and doctorsare concerned aboutlong-term damage. How the heart heals after COVID-19 could help determine whether a patient develops an irregular heartbeat.4 HypertensionSome patients havehigh blood pressureafter an acute infection,even when cases wererelatively mild andpeople were previouslyhealthy, possibly becausethe virus targets bloodvessels and heart cells.

Based on experience with other viruses, doctors can "extrapolate and anticipate" potential long-term effects of COVID-19, says Jeffrey Goldberger, chief of cardiology at the University of Miami. Like SARS-CoV-2, some other viruses, such as Epstein-Barr, can damage heart tissue, for example. In those infections, the organ sometimes heals completely. Sometimes, scarring is mild. "Or," Goldberger says, "it could be severe and lead to heart failure."

Michael Marks, an infectious disease specialist at the London School of Hygiene & Tropical Medicine who's helping lead the U.K. survivor study, says he's not too surprised at emerging aftereffects. "What we're experiencing is an epidemic of severe illness," he says. "So therefore, there is an epidemic" of chronic illness that follows it.

But outcomes following SARS-CoV-2 also appear distinct in ways both hopeful and dispiriting. Early this year, many doctors feared the virus

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would induce extensive, permanent lung damage in many survivors because two other coronaviruses, the viruses that cause the first severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome, can devastate the lungs. One study of health care workers with SARS in 2003 found that **those with lung lesions 1 year after infection** still had them after 15 years.

"We expected to see a lot of long-term damage from COVID-19: scarring, decreased lung function, decreased exercise capacity," says Ali Gholamrezanezhad, a radiologist at the Keck School of Medicine at the University of Southern California who in mid-January began to review lung scans from COVID-19 patients in Asia. Hundreds of scans later, he has concluded that COVID-19 ravages the lungs less consistently and aggressively than SARS did, when about 20% of patients sustained lasting lung damage. "COVID-19 is in general a milder disease," he says.

At the same time, the sheer breadth of complications linked to COVID-19 is mind-boggling. In late April, Akrami collaborated with Body Politic, a group of COVID-19 survivors, to survey more than 600 who still had symptoms after 2 weeks. She **logged 62 different symptoms** and is now readying the findings for publication and developing a second survey to capture longer term ailments. "Even though it's one virus, it can cause all different kinds of diseases in people," says Akiko Iwasaki, an immunologist at Yale University who is studying lingering effects on the immune system.

BY NOW IT'S CLEAR that many **people with COVID-19 severe enough to put them in a hospital** face a long recovery. The virus ravages the heart, for example, in multiple ways. Direct invasion of heart cells can damage or destroy them. Massive inflammation can affect cardiac function. The virus can blunt the function of ACE2 receptors, which normally help protect heart cells and degrade angiotensin II, a hormone that increases blood pressure. Stress on the body from fighting the virus can prompt release of adrenaline and epinephrine, which can also "have a deleterious effect on the heart," says Raul Mitrani, a cardiac electrophysiologist at the University of Miami who collaborates with Goldberger.

Mitrani and Goldberger, who co-authored a June paper in *Heart Rhythm* **urging follow-up of patients who might have heart damage**, worry in particular about the enzyme troponin, which is elevated in 20% to 30% of hospitalized COVID-19 patients and signifies cardiac damage. (Troponin is sky-high during a heart attack, for example.) How the heart heals following COVID-19 might determine whether an irregular heartbeat develops or persists, Goldberger believes. "We have one guy

in the hospital right now who had COVID 2 months ago and had all sorts of arrhythmia problems" then, Goldberger says. "He's recovered from his COVID, but still has the arrhythmia." For some patients with coronavirus-induced heart problems, treatments as simple as cholesterol-lowering drugs, aspirin, or beta blockers could help, Goldberger says.

Many people the pair has seen with heart complications post–COVID-19 had preexisting conditions, most commonly diabetes and hypertension. COVID-19, Goldberger suspects, tips them into more hazardous terrain or accelerates the onset of heart problems that, absent the coronavirus, might have developed later.

But other patients are affected without apparent risk factors: A paper this week in *JAMA Cardiology* found that 78 of 100 people diagnosed with COVID-19 had cardiac abnormalities when their heart was imaged on average 10 weeks later, most often inflammation in heart muscle. Many of the participants in that study were previously healthy, and some even caught the virus while on ski trips, according to the authors.

Severe lung scarring appears less common than feared—Gholamrezanezhad knows of only one recovered patient who still needs oxygen at rest. Scarring seems most likely to accompany underlying lung disease, hypertension, obesity, and other conditions. Lung damage is also seen in people who spend weeks on a ventilator. Gholamrezanezhad suspects that, as with harm to the heart, previously healthy people are not exempt from the virus' long-term effects on the lungs, though their risk is likely lower.

Then there's the nervous system, a worrying target. Severe complications seem relatively rare but aren't limited to those whom the virus renders critically ill. Brown, Zandi, and colleagues described 43 people with neurologic complications **this month in *Brain***; many had been hospitalized during their acute infection, but not always for long—and for some, neurologic problems were their most debilitating symptom and the reason for hospital admission. Several were struggling to recover from encephalitis. Others had inflammation in their brain's white matter, which helps transmit electrical signals.

Separately, doctors are starting to see a class of patients who, like Akrami, struggle to think clearly—another outcome physicians have come upon in the past. After some severe viral infections, there are "those people who still don't feel quite right afterward, but have normal brain scans," Brown says. Some neurologists and patients describe the phenomenon as "brain

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fog." It's largely a mystery, though one theory suggests it's similar to a "postviral fatigue related to inflammation in the body," Brown says.

Could that be happening here? "Who knows, really?" Brown asks. "These patients need to be followed."

PEOPLE LIKE THESE pose a growing concern (though they are also often dismissed by physicians). Collectively, these "long-haulers" describe dozens of symptoms, including many that could have multiple causes, such as fatigue, joint pain, and fever. "It's time to give some voice to this huge population of patients," Akrami says.

The most bedeviling and common lingering symptom seems to be fatigue, but researchers caution against calling it chronic fatigue syndrome. That's "a specific diagnosis," Marks says. "You might have fibrosis in the lungs, and that will make you feel fatigued; you might have impaired heart function, and that will make you feel fatigued." Trying to trace symptoms to their source is critical to understanding and ultimately managing them, he says.

Iwasaki agrees. Doctors would treat symptoms differently depending on whether they result from a lingering infection or are rooted in autoimmune abnormalities. She has begun to recruit people who weren't hospitalized when they had COVID-19 and will sift through her volunteers' immune cells, examine whether they're primed to attack, and measure whether the balance among different cell types is as it should be. She'll also hunt for virus in saliva. "We're pretty much searching for anything," she says.

Iwasaki is especially struck by the number of young, healthy, active people—people like Akrami—who fall into the long-hauler category. As she and others struggle to find ways to help them, she wonders what might head off their symptoms. One possibility, she says, is monoclonal antibodies, which are now being **tested as a treatment for acute infection** and might also forestall lasting immune problems.

Hers is one of several survivor studies now underway. While Goldberger's hometown of Miami faces a surge of acutely ill patients, he is looking ahead, applying for funding to image the heart and map its electrical activity in COVID-19 patients after they leave the hospital. Gholamrezanezhad is recruiting 100 patients after hospital discharge to follow for up to 2 years for lung assessments. Like many physicians, he fears the societal impact of even uncommon complications, including in the millions of people never hospitalized. "When you consider how many people are getting the disease, it's a big problem," he says.

Across the Atlantic Ocean, Richter has recruited 300 volunteers in Germany for long-term follow-up, including lung scans. In the United Kingdom, patients will soon be able to sign up for that country's survivor study, with many giving blood samples and being examined by specialists. The researchers will probe patients' DNA and examine other characteristics such as age and health history to learn what might protect them from, or make them susceptible to, a range of COVID-19 induced health problems. Knowing who's at risk of, say, kidney failure or cardiac arrhythmia could mean more targeted follow-up. The U.K. researchers are also keen to see whether patients who received certain treatments in the acute phase of illness, such as steroids or blood thinners, are less prone to later complications.

For her part, Akrami is one of 2 million people infected weeks or months ago participating in the COVID Symptom Study. The study welcomes anyone infected, and with 10% to 15% of people who use the app reporting ongoing symptoms, it has already yielded a welter of data, says Andrew Chan, an epidemiologist and physician at Harvard Medical School.

As he and his colleagues parse the data, they are identifying **distinct "types" of acute illness**, based on clusters of symptoms. Chan wonders whether certain early symptoms correlate with specific ones that linger. He acknowledges the risk that the app's data could be skewed, because people who aren't feeling well may be more likely to participate than those who have smooth recoveries. "We're trying to develop data analysis tools" to account for that tilt, he says, "similar to methods used in polling. You have to weigh the biases."

One of the few systematic, long-term studies of COVID-19 patients with mild acute symptoms is underway in San Francisco, where researchers are recruiting 300 adults from local doctors and hospitals, for 2 years of follow-up. "We don't have a broad idea of what's happening" after the initial illness, says Steven Deeks, an HIV researcher at the University of California, San Francisco, who is leading the study, modeled on HIV cohorts he has followed for decades. What does "ongoing symptoms" even mean, Deeks asks. "Is that weeks, months? We don't know that it's years."

More than 100 people ranging in age from 18 to 80 have signed up so far. Cardiologists, neurologists, pulmonologists, and others are assessing the volunteers, and blood, saliva, and other biological specimens are being banked and analyzed.

Although scientists hope they'll learn how to avert chronic symptoms and help patients currently suffering, this latest chapter in the COVID-19

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chronicle has been sobering. The message many researchers want to impart: Don't underestimate the force of this virus. "Even if the story comes out a little scary, we need a bit of that right now," Iwasaki says, because the world needs to know how high the stakes are. "Once the disease is established, it's really hard to go backward."

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How the pandemic defeated America

2020-08-04

How did it come to this? A virus a thousand times smaller than a dust mote has humbled and humiliated the planet's most powerful nation. America has failed to protect its people, leaving them with illness and financial ruin. It has lost its status as a global leader. It has careened between inaction and ineptitude. The breadth and magnitude of its errors are difficult, in the moment, to truly fathom.

America has failed to protect its people, leaving them with illness and financial ruin.

In the first half of 2020, SARSCoV2—the new coronavirus behind the disease COVID19—infected 10 million people around the world and killed about half a million. But few countries have been as severely hit as the United States, which has just 4 percent of the world's population but a quarter of its confirmed COVID19 cases and deaths. These numbers are estimates. The actual toll, though undoubtedly higher, is unknown, because the richest country in the world still lacks sufficient testing to accurately count its sick citizens.

Despite ample warning, the U.S. squandered every possible opportunity to control the coronavirus. And despite its considerable advantages—immense resources, biomedical might, scientific expertise—it floundered. While countries as different as South Korea, Thailand, Iceland, Slovakia, and Australia acted decisively to bend the curve of infections downward, the U.S. achieved merely a plateau in the spring, which changed to an appalling upward slope in the summer. "The U.S. fundamentally failed in ways that were worse than I ever could have imagined," Julia Marcus, an infectious-disease epidemiologist at Harvard Medical School, told me.

Since the pandemic began, I have spoken with more than 100 experts in a variety of fields. I've learned that almost everything that went wrong with America's response to the pandemic was predictable and preventable. A sluggish response by a government denuded of expertise allowed the coronavirus to gain a foothold. Chronic underfunding of public

health neutered the nation's ability to prevent the pathogen's spread. A bloated, inefficient health-care system left hospitals ill-prepared for the ensuing wave of sickness. Racist policies that have endured since the days of colonization and slavery left Indigenous and Black Americans especially vulnerable to COVID19. The decades-long process of shredding the nation's social safety net forced millions of essential workers in low-paying jobs to risk their life for their livelihood. The same social-media platforms that sowed partisanship and misinformation during the 2014 Ebola outbreak in Africa and the 2016 U.S. election became vectors for conspiracy theories during the 2020 pandemic.

The U.S. has little excuse for its inattention. In recent decades, epidemics of SARS, MERS, Ebola, H1N1 flu, Zika, and monkeypox showed the havoc that new and reemergent pathogens could wreak. Health experts, business leaders, and even middle schoolers ran simulated exercises to game out the spread of new diseases. In 2018, I wrote an article for The Atlantic arguing that the U.S. was not ready for a pandemic, and sounded warnings about the fragility of the nation's health-care system and the slow process of creating a vaccine. But the COVID19 debacle has also touched—and implicated—nearly every other facet of American society: its shortsighted leadership, its disregard for expertise, its racial inequities, its social-media culture, and its fealty to a dangerous strain of individualism.

SARSCoV2 is something of an anti-Goldilocks virus: just bad enough in every way. Its symptoms can be severe enough to kill millions but are often mild enough to allow infections to move undetected through a population. It spreads quickly enough to overload hospitals, but slowly enough that statistics don't spike until too late. These traits made the virus harder to control, but they also softened the pandemic's punch. SARSCoV2 is neither as lethal as some other coronaviruses, such as SARS and MERS, nor as contagious as measles. Deadlier pathogens almost certainly exist. Wild animals harbor an estimated 40,000 unknown viruses, a quarter of which could potentially jump into humans. How will the U.S. fare when "we can't even deal with a starter pandemic?," Zeynep Tufekci, a sociologist at the University of North Carolina and an *Atlantic* contributing writer, asked me.

