The Lord is like a strong tower, where the righteous can go and be safe.
Proverbs 18:10

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Be alert, stand firm in the faith, be brave, be strong.
1 Corinthians 16:13

Cavite adopts COVID-19 anti-discrimination ordinance

The Cavite provincial government has approved the coronavirus disease 2019 (CoVID-19) anti-discrimination ordinance which imposes punishment and penalties against those who will harm, harass, humiliate or shame any COVID-19-affected patient, health workers or front liners serving in the fight against the dreaded disease.

Under the ordinance, which was signed by Governor Juanito Victor C. Remulla Jr. last April 20, an individual found violating the ordinance shall be fined P5,000 or imprisoned for six months as maximum, or both , at the discretion of the court.

If the offender is a public officer, the maximum penalty shall be imposed.

The ordinance stated that it shall be unlawful for any persons to commit acts or utterances against suspected, probable or patients or health workers and front-liners.


REMULLA


REVILLA

The public officer-violator refers to one who refuses or fails to give assistance to patients or front-liners infected or under probable or suspected status due to COVID-19 who intends to return to his place of residence or domicile.

The ordinance was made following reports that some former patients and front-liners have been rejected from their residences even after having recovered from CoVID-19.

Vice Governor Ramon Jolo B. Revilla III and the 19-man Sangguniang Panlalawigan (SP) or Provincial Board unanimously passed the four-page decree entitled "Anti-CoVID-19 Discrimination in the Province of Cavite" last April 6.

The ordinance

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Carmona launches mobile circumcision amid ECQ

In the Philippines, it is a rite of passage for boys to claim that they have reached adulthood when they are circumcised.

And usually, this practice is done just about within this time of the year. However, with the enhanced community quarantine (ECQ), how can the government enforce this project?

In the time where rolling stores are becoming a norm, the local municipality of Carmona, Cavite has a solution to that, with "Oplan Manhood on Wheels" or "Tuli on Wheels".

This annual, medical tradition in Cavite that runs for the months of April or May is ditching long lines of registration in favor of house-to-house calls. It's a project of the Provincial Government of Cavite thru the Office of the Governor Extension in partnership with the Provincial Health Office for Cavite residents.

The vehicle that Cavite is using once served as a transport for the government's dental programs. It was then converted as a mobile bus that goes around its municipalities.

The doctors and nurses in the project, who are noticeably garbed in protective equipment from head-to-toe, can circumcise around 15 to 20 boys each day.

Patients are initially screened by undergoing temperature

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Research reveals a new malaria vaccine candidate

Researchers have discovered a promising new strategy for combating malaria, a mosquito-borne parasite that claims nearly a half-million lives each year.

For a study reported in the journal Nature, researchers screened blood samples from children who had natural immune resistance to severe malaria infection. The study identified an antibody to a particular malaria protein, called PfGARP, that appears to protect resistant children from severe disease. Lab tests showed that antibodies to PfGARP seem to activate a malarial self-destruct mechanism, causing parasitic cells living inside human red blood cells to undergo a form of programmed cell death.

The team is hopeful that vaccinating individuals with PfGARP to generate anti-PfGARP antibodies, or directly infusing anti-PfGARP antibodies, would protect them against severe malaria. The team developed preliminary versions of those vaccines, and testing in nonhuman primates has shown promise, the researchers report.

A breakthrough in estimating the size of a (mostly hidden) network

A newly discovered connection between control theory and network dynamical systems could help estimate the size of a network even when a small portion is accessible.

Understanding the spread of coronavirus may be the most alarming and recent example of a problem that could benefit from a fuller knowledge of network dynamical systems, but scientists and mathematicians have been grappling for years with ways to draw accurate inferences about these complex systems by working with partial data from available measurements.

In a new Physical Review Letters paper, New York University Tandon School of Engineering Institute Professor Maurizio Porfiri demonstrates a profound connection between mathematical control theory and the problem of determining the size of a network dynamical system from the time series of some accessible units. For homogeneous networks — in which every unit plays the same – according a mere 10% of the units could be sufficient to exactly infer the size of the entire network, Porfiri concludes.