Despite its epochal effects, COVID19 is merely a harbinger of worse plagues to come. The U.S. cannot prepare for these inevitable crises if it returns to normal, as many of its people ache to do. Normal led to this. Normal was a world ever more prone to a pandemic but ever less ready for one. To avert another catastrophe, the U.S. needs to grapple with all the

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ways normal failed us. It needs a full accounting of every recent misstep and foundational sin, every unattended weakness and unheeded warning, every festering wound and reopened scar.

A pandemic can be prevented in two ways: Stop an infection from ever arising, or stop an infection from becoming thousands more. The first way is likely impossible. There are simply too many viruses and too many animals that harbor them. Bats alone could host thousands of unknown coronaviruses; in some Chinese caves, one out of every 20 bats is infected. Many people live near these caves, shelter in them, or collect guano from them for fertilizer. Thousands of bats also fly over these people's villages and roost in their homes, creating opportunities for the bats' viral stowaways to spill over into human hosts. Based on antibody testing in rural parts of China, Peter Daszak of EcoHealth Alliance, a nonprofit that studies emerging diseases, estimates that such viruses infect a substantial number of people every year. "Most infected people don't know about it, and most of the viruses aren't transmissible," Daszak says. But it takes just one transmissible virus to start a pandemic.

Sometime in late 2019, the wrong virus left a bat and ended up, perhaps via an intermediate host, in a human—and another, and another. Eventually it found its way to the Huanan seafood market, and jumped into dozens of new hosts in an explosive super-spreading event. The COVID19 pandemic had begun.

"There is no way to get spillover of everything to zero," Colin Carlson, an ecologist at Georgetown University, told me. Many conservationists jump on epidemics as opportunities to ban the wildlife trade or the eating of "bush meat," an exoticized term for "game," but few diseases have emerged through either route. Carlson said the biggest factors behind spillovers are land-use change and climate change, both of which are hard to control. Our species has relentlessly expanded into previously wild spaces. Through intensive agriculture, habitat destruction, and rising temperatures, we have uprooted the planet's animals, forcing them into new and narrower ranges that are on our own doorsteps. Humanity has squeezed the world's wildlife in a crushing grip—and viruses have come bursting out.

Curtailing those viruses after they spill over is more feasible, but requires knowledge, transparency, and decisiveness that were lacking in 2020. Much about coronaviruses is still unknown. There are no surveillance networks for detecting them as there are for influenza. There are no approved treatments or vaccines. Coronaviruses were formerly a niche

family, of mainly veterinary importance. Four decades ago, just 60 or so scientists attended the first international meeting on coronaviruses. Their ranks swelled after SARS swept the world in 2003, but quickly dwindled as a spike in funding vanished. The same thing happened after MERS emerged in 2012. This year, the world's coronavirus experts—and there still aren't many—had to postpone their triennial conference in the Netherlands because SARSCoV2 made flying too risky.

In the age of cheap air travel, an outbreak that begins on one continent can easily reach the others. SARS already demonstrated that in 2003, and more than twice as many people now travel by plane every year. To avert a pandemic, affected nations must alert their neighbors quickly. In 2003, China covered up the early spread of SARS, allowing the new disease to gain a foothold, and in 2020, history repeated itself. The Chinese government downplayed the possibility that SARSCoV2 was spreading among humans, and only confirmed as much on January 20, after millions had traveled around the country for the lunar new year. Doctors who tried to raise the alarm were censured and threatened. One, Li Wenliang, later died of COVID19. The World Health Organization initially parroted China's line and did not declare a public-health emergency of international concern until January 30. By then, an estimated 10,000 people in 20 countries had been infected, and the virus was spreading fast.

The United States has correctly castigated China for its duplicity and the WHO for its laxity—but the U.S. has also failed the international community. Under President Donald Trump, the U.S. has withdrawn from several international partnerships and antagonized its allies. It has a seat on the WHO's executive board, but left that position empty for more than two years, only filling it this May, when the pandemic was in full swing. Since 2017, Trump has pulled more than 30 staffers out of the Centers for Disease Control and Prevention's office in China, who could have warned about the spreading coronavirus. Last July, he defunded an American epidemiologist embedded within China's CDC. America First was America oblivious.

Even after warnings reached the U.S., they fell on the wrong ears. Since before his election, Trump has cavalierly dismissed expertise and evidence. He filled his administration with inexperienced newcomers, while depicting career civil servants as part of a "deep state." In 2018, he dismantled an office that had been assembled specifically to prepare for nascent pandemics. American intelligence agencies warned about the coronavirus threat in January, but Trump habitually disregards intelligence

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briefings. The secretary of health and human services, Alex Azar, offered similar counsel, and was twice ignored.

Being prepared means being ready to spring into action, "so that when something like this happens, you're moving quickly," Ronald Klain, who coordinated the U.S. response to the West African Ebola outbreak in 2014, told me. "By early February, we should have triggered a series of actions, precisely zero of which were taken." Trump could have spent those crucial early weeks mass-producing tests to detect the virus, asking companies to manufacture protective equipment and ventilators, and otherwise steeling the nation for the worst. Instead, he focused on the border. On January 31, Trump announced that the U.S. would bar entry to foreigners who had recently been in China, and urged Americans to avoid going there.

Travel bans make intuitive sense, because travel obviously enables the spread of a virus. But in practice, travel bans are woefully inefficient at restricting either travel or viruses. They prompt people to seek indirect routes via third-party countries, or to deliberately hide their symptoms. They are often porous: Trump's included numerous exceptions, and allowed tens of thousands of people to enter from China. Ironically, they *create* travel: When Trump later announced a ban on flights from continental Europe, a surge of travelers packed America's airports in a rush to beat the incoming restrictions. Travel bans may sometimes work for remote island nations, but in general they can only delay the spread of an epidemic—not stop it. And they can create a harmful false confidence, so countries "rely on bans to the exclusion of the things they actually need to do—testing, tracing, building up the health system," says Thomas Bollyky, a global-health expert at the Council on Foreign Relations. "That sounds an awful lot like what happened in the U.S."

This was predictable. A president who is fixated on an ineffectual border wall, and has portrayed asylum seekers as vectors of disease, was always going to reach for travel bans as a first resort. And Americans who bought into his rhetoric of xenophobia and isolationism were going to be especially susceptible to thinking that simple entry controls were a panacea.

And so the U.S. wasted its best chance of restraining COVID19. Although the disease first arrived in the U.S. in mid-January, genetic evidence shows that the specific viruses that triggered the first big outbreaks, in Washington State, didn't land until mid-February. The country could have used that time to prepare. Instead, Trump, who had spent his entire presidency learning that he could say whatever he wanted without

consequence, assured Americans that "the coronavirus is very much under control," and "like a miracle, it will disappear." With impunity, Trump lied. With impunity, the virus spread.

On February 26, Trump asserted that cases were "going to be down to close to zero." Over the next two months, at least 1 million Americans were infected.

As the coronavirus established itself in the U.S., it found a nation through which it could spread easily, without being detected. For years, Pardis Sabeti, a virologist at the Broad Institute of Harvard and MIT, has been trying to create a surveillance network that would allow hospitals in every major U.S. city to quickly track new viruses through genetic sequencing. Had that network existed, once Chinese scientists published SARSCoV2's genome on January 11, every American hospital would have been able to develop its own diagnostic test in preparation for the virus's arrival. "I spent a lot of time trying to convince many funders to fund it," Sabeti told me. "I never got anywhere."

The CDC developed and distributed its own diagnostic tests in late January. These proved useless because of a faulty chemical component. Tests were in such short supply, and the criteria for getting them were so laughably stringent, that by the end of February, tens of thousands of Americans had likely been infected but only hundreds had been tested. The official data were so clearly wrong that *The Atlantic* developed its own volunteer-led initiative—the COVID Tracking Project—to count cases.

Diagnostic tests are easy to make, so the U.S. failing to create one seemed inconceivable. Worse, it had no Plan B. Private labs were strangled by FDA bureaucracy. Meanwhile, Sabeti's lab developed a diagnostic test in mid-January and sent it to colleagues in Nigeria, Sierra Leone, and Senegal. "We had working diagnostics in those countries well before we did in any U.S. states," she told me.

It's hard to overstate how thoroughly the testing debacle incapacitated the U.S. People with debilitating symptoms couldn't find out what was wrong with them. Health officials couldn't cut off chains of transmission by identifying people who were sick and asking them to isolate themselves.

Water running along a pavement will readily seep into every crack; so, too, did the unchecked coronavirus seep into every fault line in the modern world. Consider our buildings. In response to the global energy crisis of the 1970s, architects made structures more energy-efficient by sealing them off from outdoor air, reducing ventilation rates. Pollutants and pathogens

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built up indoors, "ushering in the era of 'sick buildings,'" says Joseph Allen, who studies environmental health at Harvard's T. H. Chan School of Public Health. Energy efficiency is a pillar of modern climate policy, but there are ways to achieve it without sacrificing well-being. "We lost our way over the years and stopped designing buildings for people," Allen says.

The indoor spaces in which Americans spend 87 percent of their time became staging grounds for super-spreading events. One study showed that the odds of catching the virus from an infected person are roughly 19 times higher indoors than in open air. Shielded from the elements and among crowds clustered in prolonged proximity, the coronavirus ran rampant in the conference rooms of a Boston hotel, the cabins of the Diamond Princess cruise ship, and a church hall in Washington State where a choir practiced for just a few hours.

The hardest-hit buildings were those that had been jammed with people for decades: prisons. Between harsher punishments doled out in the War on Drugs and a tough-on-crime mindset that prizes retribution over rehabilitation, America's incarcerated population has swelled sevenfold since the 1970s, to about 2.3 million. The U.S. imprisons five to 18 times more people per capita than other Western democracies. Many American prisons are packed beyond capacity, making social distancing impossible. Soap is often scarce. Inevitably, the coronavirus ran amok. By June, two American prisons each accounted for more cases than all of New Zealand. One, Marion Correctional Institution, in Ohio, had more than 2,000 cases among inmates despite having a capacity of 1,500. Other densely packed facilities were also besieged. America's nursing homes and long-term-care facilities house less than 1 percent of its people, but as of mid-June, they accounted for 40 percent of its coronavirus deaths. More than 50,000 residents and staff have died. At least 250,000 more have been infected. These grim figures are a reflection not just of the greater harms that COVID19 inflicts upon elderly physiology, but also of the care the elderly receive. Before the pandemic, three in four nursing homes were understaffed, and four in five had recently been cited for failures in infection control. The Trump administration's policies have exacerbated the problem by reducing the influx of immigrants, who make up a quarter of long-term caregivers.

Even though a Seattle nursing home was one of the first COVID19 hot spots in the U.S., similar facilities weren't provided with tests and protective equipment. Rather than girding these facilities against the pandemic, the Department of Health and Human Services paused nursing-home inspections in March, passing the buck to the states. Some nursing

homes avoided the virus because their owners immediately stopped visitations, or paid caregivers to live on-site. But in others, staff stopped working, scared about infecting their charges or becoming infected themselves. In some cases, residents had to be evacuated because no one showed up to care for them.

America's neglect of nursing homes and prisons, its sick buildings, and its botched deployment of tests are all indicative of its problematic attitude toward health: "Get hospitals ready and wait for sick people to show," as Sheila Davis, the CEO of the nonprofit Partners in Health, puts it. "Especially in the beginning, we catered our entire [COVID19] response to the 20 percent of people who required hospitalization, rather than preventing transmission in the community." The latter is the job of the public-health system, which prevents sickness in populations instead of merely treating it in individuals. That system pairs uneasily with a national temperament that views health as a matter of personal responsibility rather than a collective good.

At the end of the 20th century, public-health improvements meant that Americans were living an average of 30 years longer than they were at the start of it. Maternal mortality had fallen by 99 percent; infant mortality by 90 percent. Fortified foods all but eliminated rickets and goiters. Vaccines eradicated smallpox and polio, and brought measles, diphtheria, and rubella to heel. These measures, coupled with antibiotics and better sanitation, curbed infectious diseases to such a degree that some scientists predicted they would soon pass into history. But instead, these achievements brought complacency. "As public health did its job, it became a target" of budget cuts, says Lori Freeman, the CEO of the National Association of County and City Health Officials.

Today, the U.S. spends just 2.5 percent of its gigantic health-care budget on public health. Underfunded health departments were already struggling to deal with opioid addiction, climbing obesity rates, contaminated water, and easily preventable diseases. Last year saw the most measles cases since 1992. In 2018, the U.S. had 115,000 cases of syphilis and 580,000 cases of gonorrhea—numbers not seen in almost three decades. It has 1.7 million cases of chlamydia, the highest number ever recorded.

Since the last recession, in 2009, chronically strapped local health departments have lost 55,000 jobs—a quarter of their workforce. When COVID19 arrived, the economic downturn forced overstretched departments to furlough more employees. When states needed

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battalions of public-health workers to find infected people and trace their contacts, they had to hire and train people from scratch. In May, Maryland Governor Larry Hogan asserted that his state would soon have enough people to trace 10,000 contacts every day. Last year, as Ebola tore through the Democratic Republic of Congo—a country with a quarter of Maryland's wealth and an active war zone—local health workers and the WHO traced twice as many people.

Ripping unimpeded through American communities, the coronavirus created thousands of sickly hosts that it then rode into America's hospitals. It should have found facilities armed with state-of-the-art medical technologies, detailed pandemic plans, and ample supplies of protective equipment and life-saving medicines. Instead, it found a brittle system in danger of collapse.

Compared with the average wealthy nation, America spends nearly twice as much of its national wealth on health care, about a quarter of which is wasted on inefficient care, unnecessary treatments, and administrative chicanery. The U.S. gets little bang for its exorbitant buck. It has the lowest life-expectancy rate of comparable countries, the highest rates of chronic disease, and the fewest doctors per person. This profit-driven system has scant incentive to invest in spare beds, stockpiled supplies, peacetime drills, and layered contingency plans—the essence of pandemic preparedness. America's hospitals have been pruned and stretched by market forces to run close to full capacity, with little ability to adapt in a crisis.

When hospitals do create pandemic plans, they tend to fight the last war. After 2014, several centers created specialized treatment units designed for Ebola—a highly lethal but not very contagious disease. These units were all but useless against a highly transmissible airborne virus like SARSCoV2. Nor were hospitals ready for an outbreak to drag on for months. Emergency plans assumed that staff could endure a few days of exhausting conditions, that supplies would hold, and that hard-hit centers could be supported by unaffected neighbors. "We're designed for discrete disasters" like mass shootings, traffic pileups, and hurricanes, says Esther Choo, an emergency physician at Oregon Health and Science University. The COVID19 pandemic is not a discrete disaster. It is a 50-state catastrophe that will likely continue at least until a vaccine is ready.

Wherever the coronavirus arrived, hospitals reeled. Several states asked medical students to graduate early, reenlisted retired doctors, and deployed dermatologists to emergency departments. Doctors and nurses

endured grueling shifts, their faces chapped and bloody when they finally doffed their protective equipment. Soon, that equipment—masks, respirators, gowns, gloves—started running out.

In the middle of the greatest health and economic crises in generations, millions of Americans have found themselves impoverished and disconnected from medical care.

American hospitals operate on a just-in-time economy. They acquire the goods they need in the moment through labyrinthine supply chains that wrap around the world in tangled lines, from countries with cheap labor to richer nations like the U.S. The lines are invisible until they snap. About half of the world's face masks, for example, are made in China, some of them in Hubei province. When that region became the pandemic epicenter, the mask supply shriveled just as global demand spiked. The Trump administration turned to a larder of medical supplies called the Strategic National Stockpile, only to find that the 100 million respirators and masks that had been dispersed during the 2009 flu pandemic were never replaced. Just 13 million respirators were left.

In April, four in five frontline nurses said they didn't have enough protective equipment. Some solicited donations from the public, or navigated a morass of back-alley deals and internet scams. Others fashioned their own surgical masks from bandannas and gowns from garbage bags. The supply of nasopharyngeal swabs that are used in every diagnostic test also ran low, because one of the largest manufacturers is based in Lombardy, Italy—initially the COVID19 capital of Europe. About 40 percent of critical-care drugs, including antibiotics and painkillers, became scarce because they depend on manufacturing lines that begin in China and India. Once a vaccine is ready, there might not be enough vials to put it in, because of the long-running global shortage of medical-grade glass—literally, a bottle-neck bottleneck.

The federal government could have mitigated those problems by buying supplies at economies of scale and distributing them according to need. Instead, in March, Trump told America's governors to "try getting it yourselves." As usual, health care was a matter of capitalism and connections. In New York, rich hospitals bought their way out of their protective-equipment shortfall, while neighbors in poorer, more diverse parts of the city rationed their supplies.

While the president prevaricated, Americans acted. Businesses sent their employees home. People practiced social distancing, even before Trump finally declared a national emergency on March 13, and before governors

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and mayors subsequently issued formal stay-at-home orders, or closed schools, shops, and restaurants. A study showed that the U.S. could have averted 36,000 COVID19 deaths if leaders had enacted social-distancing measures just a week earlier. But better late than never: By collectively reducing the spread of the virus, America flattened the curve. Ventilators didn't run out, as they had in parts of Italy. Hospitals had time to add extra beds.

Social distancing worked. But the indiscriminate lockdown was necessary only because America's leaders wasted months of prep time. Deploying this blunt policy instrument came at enormous cost. Unemployment rose to 14.7 percent, the highest level since record-keeping began, in 1948. More than 26 million people lost their jobs, a catastrophe in a country that—uniquely and absurdly—ties health care to employment. Some COVID19 survivors have been hit with seven-figure medical bills. In the middle of the greatest health and economic crises in generations, millions of Americans have found themselves disconnected from medical care and impoverished. They join the millions who have always lived that way.

The coronavirus found, exploited, and widened every inequity that the U.S. had to offer. Elderly people, already pushed to the fringes of society, were treated as acceptable losses. Women were more likely to lose jobs than men, and also shouldered extra burdens of child care and domestic work, while facing rising rates of domestic violence. In half of the states, people with dementia and intellectual disabilities faced policies that threatened to deny them access to lifesaving ventilators. Thousands of people endured months of COVID19 symptoms that resembled those of chronic postviral illnesses, only to be told that their devastating symptoms were in their head. Latinos were three times as likely to be infected as white people. Asian Americans faced racist abuse. Far from being a "great equalizer," the pandemic fell unevenly upon the U.S., taking advantage of injustices that had been brewing throughout the nation's history.

Of the 3.1 million Americans who still cannot afford health insurance in states where Medicaid has not been expanded, more than half are people of color, and 30 percent are Black.* This is no accident. In the decades after the Civil War, the white leaders of former slave states deliberately withheld health care from Black Americans, apportioning medicine more according to the logic of Jim Crow than Hippocrates. They built hospitals away from Black communities, segregated Black patients into separate wings, and blocked Black students from medical school. In the 20th century, they helped construct America's system of private, employer-based insurance, which has kept many Black people from receiving adequate medical

treatment. They fought every attempt to improve Black people's access to health care, from the creation of Medicare and Medicaid in the '60s to the passage of the Affordable Care Act in 2010.

A number of former slave states also have among the lowest investments in public health, the lowest quality of medical care, the highest proportions of Black citizens, and the greatest racial divides in health outcomes. As the COVID19 pandemic wore on, they were among the quickest to lift social-distancing restrictions and reexpose their citizens to the coronavirus. The harms of these moves were unduly foisted upon the poor and the Black.

As of early July, one in every 1,450 Black Americans had died from COVID19—a rate more than twice that of white Americans. That figure is both tragic and wholly expected given the mountain of medical disadvantages that Black people face. Compared with white people, they die three years younger. Three times as many Black mothers die during pregnancy. Black people have higher rates of chronic illnesses that predispose them to fatal cases of COVID19. When they go to hospitals, they're less likely to be treated. The care they do receive tends to be poorer. Aware of these biases, Black people are hesitant to seek aid for COVID19 symptoms and then show up at hospitals in sicker states. "One of my patients said, 'I don't want to go to the hospital, because they're not going to treat me well,'" says Uché Blackstock, an emergency physician and the founder of Advancing Health Equity, a nonprofit that fights bias and racism in health care. "Another whispered to me, 'I'm so relieved you're Black. I just want to make sure I'm listened to.'"

Rather than countering misinformation during the pandemic, trusted sources often made things worse.

Black people were both more worried about the pandemic and more likely to be infected by it. The dismantling of America's social safety net left Black people with less income and higher unemployment. They make up a disproportionate share of the low-paid "essential workers" who were expected to staff grocery stores and warehouses, clean buildings, and deliver mail while the pandemic raged around them. Earning hourly wages without paid sick leave, they couldn't afford to miss shifts even when symptomatic. They faced risky commutes on crowded public transportation while more privileged people teleworked from the safety of isolation. "There's nothing about Blackness that makes you more prone to COVID," says Nicolette Louissaint, the executive director of Healthcare

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Ready, a nonprofit that works to strengthen medical supply chains. Instead, existing inequities stack the odds in favor of the virus.

Native Americans were similarly vulnerable. A third of the people in the Navajo Nation can't easily wash their hands, because they've been embroiled in long-running negotiations over the rights to the water on their own lands. Those with water must contend with runoff from uranium mines. Most live in cramped multigenerational homes, far from the few hospitals that service a 17-million-acre reservation. As of mid-May, the Navajo Nation had higher rates of COVID19 infections than any U.S. state.

Americans often misperceive historical inequities as personal failures. Stephen Huffman, a Republican state senator and doctor in Ohio, suggested that Black Americans might be more prone to COVID19 because they don't wash their hands enough, a remark for which he later apologized. Republican Senator Bill Cassidy of Louisiana, also a physician, noted that Black people have higher rates of chronic disease, as if this were an answer in itself, and not a pattern that demanded further explanation.

Clear distribution of accurate information is among the most important defenses against an epidemic's spread. And yet the largely unregulated, social-media-based communications infrastructure of the 21st century almost ensures that misinformation will proliferate fast. "In every outbreak throughout the existence of social media, from Zika to Ebola, conspiratorial communities immediately spread their content about how it's all caused by some government or pharmaceutical company or Bill Gates," says Renée DiResta of the Stanford Internet Observatory, who studies the flow of online information. When COVID19 arrived, "there was no doubt in my mind that it was coming."

Sure enough, existing conspiracy theories—George Soros! 5G! Bioweapons!—were repurposed for the pandemic. An infodemic of falsehoods spread alongside the actual virus. Rumors coursed through online platforms that are designed to keep users engaged, even if that means feeding them content that is polarizing or untrue. In a national crisis, when people need to act in concert, this is calamitous. "The social internet as a system is broken," DiResta told me, and its faults are readily abused.

Beginning on April 16, DiResta's team noticed growing online chatter about Judy Mikovits, a discredited researcher turned anti-vaccination champion. Posts and videos cast Mikovits as a whistleblower who claimed that the new coronavirus was made in a lab and described Anthony Fauci

of the White House's coronavirus task force as her nemesis. Ironically, this conspiracy theory was nested inside a larger conspiracy—part of an orchestrated PR campaign by an anti-vaxxer and QAnon fan with the explicit goal to "take down Anthony Fauci." It culminated in a slickly produced video called *Plandemic*, which was released on May 4. More than 8 million people watched it in a week.

Doctors and journalists tried to debunk Plandemic's many misleading claims, but these efforts spread less successfully than the video itself. Like pandemics, infodemics quickly become uncontrollable unless caught early. But while health organizations recognize the need to surveil for emerging diseases, they are woefully unprepared to do the same for emerging conspiracies. In 2016, when DiResta spoke with a CDC team about the threat of misinformation, "their response was: 'That's interesting, but that's just stuff that happens on the internet.'"

Rather than countering misinformation during the pandemic's early stages, trusted sources often made things worse. Many health experts and government officials downplayed the threat of the virus in January and February, assuring the public that it posed a low risk to the U.S. and drawing comparisons to the ostensibly greater threat of the flu. The WHO, the CDC, and the U.S. surgeon general urged people not to wear masks, hoping to preserve the limited stocks for health-care workers. These messages were offered without nuance or acknowledgement of uncertainty, so when they were reversed—the virus is worse than the flu; wear masks—the changes seemed like befuddling flip-flops.

The media added to the confusion. Drawn to novelty, journalists gave oxygen to fringe anti-lockdown protests while most Americans quietly stayed home. They wrote up every incremental scientific claim, even those that hadn't been verified or peer-reviewed.

There were many such claims to choose from. By tying career advancement to the publishing of papers, academia already creates incentives for scientists to do attention-grabbing but irreproducible work. The pandemic strengthened those incentives by prompting a rush of panicked research and promising ambitious scientists global attention.

In March, a small and severely flawed French study suggested that the antimalarial drug hydroxychloroquine could treat COVID19. Published in a minor journal, it likely would have been ignored a decade ago. But in 2020, it wended its way to Donald Trump via a chain of credulity that included Fox News, Elon Musk, and Dr. Oz. Trump spent months touting the drug as a miracle cure despite mounting evidence to the contrary, causing

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shortages for people who actually needed it to treat lupus and rheumatoid arthritis. The hydroxychloroquine story was muddied even further by a study published in a top medical journal, *The Lancet,* that claimed the drug was not effective and was potentially harmful. The paper relied on suspect data from a small analytics company called Surgisphere, and was retracted in June.**

Science famously self-corrects. But during the pandemic, the same urgent pace that has produced valuable knowledge at record speed has also sent sloppy claims around the world before anyone could even raise a skeptical eyebrow. The ensuing confusion, and the many genuine unknowns about the virus, has created a vortex of fear and uncertainty, which grifters have sought to exploit. Snake-oil merchants have peddled ineffectual silver bullets (including actual silver). Armchair experts with scant or absent qualifications have found regular slots on the nightly news. And at the center of that confusion is Donald Trump.