But the same approach fails for heterogeneous networks, which are far more common in the field of complex systems. Think of the early stage of the novel coronavirus outbreak, in which every person experienced a widely different range of contacts due to their social and professional lives. Hence, the author recommends prudence in the inference of the size of a network dynamical system from available measurements when information on the nature of the network is lacking.

"From natural to technological settings, network dynamical systems constitute a powerful approach to study collective dynamics. The size of the system is arguably its most fundamental property, but seldom do we have access to such critical information," Porfiri explained. His research provides mathematical proof for a model-free approach published last year by researchers from the University of Oldenburg and the Technical University of Dresden.

(CAVITE... from page 1)

was sponsored by Revilla and 6th District Board Member Kerby J. Salazar and co-sponsored by all the SP members.

The Department of the Interior and Local Government (DILG) earlier called on the local government units to pass and enforce the anti-discrimination/harassment ordinance that will protect the COVID-19 patients and the front-liners rendering service during the pandemic.

(CARMONA... from page 1)

ture checks before they undergo with the procedure. Their vital signs are also checked, and a skin testing of lidocaine is also done to the patient. If the child gets a negative skin test, then he gets circumcised and given free medications right after the operation.

Researchers identify cells likely targeted by COVID-19 virus

Researchers at MIT, the Ragon Institute of MGH, MIT, and Harvard, and the Broad Institute of MIT and Harvard, along with colleagues from around the world have identified specific types of cells that appear to be targets of the coronavirus that is causing the Covid-19 pandemic.

Using existing data on the RNA found in different types of cells, the researchers were able to search for cells that express the two proteins that help the SARS-CoV-2 virus enter human cells. They found subsets of cells in the lung, the nasal passages, and the intestine that express RNA for both of these proteins much more than other cells.

The researchers hope that their findings will help guide scientists who are working on developing new drug treatments or testing existing drugs that could be repurposed for treating Covid-19.

"Our goal is to get information out to the community and to share data as soon as is humanly possible, so that we can help accelerate ongoing efforts in the scientific and medical communities," says Alex K. Shalek, the Pfizer-Laubach Career Development Associate Professor of Chemistry, a core member of MIT's Institute for Medical Engineering and Science (IMES), an extramural member of the Koch Institute for Integrative Cancer Research, an associate member of the Ragon Institute, and an institute member of the Broad Institute.

Shalek and Jose Ordovas-Montanes, a former MIT postdoc who now runs his own lab at Boston Children's Hospital, are the senior authors of the study, which appears April 22, 2020 in Cell. The paper's lead authors are MIT graduate students Carly Ziegler, Samuel Allon, and Sarah Nyquist; and Ian Mbano, a researcher at the Africa Health Research Institute in Durban, South Africa.

Not long after the SARS-CoV-2 outbreak began, scientists discovered that the viral "spike" protein binds to a receptor on human cells known as angiotensin-converting enzyme 2 (ACE2). Another human protein, an enzyme called TMPRSS2, helps to activate the coronavirus spike protein, to allow for cell entry. The combined binding and activation allows the virus to get into host cells.

"As soon as we realized that the role of these proteins had been biochemically confirmed, we started looking to see where those genes were in our existing datasets," Ordovas-Montanes says. "We were really in a good position to start to investigate which are the cells that this virus might actually target."

Shalek's lab, and many other labs around the world, have performed large-scale studies of tens of thousands of human, non-human primate, and mouse cells, in which they use single-cell RNA sequencing technology to determine which genes are turned on in a given cell type. Since last year, Nyquist has been building a database with partners at the Broad Institute to store a huge collection of these datasets in one place, allowing researchers to study potential roles for particular cells in a variety of infectious diseases.

Much of the data came from labs that belong to the Human Cell Atlas project, whose goal is to catalog the distinctive patterns of gene activity for every cell type in the human body. The datasets that the MIT team used for this study included hundreds of cell types from the lungs, nasal passages, and intestine. The researchers chose those organs for the Covid-19 study because previous evidence had indicated that the virus can infect each of them. They then compared their results to cell types from unaffected organs.

"Because we have this incredible repository of information, we were able to begin to look at what would be likely target cells for infection," Shalek says. "Even though these datasets weren't designed specifically to study Covid, it's hopefully given us a jump start on identifying some of the things that might be relevant there."

In the nasal passages, the researchers found that goblet secretory cells, which produce mucus, express RNAs for both of the proteins that SARS-CoV-2 uses to infect cells. In the lungs, they found the RNAs for those proteins mainly in cells called type II pneumocytes. These cells line the alveoli (air sacs) of the lungs and are responsible for keeping them open.

In the intestine, they found that cells called absorptive enterocytes, which are responsible for the absorption of some nutrients, express the RNAs for these two proteins more than any other intestinal cell type.

Link between obesity and sleep loss

Can staying up late make you fat? A growing body of research has suggested that poor sleep quality is linked to an increased risk of obesity by deregulating appetite, which in turn leads to more calorie consumption.

But a new study published last week in PLOS Biology found that the direction of this reaction might actually be flipped: It's not the sleep loss that leads to obesity, but rather that excess weight can cause poor sleep, according to researchers from the University of Pennsylvania's Perelman School of Medicine and the University of Nevada, Reno, who discovered their findings in the microscopic worm Caenorhabditis elegans (C. elegans).

"We think that sleep is a function of the body trying to conserve energy in a setting where energetic levels are going down. Our findings suggest that if you were to fast for a day, we would predict you might get sleepy because your energetic stores would be depleted," said study co-author David Raizen, MD, PhD, an associate professor of Neurology and member of the Chronobiology and Sleep Institute at Penn.

Raizen emphasized that while these findings in worms may not translate directly to humans, C. elegans offer a surprisingly good model for studying mammalian slumber. Like all other animals that have nervous systems, they need sleep. But unlike humans, who have complex neural circuitry and are difficult to study, a C. elegans has only 302 neurons — one of which scientists know for certain is a sleep regulator.

In humans, acute sleep disruption can result in increased appetite and insulin resistance, and people who chronically get fewer than six hours of sleep per night are more likely to be obese and diabetic. Moreover, starvation in humans, rats, fruit flies, and worms has been shown to affect sleep, indicating that it is regulated, at least in part, by nutrient availability. However, the ways in which sleeping and eating work in tandem has remained unclear.

"We wanted to know, what is sleep actually doing? Short sleep and other chronic conditions, like diabetes, are linked, but it's just an association. It's not clear if short sleep is causing the propensity for obesity, or that the obesity, perhaps, causes the propensity for short sleep," said study co-author Alexander van der Linden, PhD, an associate professor of Biology at the University of Nevada, Reno.

To study the association between metabolism and sleep, the researchers genetically modified C. elegans to "turn off" a neuron that controls sleep. These worms could still eat, breathe, and reproduce, but they lost their ability to sleep. With this neuron turned off, the researchers saw a severe drop in adenosine triphosphate (ATP) levels, which is the body's energy currency.

"That suggests that sleep is an attempt to conserve energy; it's not actually causing the loss of energy," Raizen explained.

In previous research, the van der Linden lab studied a gene in C. elegans called KIN-29. This gene is homologous to the Salt-Inducible Kinase (SIK-3) gene in humans, which was already known to signal sleep pressure. Surprisingly, when the researchers knocked out the KIN-29 gene to create sleepless worms, the mutant C. elegans accumulated excess fat — resembling the human obesity condition — even though their ATP levels lowered.

The researchers hypothesized that the release of fat stores is a mechanism for which sleep is promoted, and that the reason KIN-29 mutants did not sleep is because they were unable to liberate their fat. To test this hypothesis, the researchers again manipulated the KIN-29 mutant worms, this time expressing an enzyme that "freed" their fat. With that manipulation, the worms were again able to sleep.