During a pandemic, leaders must rally the public, tell the truth, and speak clearly and consistently. Instead, Trump repeatedly contradicted public-health experts, his scientific advisers, and himself. He said that "nobody ever thought a thing like [the pandemic] could happen" and also that he "felt it was a pandemic long before it was called a pandemic." Both statements cannot be true at the same time, and in fact neither is true.

A month before his inauguration, I wrote that "the question isn't whether [Trump will] face a deadly outbreak during his presidency, but when." Based on his actions as a media personality during the 2014 Ebola outbreak and as a candidate in the 2016 election, I suggested that he would fail at diplomacy, close borders, tweet rashly, spread conspiracy theories, ignore experts, and exhibit reckless self-confidence. And so he did.

No one should be shocked that a liar who has made almost 20,000 false or misleading claims during his presidency would lie about whether the U.S. had the pandemic under control; that a racist who gave birth to birtherism would do little to stop a virus that was disproportionately killing Black people; that a xenophobe who presided over the creation of new immigrant-detention centers would order meatpacking plants with a substantial immigrant workforce to remain open; that a cruel man devoid of empathy would fail to calm fearful citizens; that a narcissist who cannot stand to be upstaged would refuse to tap the deep well of experts at his disposal; that a scion of nepotism would hand control of a shadow coronavirus task force to his unqualified son-in-law; that an

armchair polymath would claim to have a "natural ability" at medicine and display it by wondering out loud about the curative potential of injecting disinfectant; that an egotist incapable of admitting failure would try to distract from his greatest one by blaming China, defunding the WHO, and promoting miracle drugs; or that a president who has been shielded by his party from any shred of accountability would say, when asked about the lack of testing, "I don't take any responsibility at all."

Trump is a comorbidity of the COVID19 pandemic. He isn't solely responsible for America's fiasco, but he is central to it. A pandemic demands the coordinated efforts of dozens of agencies. "In the best circumstances, it's hard to make the bureaucracy move quickly," Ron Klain said. "It moves if the president stands on a table and says, 'Move quickly.' But it *really* doesn't move if he's sitting at his desk saying it's not a big deal."

In the early days of Trump's presidency, many believed that America's institutions would check his excesses. They have, in part, but Trump has also corrupted them. The CDC is but his latest victim. On February 25, the agency's respiratory-disease chief, Nancy Messonnier, shocked people by raising the possibility of school closures and saying that "disruption to everyday life might be severe." Trump was reportedly enraged. In response, he seems to have benched the entire agency. The CDC led the way in every recent domestic disease outbreak and has been the inspiration and template for public-health agencies around the world. But during the three months when some 2 million Americans contracted COVID19 and the death toll topped 100,000, the agency didn't hold a single press conference. Its detailed guidelines on reopening the country were shelved for a month while the White House released its own uselessly vague plan.

Again, everyday Americans did more than the White House. By voluntarily agreeing to months of social distancing, they bought the country time, at substantial cost to their financial and mental well-being. Their sacrifice came with an implicit social contract—that the government would use the valuable time to mobilize an extraordinary, energetic effort to suppress the virus, as did the likes of Germany and Singapore. But the government did not, to the bafflement of health experts. "There are instances in history where humanity has really moved mountains to defeat infectious diseases," says Caitlin Rivers, an epidemiologist at the Johns Hopkins Center for Health Security. "It's appalling that we in the U.S. have not summoned that energy around COVID19."

Instead, the U.S. sleepwalked into the worst possible scenario: People suffered all the debilitating effects of a lockdown with few of the benefits.

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Most states felt compelled to reopen without accruing enough tests or contact tracers. In April and May, the nation was stuck on a terrible plateau, averaging 20,000 to 30,000 new cases every day. In June, the plateau again became an upward slope, soaring to record-breaking heights.

Trump never rallied the country. Despite declaring himself a "wartime president," he merely presided over a culture war, turning public health into yet another politicized cage match. Abetted by supporters in the conservative media, he framed measures that protect against the virus, from masks to social distancing, as liberal and anti-American. Armed anti-lockdown protesters demonstrated at government buildings while Trump egged them on, urging them to "LIBERATE" Minnesota, Michigan, and Virginia. Several public-health officials left their jobs over harassment and threats.

It is no coincidence that other powerful nations that elected populist leaders—Brazil, Russia, India, and the United Kingdom—also fumbled their response to COVID19. "When you have people elected based on undermining trust in the government, what happens when trust is what you need the most?" says Sarah Dalglish of the Johns Hopkins Bloomberg School of Public Health, who studies the political determinants of health.

"Trump is president," she says. "How could it go well?"

THE COUNTRIES THAT fared better against COVID19 didn't follow a universal playbook. Many used masks widely; New Zealand didn't. Many tested extensively; Japan didn't. Many had science-minded leaders who acted early; Hong Kong didn't—instead, a grassroots movement compensated for a lax government. Many were small islands; not large and continental Germany. Each nation succeeded because it did enough things right.

Meanwhile, the United States underperformed across the board, and its errors compounded. The dearth of tests allowed unconfirmed cases to create still more cases, which flooded the hospitals, which ran out of masks, which are necessary to limit the virus's spread. Twitter amplified Trump's misleading messages, which raised fear and anxiety among people, which led them to spend more time scouring for information on Twitter. Even seasoned health experts underestimated these compounded risks. Yes, having Trump at the helm during a pandemic was worrying, but it was tempting to think that national wealth and technological superiority would save America. "We are a rich country, and we think we can stop any infectious disease because of that," says Michael Osterholm, the director of

the Center for Infectious Disease Research and Policy at the University of Minnesota. "But dollar bills alone are no match against a virus."

COVID-19 is an assault on America's body, and a referendum on the ideas that animate its culture.

Public-health experts talk wearily about the panic-neglect cycle, in which outbreaks trigger waves of attention and funding that quickly dissipate once the diseases recede. This time around, the U.S. is *already* flirting with neglect, before the panic phase is over. The virus was never beaten in the spring, but many people, including Trump, pretended that it was. Every state reopened to varying degrees, and many subsequently saw record numbers of cases. After Arizona's cases started climbing sharply at the end of May, Cara Christ, the director of the state's health-services department, said, "We are not going to be able to stop the spread. And so we can't stop living as well." The virus may beg to differ.

At times, Americans have seemed to collectively surrender to COVID19. The White House's coronavirus task force wound down. Trump resumed holding rallies, and called for *less* testing, so that official numbers would be rosier. The country behaved like a horror-movie character who believes the danger is over, even though the monster is still at large. The long wait for a vaccine will likely culminate in a predictable way: Many Americans will refuse to get it, and among those who want it, the most vulnerable will be last in line.

Still, there is some reason for hope. Many of the people I interviewed tentatively suggested that the upheaval wrought by COVID19 might be so large as to permanently change the nation's disposition. Experience, after all, sharpens the mind. East Asian states that had lived through the SARS and MERS epidemics reacted quickly when threatened by SARSCoV2, spurred by a cultural memory of what a fast-moving coronavirus can do. But the U.S. had barely been touched by the major epidemics of past decades (with the exception of the H1N1 flu). In 2019, more Americans were concerned about terrorists and cyberattacks than about outbreaks of exotic diseases. Perhaps they will emerge from this pandemic with immunity both cellular and cultural.

There are also a few signs that Americans are learning important lessons. A June survey showed that 60 to 75 percent of Americans were still practicing social distancing. A partisan gap exists, but it has narrowed. "In public-opinion polling in the U.S., high-60s agreement on anything is an amazing accomplishment," says Beth Redbird, a sociologist at Northwestern University, who led the survey. Polls in May also showed

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that most Democrats and Republicans supported mask wearing, and felt it should be mandatory in at least some indoor spaces. It is almost unheard-of for a public-health measure to go from zero to majority acceptance in less than half a year. But pandemics are rare situations when "people are desperate for guidelines and rules," says Zoë McLaren, a health-policy professor at the University of Maryland at Baltimore County. The closest analogy is pregnancy, she says, which is "a time when women's lives are changing, and they can absorb a ton of information. A pandemic is similar: People are actually paying attention, and learning."

Redbird's survey suggests that Americans indeed sought out new sources of information—and that consumers of news from conservative outlets, in particular, expanded their media diet. People of all political bents became more dissatisfied with the Trump administration. As the economy nose-dived, the health-care system ailed, and the government fumbled, belief in American exceptionalism declined. "Times of big social disruption call into question things we thought were normal and standard," Redbird told me. "If our institutions fail us here, in what ways are they failing elsewhere?" And whom are they failing the most?

Americans were in the mood for systemic change. Then, on May 25, George Floyd, who had survived COVID19's assault on his airway, asphyxiated under the crushing pressure of a police officer's knee. The excruciating video of his killing circulated through communities that were still reeling from the deaths of Breonna Taylor and Ahmaud Arbery, and disproportionate casualties from COVID19. America's simmering outrage came to a boil and spilled into its streets.

Defiant and largely cloaked in masks, protesters turned out in more than 2,000 cities and towns. Support for Black Lives Matter soared: For the first time since its founding in 2013, the movement had majority approval across racial groups. These protests were not about the pandemic, but individual protesters had been primed by months of shocking governmental missteps. Even people who might once have ignored evidence of police brutality recognized yet another broken institution. They could no longer look away.

It is hard to stare directly at the biggest problems of our age. Pandemics, climate change, the sixth extinction of wildlife, food and water shortages—their scope is planetary, and their stakes are overwhelming. We have no choice, though, but to grapple with them. It is now abundantly clear what happens when global disasters collide with historical negligence.

COVID19 is an assault on America's body, and a referendum on the ideas that animate its culture. Recovery is possible, but it demands radical introspection. America would be wise to help reverse the ruination of the natural world, a process that continues to shunt animal diseases into human bodies. It should strive to prevent sickness instead of profiting from it. It should build a health-care system that prizes resilience over brittle efficiency, and an information system that favors light over heat. It should rebuild its international alliances, its social safety net, and its trust in empiricism. It should address the health inequities that flow from its history. Not least, it should elect leaders with sound judgment, high character, and respect for science, logic, and reason.

The pandemic has been both tragedy and teacher. Its very etymology offers a clue about what is at stake in the greatest challenges of the future, and what is needed to address them. *Pandemic. Pan* and *demos*. All people.

theatlantic.com, 4 August 2020

https://www.theatlantic.com

This dairy farmer figured out how to power your BMW with cow manure

2020-08-04

Since taking over management of the family's California dairy farm, in 1977, Albert Straus has used it as a force for change, becoming the first 100% certified organic, zero-waste, non-GMO creamery in North America, processing milk from about a dozen partner farms. Since 2004, Straus Family Creamery has been using bio "digesters" to convert methane produced from manure into biogas, which fuels an on-farm generator cranking out 350,000 kilowatt-hours of certified renewable electricity annually—enough for him to sell the excess back to the local utility. Last November, Straus entered into a first-of-its-kind agreement with carmaker BMW Group to supply cow-to-car electricity for BMW's electric vehicles in California. The agreement helps BMW qualify for state carbon credits and pays farmers up to 10 times what they'd earn through regular power-purchase agreements with a public utility. (Any dairy farmer in the state can participate.) "Animals have an essential role in reversing climate change," says Straus, who is pioneering a program on the farm to feed locally harvested red seaweed to cows.

fastcompany.com, 4 August 2020

https://www.fastcompany.com

"Animals have an essential role in reversing climate change," says Straus, who is pioneering a program on the farm to feed locally harvested red seaweed to cows.

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Mercury makes it deep into marine trenches

2020-08-04

Mercury pollution, already known to be widespread, has reached previously unplumbed depths. Researchers sampled shrimp-like amphipods from the bottom of three deep-sea trenches and found high concentrations of mercury and methylmercury—higher even than many other aquatic environments (*Environ. Sci. Technol. Lett.* 2020, DOI: 10.1021/acs.estlett.0c00299).

The study "provides a very unique dataset," says Celia Y. Chen, an aquatic ecologist and director of Dartmouth University's Toxic Metals Superfund Research Program, who was not involved with the work. She highlights the difficulty of collecting samples from this "very understudied ecosystem."

Marine trenches—long, V-shaped chasms slashed deep into the seafloor—are some of the most remote ocean habitats, home to bizarre and little-studied organisms. The ecosystems along the bottom of these trenches, which lie as far as 11 km below the surface, thrive on dead organisms and particulate matter funneled down from above.

Such sustenance can also deliver mercury, this study shows.

Most mercury pollution is launched into the atmosphere through fossil-fuel burning and mining, then redeposits around the globe. Methylmercury, formed in aquatic ecosystems when microbes take up and methylate deposited elemental mercury, is a potent neurotoxin that can bioaccumulate in marine food webs. Some apex predators can accumulate methylmercury in concentrations up to a million times that of seawater, says study coauthor Maodian Liu, now a postdoctoral fellow at Yale University.

To better understand the extent of mercury contamination and bioaccumulation in deep-sea ecosystems, the researchers, led by Xuejun Wang of Peking University and Yunping Xu of Shanghai Ocean University, sampled sediment and collected three species of amphipods from four locations 6,990–10,840 m below the surface of the ocean.

The team found that average methylmercury concentrations in amphipods from trenches were roughly three times as high as those of amphipods from freshwater environments and more than twice as high as those found in amphipods in coastal oceans. The average total mercury level in amphipods from marine trenches was more than seven times that

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of amphipods off the coast of New Jersey, one of the most industrialized and highly populated areas in North America.

Analyzing specific fatty acids, which can provide information on an organism's diet, in the amphipods supported the researchers' suspicions that one of the primary sources of mercury in deep-sea trenches is input of carrion from surface ocean environments.

The high amphipod methylmercury levels found in this study also help explain high concentrations previously reported in some deep-sea fish species that feed on the amphipods, says coauthor Wenjie Xiao. Such high contamination might be common throughout deep-sea food webs, he adds.

The study is "a fascinating and significant contribution," says Lisa A. Levin, a biological oceanographer at Scripps Institution of Oceanography. "This paper furthers our understanding of the reach of this potent neurotoxin and forces the realization that what should be the most pristine of earth's environments—the ultradeep ocean—is in fact highly vulnerable to contamination."

cen.acs.org, 4 August 2020

https://www.cen.acs.org

Survivors of Covid-19 show increased rate of psychiatric disorders, study finds

2020-08-04

More than half of people who received hospital treatment for Covid-19 were found to be suffering from a psychiatric disorder a month later, a study has found.

Out of 402 patients monitored after being treated for the virus, 55% were found to have at least one psychiatric disorder, experts from San Raffaele hospital in Milan found. The results, based on clinical interviews and self-assessment questionnaires, showed post-traumatic stress disorder (PTSD) in 28% of cases, depression in 31% and anxiety in 42%. Additionally, 40% of patients had insomnia and 20% had obsessive-compulsive (OC) symptoms.

The findings will increase concerns about the psychological effects of the virus. The paper, published on Monday in the journal Brain, Behavior and Immunity, says: "PTSD, major depression, and anxiety are all high-burden

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non-communicable conditions associated with years of life lived with disability.

"Considering the alarming impact of Covid-19 infection on mental health, the current insights on inflammation in psychiatry, and the present observation of worse inflammation leading to worse depression, we recommend to assess psychopathology of Covid-19 survivors and to deepen research on inflammatory biomarkers, in order to diagnose and treat emergent psychiatric conditions."

The study of 265 men and 137 women found that women – who are less likely to die from Covid than men – suffered more than men psychologically. Patients with positive previous psychiatric diagnoses suffered more than those without a history of psychiatric disorder. The researchers, led by Dr Mario Gennaro Mazza, said these results were consistent with previous epidemiological studies.

They said psychiatric effects could be caused "by the immune response to the virus itself, or by psychological stressors such as social isolation, psychological impact of a novel severe and potentially fatal illness, concerns about infecting others, and stigma."

Outpatients showed increased anxiety and sleep disturbances, while – perhaps surprisingly – the duration of hospitalisation inversely correlated with symptoms of PTSD, depression, anxiety and OC.

The researchers said: "Considering the worse severity of Covid-19 in hospitalised patients, this observation suggests that less healthcare support could have increased the social isolation and loneliness typical of Covid-19 pandemics."

They said their findings mirrored those from previous studies in outbreaks of coronaviruses, including Sars, where the psychiatric morbidities ranged from 10% to 35% in the post-illness stage.

There have been warnings from UK experts about brain disorders in Covid-19 patients. Problems including brain inflammation, stroke and psychosis have been linked to the virus.

theguardian.com, 4 August 2020

https://www.theguardian.com

Researchers discover new electrocatalyst for turning carbon dioxide into liquid fuel

2020-08-05

Catalysts speed up chemical reactions and form the backbone of many industrial processes. For example, they are essential in transforming heavy oil into gasoline or jet fuel. Today, catalysts are involved in over 80 percent of all manufactured products.

A research team, led by the U.S. Department of Energy's (DOE) Argonne National Laboratory in collaboration with Northern Illinois University, has discovered a new electrocatalyst that converts carbon dioxide (CO_2) and water into ethanol with very high energy efficiency, high selectivity for the desired final product and low cost. Ethanol is a particularly desirable commodity because it is an ingredient in nearly all U.S. gasoline and is widely used as an intermediate product in the chemical, pharmaceutical and cosmetics industries.

"The process resulting from our catalyst would contribute to the circular carbon economy, which entails the reuse of carbon dioxide,» said Di-Jia Liu, senior chemist in Argonne›s Chemical Sciences and Engineering division and a UChicago CASE scientist in the Pritzker School of Molecular Engineering, University of Chicago. This process would do so by electrochemically converting the CO_2 emitted from industrial processes, such as fossil fuel power plants or alcohol fermentation plants, into valuable commodities at reasonable cost.

Today, catalysts are involved in over 80 percent of all manufactured products.

The team's catalyst consists of atomically dispersed copper on a carbon-powder support. By an electrochemical reaction, this catalyst breaks down CO_2 and water molecules and selectively reassembles the broken molecules into ethanol under an external electric field. The electrocatalytic selectivity, or «Faradaic efficiency,» of the process is over 90 percent, much higher than any other reported process. What is more, the catalyst operates stably over extended operation at low voltage.

"With this research, we've discovered a new catalytic mechanism for converting carbon dioxide and water into ethanol," said Tao Xu, a professor in physical chemistry and nanotechnology from Northern Illinois University. "The mechanism should also provide a foundation for development of highly efficient electrocatalysts for carbon dioxide conversion to a vast array of value-added chemicals.»

Because CO_2 is a stable molecule, transforming it into a different molecule is normally energy intensive and costly. However, according to Liu, «We could couple the electrochemical process of CO_2-to-ethanol conversion using our catalyst to the electric grid and take advantage of the low-cost electricity available from renewable sources like solar and wind during off-peak hours." Because the process runs at low temperature and pressure, it can start and stop rapidly in response to the intermittent supply of the renewable electricity.

The team's research benefited from two DOE Office of Science User Facilities at Argonne—the Advanced Photon Source (APS) and Center for Nanoscale Materials (CNM)—as well as Argonne's Laboratory Computing Resource Center (LCRC). "Thanks to the high photon flux of the X-ray beams at the APS, we have captured the structural changes of the catalyst during the electrochemical reaction," said Tao Li, an assistant professor in the Department of Chemistry and Biochemistry at Northern Illinois University and an assistant scientist in Argonne's X-ray Science division. These data along with high-resolution electron microscopy at CNM and computational modeling using the LCRC revealed a reversible transformation from atomically dispersed copper to clusters of three copper atoms each on application of a low voltage. The CO_2-to-ethanol catalysis occurs on these tiny copper clusters. This finding is shedding light on ways to further improve the catalyst through rational design.

"We have prepared several new catalysts using this approach and found that they are all highly efficient in converting CO_2 to other hydrocarbons,» said Liu. "We plan to continue this research in collaboration with industry to advance this promising technology."

phys.org, 5 August 2020

https://www.phys.org

Heavy drinking drove hundreds of thousands of Americans to early graves

2020-08-03

Heavy drinking is robbing Americans of decades of life.

From 2011 to 2015, an average of 93,296 deaths annually could be tied to excessive alcohol use, or 255 deaths per day. Excessive drinking brought death early, typically 29 years sooner than would have been expected.

Excessive drinking brought death early, typically 29 years sooner than would have been expected.

All told, the United States saw 2.7 million years of potential life lost each year, researchers report in the July 31 *Mortality and Morbidity Weekly Report*.

The researchers used a program developed by the U.S. Centers for Disease Control and Prevention that estimates annual deaths and years of potential life lost due to an individual's own or another's excessive drinking. The tool takes into account whether the cause of death is fully attributable to alcohol, such as alcoholic liver cirrhosis, or whether excessive drinking can partially contribute to a condition, such as breast cancer.

Annually, about 51,000 of the deaths were from chronic conditions. The rest were sudden demises such as poisonings that involved another substance along with alcohol or alcohol-related car crashes.

The CDC defines excessive alcohol use as binging — drinking five or more drinks at a time for men, four or more for women — or drinking heavily over the course of the week. Men qualify at 15 or more drinks per week; for women, it's eight or more.

The numbers of deaths and years of life extinguished due to excessive drinking have gone up since the last report. That assessment covered 2006 to 2010 and reported close to 88,000 deaths and 2.5 million lost years annually. Recommendations from the Community Preventive Services Task Force, made up of public health and prevention experts, to stem excessive drinking include raising taxes on alcohol and regulating the number of places that sell alcoholic beverages (*SN: 8/9/17*).

sciencenews.org, 3 August 2020

https://www.sciencenews.org

Hydroxychloroquine can't stop COVID-19, It's time to move on, scientists say

2020-08-02

As a frontline doctor working with COVID-19 patients at Columbia University Medical Center in New York City, Neil Schluger had horrific days.

"I would come into the ward in the morning to make rounds and say to the intern, 'How did we do last night?' And the intern said, 'Well, I had 10 COVID admissions, and three of them have already died.' It was like nothing I've experienced in 35 years of being a physician," Schluger says.

When he first heard about hydroxychloroquine, he hoped it would work for his patients. He and colleagues prescribed the antimalarial drug for 811 of the 1,446 patients hospitalized at the medical center from March 7 to April 8. But the drug didn't seem to help, Schluger and colleagues reported May 17 in the *New England Journal of Medicine*.

As a result, "we stopped giving hydroxychloroquine sometime in April," he says.

As a result, "we stopped giving hydroxychloroquine sometime in April," he says.

And yet the numbers of cases and deaths from COVID-19 in New York City have continued to fall. "If we'd taken away a lifesaving drug, you wouldn't expect that to happen," he says. Instead, Schluger, now a pulmonary critical care doctor and clinical epidemiologist at New York Medical College and Westchester Medical Center in Valhalla, credits old-fashioned public health measures — mask-wearing, staying home, and social distancing — for New York's success against the virus.

Hydroxychloroquine has been tested more than any other potential COVID-19 drug but has repeatedly fallen short of expectations. Although study after study has demonstrated no benefit of hydroxychloroquine for treating people with serious coronavirus infections, some people, including President Donald Trump, still insist the drug has merit. A viral video released July 27 that made the misleading assertion that hydroxychloroquine is an effective treatment for COVID-19 spread like wildfire online.

But the overwhelming majority of scientific evidence doesn't support that claim. It's time to move on from hydroxychloroquine to test other drugs that may have more promise against COVID-19, Schluger and other experts say.

Wrong cells

Initial hope that hydroxychloroquine was useful in fighting the coronavirus stemmed from lab tests showing that the drug inhibits the virus's growth in kidney cells from monkeys by blocking its entry. But it turns out that the virus doesn't enter human lung cells in the same way.

In those initial experiments, researchers tested the drug using African green monkeys' kidney cells, known as Vero cells. Those cells are useful for virologists because they allow growth of a wide variety of viruses, says Stefan Pöhlmann, a virologist at the German Primate Center in Göttingen. But the way SARS-CoV-2, the coronavirus that causes COVID-19, infects monkey kidney cells is different from the way it infects human lung cells, Pöhlmann and colleagues report July 22 in *Nature*.

To infect different types of cells, the coronavirus has at least two major possible routes of entry. In one, the virus's spike protein (the knobby

structures on its surface) attaches to ACE2 protein on the cell membrane, and then an enzyme called TMPRSS2 cuts the spike protein. That process allows the virus to inject its genetic material into the cell, where more copies of the virus are produced.

Cutting a path

The SARS-CoV-2 virus can enter cells through at least two routes, only one of which is known to respond to hydroxychloroquine. Each likely starts with the virus (red) attaching to the ACE2 protein on a host cell's surface. In one pathway (illustrated below, at left), the enzyme TMPRSS2 cuts the spike protein, causing the cellular and viral membranes to fuse and allowing the virus's genetic material to escape into the cell. That route, which is not blocked by hydroxychloroquine, is how the virus infects human lung cells, new studies show.

In another pathway (right), the virus latches onto ACE2 and then the cell engulfs the virus in a compartment called an endosome. To dump its genetic material into the cell, a different enzyme must cut the virus' spike protein. That enzyme, called cathepsin L, is debilitated by hydroxychloroquine in monkey kidney cells, inhibiting an infection.

The second way the virus gets inside cells is via a detour through special cellular compartments called endosomes. After attaching to ACE2, the virus is engulfed by an endosome, but the pathogen needs to get its genetic material out of the compartment and into the main part of the cell. So the spike protein needs to be cleaved by an enzyme to allow the viral and cellular membranes to fuse, releasing the virus's genetic material, says Markus Hoffmann, a virologist also at the German Primate Center.

In Vero cells from monkeys, that enzyme — called cysteine protease cathepsin L, or CatL — performs the fusion-promoting slice. But the enzyme needs a certain level of acidity to make the cut. Hydroxychloroquine and chloroquine increase the pH too much for CatL to snip the spike protein, thereby inhibiting infection.