Raizen said this could explain one reason why people with obesity may experience sleep problems. "There could be a signaling problem between the fat stores and the brain cells that control sleep," he said.

While there is still much to unravel about sleep, Raizen said that this paper takes the research community one step closer to understanding one of its core functions — and how to treat common sleep disorders.

"There is a common, overarching sentiment in the sleep field that sleep is all about the brain, or the nerve cells, and our work suggests that this isn't necessarily true," he said. "There is some complex interaction between the brain and the rest of the body that connects to sleep regulation."

Which foods do you eat together? How you combine them may raise dementia risk

It's no secret that a healthy diet may benefit the brain. However, it may not only be what foods you eat, but what foods you eat together that may be associated with your risk of dementia, according to a new study published in the April 22, 2020, online issue of Neurology®, the medical journal of the American Academy of Neurology. The study looked at "food networks" and found that people whose diets contained mostly of highly processed snacks, starchy foods like potatoes, and snacks like cookies and cakes, were more likely to have dementia years later than people who ate a wide variety of healthy foods.

"There is a connection between the combination of foods in a person's diet, and it is important to understand how these different connections, or food networks, may affect the brain because diet could be a promising way to prevent dementia," said study author Cecilia Samieri, PhD, of the University of Bordeaux in France. "A number of studies have shown that eating a healthy diet, for example a diet rich in green leafy vegetables, berries, nuts, whole grains and fish, may lower a person's risk of dementia. Many of those studies are focused on quantity and frequency of foods. Our study went one step further to look at food networks and found important differences in the ways in which food items were co-consumed in people who went on to develop dementia and those who did not."

The study involved 209 people with an average age of 78 who had dementia and 418 people, matched for age, sex and educational level, who did not have dementia.

Participants had completed a food questionnaire five years previously describing what types of food they ate over the year, and how frequently, from less than once a month to more than four times a day. They also had medical checkups every two to three years. Researchers used the data from the food questionnaire to compare what foods were often eaten together by the persons with and without dementia.

Researchers found while there were few differences in the amount of individual foods that people ate, overall food groups or networks differed substantially between people who had dementia and those who did not have dementia.

"Processed meats were a "hub" in the food networks of people with dementia," said Samieri. "People who developed dementia were more likely to combine highly processed meats such as sausages, cured meats and pates with starchy foods like potatoes, alcohol, and snacks like cookies and cakes. This may suggest that frequency with which processed meat is combined with other unhealthy foods, rather than average quantity, may be important for dementia risk. For example, people with dementia were more likely, when they ate processed meat, to accompany it with potatoes and people without dementia were more likely to accompany meat with more diverse foods, including fruit and vegetables and seafood."

Overall, people who did not have dementia were more likely to have a lot of diversity in their diet, demonstrated by many small food networks that usually included healthier foods, such as fruit and vegetables, seafood, poultry or meats.

"We found that more diversity in diet, and greater inclusion of a variety of healthy foods, is related to less dementia," said Samieri. "In fact, we found differences in food networks that could be seen years before people with dementia were diagnosed. Our findings suggest that studying diet by looking at food networks may help untangle the complexity of diet and biology in health and disease."

One limitation of the study was that participants completed a food questionnaire that relied on their ability to accurately recall diet rather than having researchers monitor their diets.

Another limitation was that diets were only recorded once, years before the onset of dementia, so any changes in diet over time were unknown.

Small rises in blood glucose trigger big changes in insulin-producing cells

In diabetes, tiny clusters of insulin-producing "beta cells" in the pancreas don't produce enough of the hormone to keep people healthy, and their blood glucose levels climb. Perhaps unsurprisingly, their beta cells then function very differently than the cells do in people with normal blood glucose levels.

What's surprising is that the changes in beta-cell behavior begin to occur when the blood glucose levels are barely elevated, still within the pre-diabetes range. "These slightly high concentrations of glucose are enough to really confuse the cell," says Gordon Weir, MD, senior investigator and senior staff physician at Joslin Diabetes Center.