But when Hoffman, Pöhlmann and colleagues tested the drugs in human lung cells grown in lab dishes, the virus easily slipped into the cells. That's because in lung cells, SARS-CoV-2 takes the more direct route using TMPRSS2, which isn't found in the monkey cells and which chloroquine and hydroxychloroquine don't inhibit, says Michael Farzan, a virologist and immunologist at Scripps Research Institute in Jupiter, Fla.

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He and colleagues posted a preprint to bioRxiv.org on July 22 also showing that hydroxychloroquine doesn't block how SARS-CoV-2 enters human cells. That data has not yet been reviewed by other scientists for publication in a scientific journal.

Very little, if any, CatL is made in human lung cells, Pöhlmann says. That leaves the virus with mainly the TMPRSS2 route of entry, which is impervious to hydroxychloroquine.

Many other viruses, including the original SARS and MERS coronaviruses, use TMPRSS2 to activate their spike protein. But the TMPRSS2 entryway is much more important for SARS-CoV-2's entry to human lung cells than it was for the original SARS virus, Farzan's study demonstrates. That's because SARS-CoV-2 also uses another enzyme called furin to snip the spike protein (*SN: 3/26/20*).

That furin cleavage spot was not present in the original SARS virus, and may make it easier for SARS-CoV-2 to break into cells. Such furin cleavage sites often help make influenza and other viruses more infectious. In Farzan's study, the furin cuts make the novel coronavirus more dependent on TMPRSS2 for entry, relegating the CatL pathway to a distant plan B.

A compound called camostat mesylate effectively inhibits SARS-CoV-2 entry in cells that make TMPRSS2, both studies found. That drug is being tested against the virus in some clinical trials.

Another study from researchers in France also found that hydroxychloroquine inhibits SARS-CoV-2 infection of Vero cells, but not human lung cells. In addition, the drug did not protect another kind of monkey, cynomolgus macaques, from coronavirus infection, the researchers reported July 22 in *Nature*.

These results indicate the importance of using human lung cells to study the virus, the researchers say. "A lot of these [favorable hydroxychloroquine] studies that came out are sort of meaningless because they were done in the wrong cell [types]," says Katherine Seley-Radtke, a medicinal chemist at the University of Maryland Baltimore County.

Farzan says he doesn't blame anyone for trying hydroxychloroquine first. "We were grasping at straws" at the beginning of the coronavirus outbreak, he says. "There was a good deal of just trying things on people initially … that just ended up being essentially useless."

The new studies' implications are clear, says Seley-Radtke, who was not involved in any of the new studies. "We now have a lot more information about hydroxychloroquine, and it doesn't work. It's not a direct-acting antiviral."

That means chloroquine and hydroxychloroquine are also unlikely to prevent infection with the virus or protect people from developing serious illnesses, as some researchers had proposed. Some studies are still testing the drugs to determine whether they can prevent infection or lessen the risk of developing serious illness, although results of one such study conducted at the University of Minnesota were not encouraging (*SN: 6/4/20*). That study showed that hydroxychloroquine did not prevent coronavirus infections after exposure to the virus.

No help for the hospitalized

Antiviral activity aside, researchers had hoped hydroxychloroquine could calm the overactive immune system response, called a "cytokine storm," that leads to tissue damage and even death in some COVID-19 patients. The reason for that hope is that hydroxychloroquine is also used to treat rheumatoid arthritis and lupus, and can help regulate the immune system in those patients (*SN: 5/22/20*).

But hydroxychloroquine and chloroquine have not panned out as effective COVID-19 therapies, Shmuel Shoham, an infectious-disease specialist at Johns Hopkins School of Medicine, said June 26 during a news conference sponsored by the Infectious Diseases Society of America announcing revised treatment guidelines. The evidence that "has come through is not encouraging that that's going to be a great option," he said. The U.S. Food and Drug Administration has withdrawn its emergency use authorization for hydroxychloroquine (*SN: 6/15/20*), and several large studies have stopped testing the drug for people who are hospitalized with COVID-19.

Compared with a placebo, hydroxychloroquine did not relieve COVID-19 symptoms or prevent people from progressing to severe illness to a statistically meaningful degree, researchers reported July 16 in the *Annals of Internal Medicine*. Similarly, a randomized trial in Brazil of more than 600 COVID-19 patients with mild to moderate symptoms found no statistically meaningful benefit over a placebo of either hydroxychloroquine alone or in combination with another drug called azithromycin, researchers reported July 23 in the *New England Journal of Medicine*.

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Some studies, published after the FDA's withdrawal, have found what appears to be a benefit of taking the drug. At Henry Ford Hospital in Detroit, researchers wanted to know how well the hospital was doing with treating COVID-19 patients. So infectious disease epidemiologist Samia Arshad and colleagues looked back at patient records from March 10 to May 2. Patients with moderate to severe disease were given hydroxychloroquine, and, if a bacterial infection was suspected, also got the antibiotic azithromycin. Overall, about 18 percent of COVID-19 patients died. That percentage was lower in the hydroxychloroquine group, with 13.5 percent dying, Arshad and colleagues reported July 1 in the *International Journal of Infectious Diseases*. But about 20 percent of those who got hydroxychloroquine and azithromycin died.

Arshad says their results may differ from those of studies that didn't show a benefit because the Henry Ford patients got treatment earlier (91 percent got the drug within 48 hours of being admitted to the hospital) and because a treatment algorithm the doctors used didn't allow anyone with cardiac risk factors to take the drug. Anyone who did get the drug was closely monitored. The hospital stopped using the drug after the FDA withdrew emergency use authorization.

Another retrospective study of almost 6,500 COVID-19 patients in New York City from March 13 to April 17 also found a reduced risk of death among people taking hydroxychloroquine, researchers reported June 30 in the *Journal of General Internal Medicine*.

That's not enough to recommend hydroxychloroquine for use against the coronavirus, says David Hsieh, an oncologist at the University of Texas Southwestern Medical Center in Dallas, who has been examining clinical trials of COVID-19 around the world.

He and his brother Antony Hsieh of the Perelman School of Medicine at the University of Pennsylvania, and UT Southwestern colleague Magdalena Espinoza found that hydroxychloroquine has had more coronavirus clinical trials devoted to it and is mentioned in more publications than any other drug or therapy directed at COVID-19, the researchers reported July 4 in *Med*.

First place

Hydroxychloroquine has been studied more than any other potential COVID-19 drug, a search of scientific literature databases reveals. Preclinical studies, which examined a drug's activity in animals or in cells

grown in lab dishes, are indicated in red. Other studies, including results of clinical trials, are shown in blue.

Studies that look back at outcomes, such as retrospective studies and meta-analyses (studies that combine data from multiple studies) are great for generating hypotheses, Hsieh says. "But we're in real danger if we start using [them] to change our practice."

That's because in retrospective or observational studies, there is no guarantee that patients who got the drug and those that didn't are the same. In the Henry Ford trial, patients that got hydroxychloroquine were about five years younger, on average, than those who didn't, Schluger says. "We know that age is the single strongest predictor of mortality from this illness."

And the patients who got hydroxychloroquine were also more likely than those not on the antimalarial drug to have also gotten steroids, which other studies have demonstrated can be lifesaving (*SN: 7/22/20*). So the patients who got the drug were different from those who didn't in important ways.

Researchers "who have looked at the data carefully consider the hydroxychloroquine story to be pretty much over," Schluger says. "It would be a shame if we weren't trying other potentially promising things because we were hung up chasing down something for which there's a lot of evidence now that it doesn't really do much."

sciencenews.org, 2 August 2020

https://www.sciencenews.org

Coronavirus buy-local trend driving demand for Australian-grown medicinal cannabis

2020-08-02

An Australian medical cannabis company says the rise of the buy-local movement during COVID-19 driven has up demand for locally produced cannabinoid products.

Little Green Pharma (LGP) is based at a secret location in WA's South West and has experienced a boom in sales since the pandemic began.

Chief operating officer Paul Long said the trend was being driven by a range of social and economic factors stemming from the health crisis and not necessarily by a spike in patient numbers.

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Demand for cannabis products had been growing exponentially before the health crisis, Mr Long said

"I think COVID has put pressure on supply chains, so companies that have been importing products from outside Australia, and particularly from Canada ... have seen some challenges, with some outages of product," he said.

"There's also been this feel of 'let's support locally grown, let's support Australian business', so we've benefited from that throughout COVID and seen an uplift during the last two to three months.

"Since we started in August 2018 we've had just over 6,500 patients, but in the last three months alone we've seen 1,300 new patients."

Demand for cannabis products had been growing exponentially before the health crisis, Mr Long said.

LGP was predominantly focused on the potential of the domestic market, despite export prospects ramping up in Europe, including the UK and Germany, he said.

"Expert reports show that medicinal cannabis products get to about 1 to 2 per cent of the population, so if you look at total numbers in Australia, we've got a long way to go in that growth, which is exciting for the industry as a whole."

Matty Moore has been prescribing medicinal cannabis for about 12 months.

While legally restricted from discussing the benefits of medicinal cannabis products for his patients, he said he had been prescribing more than ever.

He said conditions he treated using the medicine included chronic pain, post-traumatic stress disorder, anxiety, cancer pain and insomnia, with patients mostly aged 60 and over.

"It's an increasing part of what I'm doing. Each week I'm seeing more and more new patients, and at some point it will be a significant part of my business."

Dr Moore said the medical profession was becoming more open to prescribing medicinal cannabis as a treatment option.

"The age-old bias is hard to break from last century when cannabis was looked down upon and tightly controlled," he said.

"We're behind on its use, but we're catching up ... it's tightly regulated and that means that we compile evidence and data.

"We're all evidence-based physicians, so as we get more and more evidence, people will be more comfortable prescribing it for their patients."

Government backs industry expansion

With an increasing number of doctors prescribing cannabinoid products for pain relief, medicinal cannabis is tipped to become a billion-dollar business in Australia.

The WA Government wants to ensure the state gets a piece of the pie, recently providing a $300,000 grant to LGP to facilitate an expansion of its manufacturing operations.

LGP now has the capacity to produce up to 110,000 bottles of finished medicine for the Australian market each year.

"We see great potential for CBD production in pharmaceuticals and nutraceuticals," she said.

"[We want] to make sure that we here in WA are leading producers, not just Australia-wide but that we become major players internationally.

"Not only is the industry going to help millions of people around the world ... it's going to drive a brilliant new industry full of interesting jobs here in the South West."

abc.net.au, 2 August 2020

https://www.abc.net.au

Stonehenge: Sarsen stones origin mystery solved

2020-07-29

A test of the metre-long core was matched with a geochemical study of the standing megaliths.

Archaeologists pinpointed the source of the stones to an area 15 miles (25km) north of the site near Marlborough.

English Heritage's Susan Greaney said the discovery was "a real thrill".

The seven-metre tall sarsens, which weigh about 20 tonnes, form all fifteen stones of Stonehenge's central horseshoe, the uprights and lintels of the outer circle, as well as outlying stones.

The monument's smaller bluestones have been traced to the Preseli Hills in Wales, but the sarsens had been impossible to identify until now.

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Archaeologists pinpointed the source of the stones to an area 15 miles (25km) north of the site near Marlborough.

The return of the core, which was removed during archaeological excavations in 1958, enabled archaeologists to analyse its chemical composition.

No-one knew where it was until Robert Phillips, 89, who was involved in those works, decided to return part of it last year.

Researchers first carried out x-ray fluorescence testing of all the remaining sarsens at Stonehenge which revealed most shared a similar chemistry and came from the same area.

They then analysed sarsen outcrops from Norfolk to Devon and compared their chemical composition with the chemistry of a piece of the returned core.

English Heritage said the opportunity to do a destructive test on the core proved "decisive", as it showed its composition matched the chemistry of sarsens at West Woods, just south of Marlborough.

Prof David Nash from Brighton University, who led the study, said: "It has been really exciting to harness 21st century science to understand the Neolithic past, and finally answer a question that archaeologists have been debating for centuries.

'Substantial stones'

"Each outcrop was found to have a different geochemical signature, but it was the chance to test the returned core that enabled us to determine the source area for the Stonehenge sarsens."

Ms Greaney said: "To be able to pinpoint the area that Stonehenge's builders used to source their materials around 2,500 BC is a real thrill.

"While we had our suspicions that Stonehenge's sarsens came from the Marlborough Downs, we didn't know for sure, and with areas of sarsens across Wiltshire, the stones could have come from anywhere.

"They wanted the biggest, most substantial stones they could find and it made sense to get them from as nearby as possible."

Ms Greaney added the evidence highlights "just how carefully considered and deliberate the building of this phase of Stonehenge was"

bbc.com, 29 July 2020

https://www.bbc.com

Remains of a 10,000-year-old woolly mammoth pulled from Siberian lake

2020-08-04

Russian scientists are poring over the uniquely well-preserved bones of a 10,000-year-old woolly mammoth after completing the operation to pull them from the bottom of a Siberian lake.

Experts spent five days scouring the silt of Lake Pechenelava-To in the remote Yamal peninsula for the remains, which include tendons, skin and even excrement, after they were spotted by local residents. About 90% of the animal has been retrieved during two expeditions.

Such finds are happening with increasing regularity in Siberia as climate change warms the Arctic at a faster pace than the rest of the world, thawing the ground in some areas long locked in permafrost.

The woolly mammoth will probably be named Tadibe, after the family who discovered the adult animal, which is thought to have been a male between 15 and 20 years old, and and about 10ft (three metres) tall.

Andrey Gusev from the Centre of Arctic Research, said the preservation of the animal was unique, with the lower spine still connected by tendons and skin, but that the retrieval operation was painstaking because the remaining bones were jumbled up.

"We assumed that the bones were preserved in the anatomical order. But the first and the second days of our expedition showed that it was true only about the back part of the skeleton," he said. "The rest of the bones were in such chaotic order that it was impossible to guess where they were."

Tendons and skin can been seen on the woolly mammoth remains. Photograph: mvk.yanao/ Instagram

Evgenia Khozyainova from the Shemanovsky museum in Salekhard, said: "We have one front and one hind foot well-preserved, with tendons, soft tissues and pieces of skin. Also we have sacrum with adjacent vertebrae, including the tail preserved with tendons and a big piece of skin."

Of particular interest is the excrement, or coprolite, because it will contain details of the animal's diet, as well as pollen and other environmental clues.

About 90% of the animal has been retrieved during two expeditions.

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The cause of the mammoth's death is not clear yet as no signs of injuries were found on the bones.

Researchers have found mammoth fossils dating from up to 30,000 years ago in Russia.

Scientists circulated images in December of a prehistoric puppy, thought to be 18,000 years old, that was found in the permafrost region of Russia's Far East in 2018.

theguardian.com, 4 August 2020

https://www.theguardian.com

'Climate change-resistant' apple that can keep its colour and crunch could be grown in Australia

2020-08-01

An apple touted as the first in the world to be resistant to climate change could be grown in Australia as early as next year.

The apple, known as HOT84A1, has been bred to withstand the world's hottest and driest conditions and is the result of 18 years' work between researchers in Spain and New Zealand.

New Zealand-headquartered T&G Global officially launched the fruit and said it was important the industry found ways to continue to grow fruit in a changing climate.

"In hotter drier climates typically, apples don't get that lovely red colour and they also don't get that crispy crunch which makes them so nice to eat," said Peter Landon-Lane, T&G's director of innovation and technical.

"Apples have a huge amount of natural genetic diversity so to get an apple that holds in hotter, drier climates it's been about 18 years to create an overnight success."

Trees trialled in hot, dry Spain

Mr Landon-Lane said with a warming climate, both countries found they had a common problem of how to get apples to grow well in hot, dry regions and that Spain was a perfect place for their first trial.

"In northern Spain the temperatures can get up to 45 degrees and that's pretty hot to get an apple with good red colour and a good crisp crunch," he said.

"Apples have a huge amount of natural genetic diversity so to get an apple that holds in hotter, drier climates it's been about 18 years to create an overnight success."

"The trees also need to have good tolerance to drought."

Could be trialled in Australia next year

Researchers started with about 10,000 seedlings, also needing to combine attributes like pest and disease resistance, good size fruit and good shelf life.

Over 18 years those seedlings where whittled down to about 10.

"What you're trying to do is get all of the traits of interest in a particular tree that produces consistent, good fruit," Mr Landon-Lane said.

"We've all become a lot more aware of the challenges of climate change and so innovation in terms of new and improved genetics is part of a continuous innovation process.

"Here we are still able to produce good, healthy, nutritious food despite those challenges."

The variety is in quarantine in Australia and Mr Landon-Lane said he hoped growers would be able to trial the hot apple as early as next year.

The company is still looking for a commercial name for the red, hot apple, once it can be sold in supermarkets.

abc.net.au, 1 August 2020

https://www.abc.net.au

How anglerfish fuse their bodies without unleashing an immune storm

2020-07-31

Many of us have dealt with a clingy boyfriend or girlfriend, but some male deep-sea anglerfish take things to the extreme: In certain species, the male latches onto the much larger female, permanently fusing his body with hers (above). This bizarre mating ritual has long puzzled scientists, as merging two individuals' blood and tissue should trigger an adverse immune reaction. Now, a new study suggests some males have lost parts of their immune system to compensate, *The New York Times* reports. Anglerfish that employ the most extreme form of sexual parasitism–where multiple males can conjoin with one female–could not produce functional antibodies and T cells, which typically fight off foreign invaders and differentiate an individual's own cells from unfamiliar ones. Monogamous anglerfish species had less extreme versions of these dampened immune

In certain species, the male latches onto the much larger female, permanently fusing his body with hers…

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systems, the team reports this week in *Science*, as these fish could still produce certain types of antibodies.

sciencemag.org, 31 July 2020

https://www.sciencemag.org

Eastern hooded plover number on the rise thanks to volunteers, fewer visitors

2020-08-02

A vulnerable shorebird found across southern Australia is showing good signs of its numbers increasing as fewer people visit beaches because of travel restrictions.

A recent survey of eastern hooded plovers in South Australia and Victoria showed a noticeable boost in the numbers of breeding pairs and fledglings.

Ninety-three breeding pairs were spotted by BirdLife Australia volunteers in SA, along with 56 fledglings.

A fledgling is the age in which a plover can begin to fly and evade predators.

The highest adult-to-chick ratio was found on Yorke Peninsula which recorded 0.77 fledglings per pair, above the state average of 0.6.

BirdLife Australia said a ratio between 0.4 and 0.5 would sustain a good population.

Spokesman Kasun Ekanayake said reduced visitor numbers to beaches this year had helped the bird.

"Reduced predator numbers, reduced visitor numbers to beaches, and also birds choosing good spots to nest as well," he said.

"They can nest anywhere between just above the high-tide mark to the dunes — if people don't spot these nests they can easily get stepped on."

The plovers are also vulnerable to human activity on beaches including 4WDs and off-leash dogs.

Mr Ekanayake said plover chicks took 28 days to hatch and another five weeks before they could fly.

He said the chicks spent all their time on the beach.

Ninety-three breeding pairs were spotted by BirdLife Australia volunteers in SA, along with 56 fledglings.

"They have to walk down to the water's edge or the high-tide mark to find food for themselves — the parents don't bring food," he said.

"If there's a lot of disturbance on these beaches in terms of off-leash dogs and humans and vehicles, these birds just hide.

"Chicks can easily starve to death, so that's essentially why their numbers are pretty low."

The survey was carried out by volunteers, who Mr Ekanayake said were essential to the bird thriving.

"When you have more volunteers on the ground, they put fences up, they put nest signs up, so people visiting beaches are made aware of these nests being there.

"Most people do the right thing by putting their dogs on leashes and walking along the water's edge, giving these nests a nice wide berth."

Only 3,000 left

Sally Box, the Commonwealth's Threatened Species Commissioner, said there were only 3,000 eastern hooded plovers left and that their protection was vital.

"Our native species are so precious and an important part of who we are," she said.

"There is wonderful support for the bird, there are lots of people who absolutely love it.

"When we've only got around 3,000 left, we need to do everything we can to protect the really unique species that call Australia home."

Dr Box said the bird's protection was a priority for the Government and part of its Threatened Species Strategy.

"There are four projects that are protecting the hooded plover in Victoria, South Australia and Tasmania," she said.

"Activities like feral animal control and protecting nests and habitats through fencing and signage and community education, and also population monitoring."

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She said the Yorke Peninsula's Great Southern Ark was one project helping to protect the hooded plover.

abc.net.au, 2 August 2020

https://www.abc.net.au

How many house plants do you need to clean the air in a small flat?

2020-07-08

AS YOU may know from my bio, I cohabit my small flat in London with more than 500 plants. I am therefore fascinated by the promise of a plethora of health benefits from gardening in the great indoors. With the current flowering of interest in the hobby, the internet is awash with handy advice for the "10 best air-purifying plants for the home" and species marketed as "Air so pure".

Being a stats geek, I wanted to calculate exactly how much the concentration of plants in my apartment could clean the air. This turned out to be a rather deep rabbit hole…

The seminal work on this subject came from NASA in 1989, after it investigated using plants as filters in space stations. In the study, researchers placed individual plants in tiny chambers filled with air contaminated with volatile organic compounds (VOCs). This group of chemicals includes known carcinogens like the benzene in cigarette smoke and the formaldehyde in paints, and it has been consistently linked to poor health outcomes.

NASA found that there was a significant improvement in the air quality in the chambers over a 24-hour period. Many headlines about how houseplants clean the air, including claims of a 90 per cent reduction in indoor air pollutants, have come from citing this three-decade-old paper.

While the study was well designed, there are a few caveats. Firstly, the "90 per cent" claim isn't for all plants, or even all pollutants, but a single stat for the ability of ivy to remove benzene from the air. Results for other plants and contaminants aren't as impressive, coming in at less than 10 per cent in some cases. So the way this study has been reported – including by me, I regret to say – exaggerates the actual ability of plants to clean the air.

"I like a challenge, but increasing my plant collection tenfold would mean entirely dispensing with furniture"

"I like a challenge, but increasing my plant collection tenfold would mean entirely dispensing with furniture"

Secondly, this experiment only looked at three VOCs. There are hundreds of other VOCs, as well as other ways to measure pollution, such as carbon dioxide and dust levels. So using this study as a definitive measure of overall "purity" in the indoor atmosphere is very tricky indeed.

However, perhaps the most important proviso is that the experiment was done in tiny, sealed chambers. So while it did a great job of demonstrating that plants can filter air, it didn't provide hard evidence that they can filter enough of it in a room setting to keep up with the level at which these pollutants are generated.

Fortunately, since NASA's study, dozens of others have investigated these issues. A review of these studies found that you would need between 10 and 1000 plants per square metre of a building's floor space to remove VOCs at the same rate as happens when air from indoors moves outside anyway.

So in my 50-square-metre flat, I would need around 5000 plants for them to be as good as simply opening a window. I like a challenge, but increasing my existing collection tenfold would mean entirely dispensing with furniture. But what about other measures of air quality? In lockdown, I have been stuck indoors, breathing out about 30 grams of CO_2 per hour. Research published by the University of Birmingham, UK, and the Royal Horticultural Society found that you would need 15 ivy or peace lily plants to reduce just 10 per cent of one person's contribution.

While fitting 150 such plants in my flat is theoretically possible, given some sort of living wall set-up, it should be pointed out that these two species were the best-performing plants in the study. With bromeliads, for instance, you would need more than 1000 specimens to do the same job.

I should have twigged this, really, as even mature tropical trees are estimated to absorb only 22 kilograms of carbon dioxide per year. I would therefore need more than 11 rainforest giants in my flat just to tackle the pollutants I breathe out. That's a lot of plants, even for me.

newscientist.com, 7 July 2020

https://www.newscientist.com

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How tuatara live so long and can withstand cool weather

2020-08-05

A tuatara may look like your average lizard, but it's not. The reptiles are the last survivors of an ancient group of reptiles that flourished when dinosaurs roamed the world. Native to New Zealand, tuatara possess a range of remarkable abilities, including a century-long life span, relative imperviousness to many infectious diseases and peak physical activity at shockingly low temperatures for a reptile. Now, scientists are figuring out how, thanks to the first-ever deciphering, or sequencing, of the tuatara's genetic instruction book.

The research reveals insights into not only the creature's evolutionary relationship with other living reptiles but also into tuatara longevity and their ability to withstand cool weather, researchers report August 5 in *Nature*.

The reptiles are the last survivors of an ancient group of reptiles that flourished when dinosaurs roamed the world.

Technically, the tuatara (*Sphenodon punctatus*) are rhynchocephalians, an order of reptiles that were once widespread during the Mesozoic Era, 66 million to 252 million years ago. But their diversity waned over millions of years, leaving the tuatara as the last of their line (*SN: 10/13/03*). The reptiles have long been of scientific interest because of their unclear evolutionary relationship with other reptiles, as they share traits with lizards and turtles as well as birds.

Tuatara were once found throughout New Zealand, but now survive in the wild mainly on offshore islands and are considered a vulnerable species. The reptiles have suffered from habitat loss and invasive species such as rats, and are especially imperiled by a warming climate (*SN: 7/3/08*).

This peril — combined with the tuatara's cherished status as a *taonga*, or special treasure, to the Indigenous Maori people — led researchers to prioritize compiling the reptile's genome, or genetic instruction book.

In 2012, Neil Gemmell, an evolutionary biologist at the University of Otago in Dunedin, New Zealand, and an international team of researchers began to assemble the tuatara genome, in close partnership with the Indigenous Ngātiwai people. The Ngātiwai are considered *kaitiaki*, or guardians, of the tuatara and were intimately involved in decisions regarding the use of genetic data from the project.

The tuatara's genome is huge, about 5 gigabases, or some 5 billion DNA base pairs in length, the researchers found. That's about two-thirds bigger

than humans' and is "unusually large" for a reptile, says Giulia Pasques, an evolutionary biologist at the University of Colorado Boulder who was not involved with the research. Lizard and snake genomes are usually around 2 gigabases, she says. Bird genomes may be half that size.

Based on the genetic analyses, the researchers confirmed that the tuatara is more closely related to snakes and lizards than to crocodilians, birds or turtles. The researchers estimate that the tuatara and their ancestors diverged from snakes and lizards about 250 million years ago, meaning the group predates even the oldest dinosaurs.