In a paper recently published in Molecular Metabolism, Weir's lab laid out a wealth of new data about how beta cells behave at slightly raised levels of blood glucose. The work provides major additional evidence of a "glucose toxicity" effect that helps to drive the development of both type 1 and type 2 diabetes.

Studying beta cells in lab rats whose blood glucose levels were slightly elevated, Weir's lab found changes in gene expression that affect not just how well the cells function but their ability to divide and grow, as well as their vulnerability to autoimmunity and inflammation.

Weir, professor of medicine at Harvard Medical School, has long studied a puzzling type 2 diabetes phenomenon called first-phase insulin release and how this release is shut down as the disease progresses.

In healthy people with normal blood glucose levels, Weir explains, the body responds quickly to glucose with a big spike of insulin secretion. "If then you take people who have slightly higher glucose levels, above 100 mg/dl, which is still not even diabetes, this first-phase insulin release is impaired," he says. "And when the level gets above 115 mg/dl, it's gone. So virtually all the beta cells don't respond to that acute stimulus." Fortunately, the cells eventually do wake up and respond to other stimuli well enough to keep blood glucose in a prediabetic range.

In earlier research, Weir and collaborators studied this phenomenon in rats who were surgically altered to generate slightly high blood glucose levels, and found that the rats' beta cells secreted less insulin. In their latest experiments, the Joslin team employed the same approach along with powerful "RNA sequencing" methods that revealed patterns of gene expression in the beta cells, either four weeks or ten weeks after surgery. "We found incredible changes in gene expression, and the higher the glucose, the worse the changes," Weir says.

As expected, genes involved in insulin secretion were highly active in the beta cells. More striking were newly discovered alterations in gene expression that could make the cells more vulnerable. Some of these changes were related to cell growth – healthy beta cells may respond to increased blood glucose levels by copying themselves, but these cells were getting stuck as they tried to divide. Additionally, the cells showed many differences in the expression of genes involved in cell inflammation and autoimmunity.

In type 1 diabetes, immune cells called "T cells" begin to kill off the beta cells and blood glucose levels start to creep up. Weir's team found that in the rats with just slightly greater glucose levels, beta cells showed dramatic increases in the expression of some key genes involved in T cell interactions. That effect could make the beta cells a better target for autoimmune attack, and thus speed the disease.

This finding may improve the understanding of the rapid death of beta cells that patients typically experience just before they are diagnosed with type 1 diabetes, Weir says. It also might shed light on the "honeymoon" period some people experience after diagnosis, in which their blood glucose levels are relatively easy to control. During this period, if insulin treatments can bring the remaining beta cells back down to only slightly elevated glucose levels, the cells can function much better, he says.

Researchers uncover mechanisms of protective antibody response during Marburg infection

A detailed study of the monoclonal antibodies from a person who survived a Marburg infection led researchers to identify novel mechanisms that constitute protection against the disease, according to the latest findings of a collaborative team led by The University of Texas Medical Branch at Galveston and Vanderbilt University Medical Center. The findings are now available in Cell Host & Microbe.

Certainly, the virus that is on everyone's mind is the novel coronavirus SARS-CoV-2 that causes COVID-19 disease, but there are other viral families that continue to cause deadly human disease. One of these viral families are the filoviruses, which includes Ebola and Marburg viruses. There are currently no vaccines or drugs approved for human use to protect against the Marburg virus, which causes severe and often lethal disease in people. The largest known outbreak of Marburg virus occurred in 2004-2005 in Angola, which had a 90 percent case fatality rate, according to the Centers for Disease Control and Prevention.

"Antibodies are currently the most promising platform for developing post-exposure treatments for Marburg virus infection and are critical tools for design of improved vaccines," said senior author Alex Bukreyev, a UTMB virologist in the departments of Pathology and Microbiology & Immunology who led the UTMB team that performed the study. "Understanding the mechanisms of antibody-mediated protection during Marburg virus infection is also useful for understanding antibody protection against other viral pathogens, including SARS-CoV-2, which causes COVID-19."