The team identified genes possibly involved in the tuatara's biological quirks including their long lives, which are the longest of any other reptiles besides tortoises. Tuatara have many genes involved in producing selenoproteins, which help protect against aging and cellular deterioration, and have more of these genes than humans do. Such insights may eventually have useful applications for human biology, says coauthor Matthieu Muffato, a comparative genomicist at the European Bioinformatics Institute in Hinxton, England.

Tuatara also appear to have an unusually high number of TRP genes, which are involved in making proteins tied to temperature sensitivity and regulation of body temperature. Those genes may be behind the reptiles' tolerance of cool temperatures, the researchers say. Tuatara have the lowest known optimal body temperature of any reptile, from 16° to 21° Celsius.

Although the new research goes a long way to dispelling some of the mystery surrounding the tuatara, there is much to learn about these scaly enigmas. "Publishing the tuatara genome is like uncovering an ancient book," Muffato says. "We have started analyzing it, and started decoding some of the genetic information, but we are still a long way off from understanding the complete genome."

sciencenews.org, 5 August 2020

https://www.sciencenews.com

Sensory scientists and taste testers create the world's first wagyu flavour wheel

2020-08-09

How many words can you think of to describe the taste, aroma and texture of beef?

"Eight hundred words to describe beef… the general public would put it down to three or four."

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Curiosities
AUG. 14, 2020

Incredibly, a team of tasters and scientists has come up with around 800.

That's as many as are used in the famously wordy world of wine.

Trent Robson is the chef at a leading steak restaurant in Brisbane and even he could only think of a handful.

"Absolutely, it's a surprise," he said.

"Eight hundred words to describe beef… the general public would put it down to three or four."

University of Queensland sensory scientist Heather Smyth had the job of finding all the words to describe wagyu, the most expensive beef produced in Australia.

"I've done some ripper projects over my years but this would be one of my highlights," Dr Smyth said.

"The [tasting] panel were absolutely excited when they heard they got to be tasting wagyu, premium wagyu at that."

Steak that sells for $200 a kilogram

Wagyu is a Japanese beef breed prized for its fine ribbons of marbled fat. It can sell for hundreds of dollars a kilogram.

Dr Smyth's expert palate and nose can detect what most of us can't.

"Roasted, caramelised, brassica, barnyard, white pepper, cheesy and fresh bread crust are [just some] of the flavours we can sense in the beef," she said.

"We know there are around 800 flavour volatiles that are present in wine, which create the diverse bouquets you experience across all the different varieties, so in beef there is a similar number of flavours."

Tasters tried dozens of two-by-two-centimetre squares of wagyu to determine exactly what those 800 words were.

Brisbane woman Tamami Kawasaki is regularly asked to bring her fine palate into the tasting laboratory.

She insists it's a tougher job than you'd think.

"We get to taste all sort of food but sometimes you're so focused, you can't even enjoy it because you have to describe what it's like," she said.

A point of difference in the market

Dr Smyth put the 800 words together on a flavour wheel.

She has done similar wheels for the seafood, coffee, native foods and wine industries, but her wagyu wheel is a world first for the beef industry.

"Genetics of the animal, the region that it was grown, the way it was processed, all of those different factors that create these flavours are then released upon cooking," Dr Smyth said.

Australia's biggest beef producer, Australian Agricultural Company (AACo), commissioned the research.

It believes providing chefs with a tool to better describe the eating qualities of wagyu will help it stand out in the high-end dining market.

"Chefs are always looking for a point of difference and the flavour wheel helps them when they think about planning menus," AACo CEO Hugh Killen said.

"Not all chefs really understand wagyu versus other types of beef," he said.

"The flavour wheel will help explain and give a reference point in terms of flavour and mouthfeel."

Mr Robson said having a deeper understanding of the taste and smell of different cuts with varying fat levels would help chefs match side dishes and wine to deliver a superior dining experience for discerning customers.

"There are definitely beef lovers out there that can taste the difference in the different cuts and different scores," he said.

AACo hopes its investment in the flavour research will boost sales here and overseas, adding to a 20-per-cent increase in wagyu sales last year.

Dr Smyth would like to see more parts of the food industry invest in flavour science.

"Consumers already think Australian product is amazing," she said.

"Let's get on board and try and describe what our point of distinctiveness is in Australia ... and communicate that to the rest of the world."

abc.net.au, 9 August 2020

https://www.abc.net.au

"It is imperative, we always need a good donor list."

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Vet says big dogs urgently needed to donate blood and save other pets

2020-08-09

If you have a big dog under seven years of age who loves their vet, you could help save another dog's life.

Without canine blood banks, vets like Michael Joubert rely on a list of big local donor dogs for when blood is needed in emergency, trauma and disease cases.

"It is a life and death situation where animals will need blood to survive," he said.

"It is imperative, we always need a good donor list."

Dr Joubert said the blood is not stored, but collected fresh when needed, making a well-populated list vital for emergencies.

"If we don't get blood into these dogs, within an hour or two they will die," he said.

"An emergency case comes in, they need blood, we'd then phone up the first available donor.

"That animal comes in immediately, we collect that blood and it would be transfused immediately."

Dr Joubert said if you sign your dog up as a donor, you are literally saving other dogs' lives.

"I had a case a few weeks back where the dog came in on deaths door, our donor came in and within two hours the dog was looking perfect and it went home in a couple of days time," he said.

"If it wasn't for that donor, the dog would have passed away."

Not all dogs are created equal

Donor dogs need to be big enough to not notice the 460 millilitres of blood taken, but also calm enough to handle the procedure.

"We like larger dogs, preferably over 30 kilograms, need to be under seven years old, in good health and of course have to be up to date on their vaccinations."

"They of course of a good temperament, so they need to love coming to the vet.

"It's just safer to do larger animals — we would never do under 20kg — it's too much to take from them.

"Dogs over 30 kilos don't even notice it — after the donation they walk out like nothing has happened."

More than just appreciation

Of course donor dogs are showered with love from the vets and vet nurses, but there is more than that.

Dr Joubert said on top of treats and attention the dogs get their bloods checked out, and a full check up from the vet.

"They get a free health check, blood screen, free tick and flea treatment, so it is beneficial to them as well," he said.

"We always give them a mild sedative as well to make it as stress-free as possible, we don't want fluffies stressing at all.

"We make it a really good experience for them.

"They get to wake up nice and calmly then with lots of cuddles and treats, it's quite a quick and easy process."

abc.net.au, 9 August 2020

https://www.abc.net.au

Why do we develop lifelong immunity to some diseases, but not others?

2020-08-09

Some diseases, like the measles, infect us once and usually grant us immunity for life. For others, like the flu, we have to get vaccinated year after year.

So why do we develop lifelong immunity to some diseases but not others? And where does the novel coronavirus fit into all this?

Whether or not we develop immunity to a disease often depends on our antibodies, which are proteins we produce in response to infection. Antibodies are one of the body's most well-known defenses: They coat invading cells and, in the best case, prevent those invaders from hijacking

It's also what keeps us from getting sick a second time — usually.

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our cells and replicating. After we clear an infection, antibody levels often wane, but at least a few stick around, ready to ramp up production again if that same disease attacks again. That's why an antibody test can tell you if you were infected in the past. It's also what keeps us from getting sick a second time — usually.

"The body doesn't really forget," said Marc Jenkins, an immunologist at the University of Minnesota Medical School. Usually, when we get reinfected with a disease, it's not because our body has lost immunity. We get reinfected either because the pathogen mutated and our immune system no longer recognizes it, or because our bodies tend to mount a much lower immune response, he said.

Take the flu. This is a virus that can change its genes easily, Jenkins said. Just as our immune systems kill off one version of the virus, another emerges that our immune systems don›t recognize. Not all viruses mutate so readily. For example, the polio virus can›t easily change its genome, Jenkins said. That›s why we›ve been so successful at (almost) eradicating it.

The common cold, and other viruses that don't typically get past our upper respiratory tract, reinfect us not necessarily because they mutate rapidly, but because our body doesn't usually produce many antibodies against these pathogens in the first place, said Mark Slifka, an immunologist at the Oregon National Primate Research Center. "Our bodies are not worried about the upper respiratory tract," he said. That's what we're seeing with mild cases of COVID-19. The virus sticks to the upper respiratory tract, where the body does not treat it like a threat. In a 2020 preprint study (meaning it hasn't been peer reviewed yet) published in the database MedRxiv, 10 out of 175 patients who had mild symptoms recovered from COVID-19 without developing detectable antibodies.

For diseases that don't fall into either of these categories — meaning they don't mutate rapidly and they generally prompt a strong immune response — immunity tends to last much longer. A 2007 study published in the New England Journal of Medicine found that it would take more than 200 years for even half of your antibodies to disappear after a measles or a mumps infection. The same study found similar results for Epstein-Barr virus, which causes mono. Still, antibody responses don't always last a lifetime. That same study found that it takes around 50 years to lose half of our chickenpox antibodies, and 11 years to lose half of our tetanus antibodies. That means that without a booster shot, you could theoretically become infected with one of these diseases as an adult.

Scientists still aren't sure why we maintain our antibody responses longer for some diseases compared with others. It's possible that some of these more common diseases, such as chickenpox and mono, actually are reinfecting us more frequently than we realize, but that the antibodies we do have crush the infection before we notice, Jenkins said. And in those cases, the immune system would be at full capacity again and again because of the reinfections. "It keeps our immunity vigilant," he noted. In contrast, "with tetanus, we're probably very rarely getting exposed, we're not stepping on a [dirty] nail very often."

Other scientists point out that the human immune system is trained to target pathogens that "look" a certain way, Slifka said. Bacteria and viruses tend to be symmetrical with a repetitive pattern of proteins across their surfaces. (Think about COVID-19 — it›s a ball with evenly spaced spikes all over it.) One theory suggests that we mount a larger and longer-lasting immune response to more repetitive-looking pathogens. For example, the antibodies we produce against variola, the highly repetitively-structured smallpox virus, last a lifetime. Tetanus, however, isn't repetitive at all. It's the toxin produced by tetanus bacteria, not the bacteria itself, that makes us sick. Based on this theory, it's possible that our bodies aren't as well-trained to target this single, asymmetrical protein, Slifka said.

So, will immunity to the new coronavirus — whether that comes from infection or a vaccine — be as long-lived as our immunity to smallpox, or will we need a new vaccine every year? While it's true that some people aren't mounting large antibody responses, Jenkins is still hopeful for the former. All the evidence both from natural infections and from vaccine trials suggest that most people are making neutralizing antibodies, the variety which prevents viruses from entering our cells, Jenkins said. And unlike the flu, SARS-CoV-2, the virus that causes COVID-19, isn't mutating quickly, Jenkins noted.

"This virus has the features of viruses that we've been very successful in vaccinating against," Jenkins said.

livescience.com, 9 August 2020

https://www.livescience.com

The algorithm then trains itself to get better and better, and fill in the pixelated gaps that occur.

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An AI can make selfies look like they're not selfies

2020-08-07

There's a solution for tourists who are reluctant to hand over their expensive camera phone to a random passer-by to snap a photo. A new computer model promises to make a selfie look like it isn't one.

The technique borrows from the growing field of artificial intelligence that can modify, or 'repose' photos in a realistic way, says Liqian Ma of KU Leuven, Belgium. Reposing uses two or more source images – one in the original position and another in the reposed position – to train its algorithms. But selfies only have a single source image: if it was possible to repose the subject to stand in a portrait taken by a third party, there would be no need for the selfie.

The "unselfie" computer algorithm trawls through a database of portraits, taken from collections of fashion stock images, and uses the information to create a computer-generated model of the person in the selfie. It then searches through the database of portraits to find one with a pose most similar to the pose desired in the modified selfie, before using the computer-generated model to map the original portrait onto the new pose.

The algorithm then trains itself to get better and better, and fill in the pixelated gaps that occur. "It's very sensitive to the imperfections of the generated models," says Ma. Interviewees gathered through Amazon's Mechanical Turk system judged the resulting "unselfied" images better than reposed images created using other AIs developed previously.

The paper uses a "clever approach" to solve the problem, says René Schulte, an AI and spatial computing developer for German company Valorem Reply. "It still has limitations and can still result in unpleasant artefacts in results, but it surely is better than previous approaches."

Ma plans to train the model further to tackle those problems, incorporating a broader range of poses and matching the background to the new limits of the reposed human more smoothly.

newscientist.com, 7 August 2020

https://www.newscientist.com

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Technical Notes

AUG. 14, 2020

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Mitigation of harmful chemical formation from pyrolysis of tobacco waste using CO 2

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Study of occupational exposure to brick kiln emissions on heavy metal burden, biochemical profile, cortisol level and reproductive health risks among female workers at Rawat, Pakistan

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PHARAMACEUTICAL/TOXICOLOGY

Primary results from CECILIA, a global single-arm phase II study evaluating bevacizumab, carboplatin and paclitaxel for advanced cervical cancer

A polycyclic aromatic hydrocarbon-enriched environmental chemical mixture enhances AhR, antiapoptotic signaling and a proliferative phenotype in breast cancer cells

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