In an earlier study, the researchers isolated a large panel of monoclonal antibodies from immune B cells of a person who survived a natural Marburg infection. In the current study, they analysed the monoclonal antibodies and learned that two of them revealed unique biological properties that protect against natural Marburg infection. These monoclonal antibodies bind to the envelope protein of Marburg virus called glycoprotein but do not kill the virus. Instead, they recruit immune cells that contribute by killing infected cells. In addition, one of these antibodies rearranges the glycoprotein in a way that facilitates access for other antibodies that are able to kill the virus, thus strengthening the protective effect.

Diagnostic biosensor quickly detects SARS-CoV-2 from nasopharyngeal swabs

According to many experts, early diagnosis and management are critical for slowing the spread of SARS-CoV-2, the new coronavirus that causes COVID-19. Therefore, the race is on to develop diagnostic tests for the virus that are faster, easier and more accurate than existing ones. Now, researchers reporting in ACS Nano have developed a field-effect transistor-based biosensor that detects SARS-CoV-2 in nasopharyngeal swabs from patients with COVID-19, in less than one minute.

Currently, most diagnostic tests for COVID-19 rely on a technique called real-time reverse transcription-polymerase chain reaction (RT-PCR), which amplifies SARS-CoV-2 RNA from patient swabs so that tiny amounts of the virus can be detected. However, the method takes at least 3 hours, including a step to prepare the viral RNA for analysis. Edmond Changyun Park, Seung Il Kim and colleagues wanted to develop a faster diagnostic test that could analyze patient samples directly from a tube of buffer containing the swabs, without any sample-preparation steps.

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Helping the heart heal itself

UT Southwestern Medical Center scientists have discovered a protein that works with others during development to put the brakes on cell division in the heart, they reported April 22, 2020 in Nature. The findings could eventually be used to reverse this developmental block and help heart cells regenerate, offering a whole new way to treat a variety of conditions in which heart muscle becomes damaged, including heart failure caused by viruses, toxins, high blood pressure, or heart attacks.

Current pharmaceutical treatments for heart failure – including ACE inhibitors and beta blockers – center on trying to stop a vicious cycle of heart muscle loss as strain further damages remaining heart muscle, causing more cells to die, explains UT Southwestern physician-researcher Hesham A. Sadek, M.D., Ph.D., a professor of internal medicine molecular biology, and biophysics. There are no existing treatments to rebuild heart muscle.

Nine years ago, Sadek and his colleagues discovered that mouse hearts can regenerate if they're damaged in the first few days of life, spurred by the division of cardiomyocytes, the cells responsible for a heart's contractile force. However, this capacity is completely lost by 7 days old, an abrupt turning point in which division of these cells dramatically slows and the cells themselves enlarge. The reasons why these cells gradually slow and stop dividing has been unclear.

Sadek and his team discovered in 2013 that a protein called Meis1, which falls into a category known as transcription factors that regulate the activity of genes, plays a key role in stopping heart cell division. However, he explains, although deleting this gene in mice extends the window of heart cell division, this effect is transient – heart cells missing this gene eventually slow and stop their multiplication.

Scientists shed new light on viral protein shell assembly

New insight on the conditions that control self-assembly in the protective shell of viruses has been published April 21, 2020 in eLife.

The study also highlights the factors that can cause more not self-assembly in the viral protein shell, otherwise known as the capsid, preventing viruses from being able to replicate. The findings suggest that manipulating these factors to induce misassembly in viral capsids could be a promising new approach to hindering viral infections.

Viruses are formed by a chain of the nucleic acids DNA or RNA that are encased in a protein shell made, in the simplest cases, from multiple copies of a single protein. This capsid protects, carries and delivers viruses to their host. Despite this apparent simplicity in their make-up, viruses are able to perform many complex functions that are essential to their replication cycle – one of these being the ability of the viral capsid to assemble itself. The resulting structure of a correctly self-assembled capsid has a very precise architecture, which in most cases is spherical and similar to an icosahedron, with 20 identical triangular faces.