

**The Lord is
like a strong
tower, where
the righteous
can go and
be safe.**

Proverbs 18:10

ISSN: 3116-4757

DIYARYO KABITENYO

News Publishing Service

Entered as FIRST CLASS MAIL at Imus Post Office with Business Mail Permit No. IC-19-06-249

Vol. 29 No. 28

August 24-30, 2026

P 10.00

**Be alert,
stand firm
in the faith,
be brave, be
strong.**

1 Corinthians 16:13

Mt. Pico de Loro temporarily closed to hikers



The Department of Environment and Natural Resources (DENR) hit the province. The closure had been implemented on Aug. 21 and was scheduled to last until Sept. 12. According to DENR Region 4A, all hiking activities at Mt. Pico de Loro and throughout the

Mounts Palay-Palay/Mataas-na-Gulod Protected Landscape (MPPMNGPL) had been suspended until Sept. 12.

The DENR also sought to repair damaged sections, make the trails safe for visitors again and

put in place additional safety measures for hikers.

Mt. Pico de Loro is one of the most popular hiking destinations in Cavite and Batangas, attracting many hiking enthusiasts.

It is part of the

Turn to page 2

DepEd officer in Bacoor found dead

A 28-year-old person with disability (PWD) working for the Department of Education (DepEd) at Bacoor National High School had been found dead in a grassy area along Molino Boulevard in Barangay San Nicolas 2, Bacoor City, Cavite, after going missing for a week. Earlier, Morales' sibling had reported his disappearance to the police.

Morales was reportedly last seen alive at Main Square Mall along Molino Boulevard in Barangay Bayanan, Bacoor City, on Aug. 13, 2026, at around 4:30 p.m. During the investigation

and review of CCTV footage, police discovered the body of a man along Molino Boulevard within Barangay San Nicolas 2 on Aug. 20. Although the remains were difficult to identify, the physical

build and clothing matched those of the missing Morales.

The victim's remains were immediately subjected to forensic examination and identification by SOCO, which positively confirmed that the body was that of Morales. The cause of death had not yet been determined. The victim had been dead for several days and had been exposed to rainwater.

DIYARYO KABITENYO

News Publishing Service

ISSN: 3116-4757

ARNULFO BARCO

(May 14, 1951 - September 2, 2024)
Founder

GENER BARCO

Publisher-Editor

Published by **DIYARYO KABITENYO News Publishing Service**, it has its editorial and business offices at Block 13 Lot 1 Liwayway Homes Subdivision, Anabu 1-C, City of Imus, Cavite. It is published and circulated weekly, every Monday, throughout the province of Cavite. It is registered with the Department of Trade and Industry with Business Name No. 6486090. Our Telephone No. is (046) 5463975, Mobile No.: 09179496918 and email address: barcogener@gmail.com.

Subscription Rate:
1 month - P 40.00
3 months - 120.00

Advertising Rate:
Commercial - P200.00/col. cm.
Legal- 160.00/col. cm.

(MT. PICO...from page 1)

MPPMNGPL, which trails, campsites and covers Ternate and summit, an unregulated Maragondon in Cavite, number of visitors, and as well as Nasugbu in widespread soil erosion Batangas. and environmental damage.

The protected landscape has three After nearly six main peaks, Mt. years of rehabilitation, Palay-Palay, Mt. it was reopened to the public in January 2023, with restrictions imposed on incoming climbers and rock formation that resembles a parrot's beak. In October 2016, an elevation of 688 meters (2,257 feet) closed to the public above sea level (highest point in Cavite) with environmental moderate (beginner-friendly with some steep parts and rope sections and about 4 growing number of vendors along the hiking).

Too much or too little sleep may accelerate aging in multiple organs

A new analysis of proteins and medical biological aging across the human body reveals that both sleeping too little and sleeping too much are associated with faster aging in the brain, heart, lungs, immune system, and other organs, linking these sleep patterns to a broad range of diseases.

Wen explains the motivation behind this approach: "Everyone is excited by these aging clocks and their ability to predict disease and mortality risk," but adds, "But to me, the more exciting question is, can we link aging clocks to a lifestyle factor that can be modified in time to slow aging?"

Driven by mounting evidence of sleep's role in health and a personal interest as a light sleeper, Wen states, "I'm also a light sleeper and was getting worried about the effects on myself," the team constructed clocks using structural measurements, organ-specific proteins, and blood molecules, noting, "In the liver, for example, we have an aging clock built with protein data, an aging clock of metabolic data, and an aging clock of imaging data," which "allows us to see whether sleep is distinctively associated with aging clocks derived from multiple omics and molecular layers."

By comparing self-reported sleep duration with biological age estimates from 23 clocks covering 17 organ systems, the researchers identified a clear U-shaped pattern where individuals sleeping fewer than 6 hours or more than 8 hours exhibited faster biological aging, while the lowest aging levels occurred in those sleeping between 6.4 and 7.8 hours.

Although the findings do not prove causation, suggesting instead that extreme sleep durations may signal poorer overall health, the study highlights a profound connection between sleep, the brain, and the rest of the body, with short sleep linked to depressive episodes, anxiety, obesity, type 2 diabetes, hypertension, and heart issues, while both short and long sleep were associated with respiratory and digestive disorders.

Emphasizing the significance of these results, Wen says, "The broad brain-body pattern is important because it tells us that sleep duration is a deeply embedded part of our entire physiology, with far-reaching implications across the body."

Further investigation into late-life depression using mediation analysis revealed that while the causal direction remains unclear, short sleep may be more directly connected to depression burden, whereas long sleep might influence depression through pathways reflected in brain and adipose tissue aging clocks, leading Wen to conclude, "This has a strong implication for future sleep management and future therapeutics," and "Our study suggests there may be different biological pathways between long and short sleepers that lead to the same outcome, late-life depression, and we shouldn't treat them the same way."



Republic of the Philippines
Province of Cavite
MUNICIPALITY OF MENDEZ-NUÑEZ
OFFICE OF THE MUNICIPAL CIVIL REGISTRAR

CCE-0037-2026 RA 10172

In compliance with the publication requirement and pursuant to OCRG Memorandum Circular No. 2013-1 Guideline in the Implementation of the Administrative Order No. 1 Series of 2012 (IRR on RA 10172), Notice is hereby served to the public that **DORINA PEREÑA ANTONIO** has filed with this Office, a petition for **Correction of Clerical Error in Date of Birth** from **APRIL 30, 1946** to **FEBRUARY 14, 1946** in the Certificate of Live Birth of **ANTONIO PEREÑA** at Mendez, Cavite and whose parents are **MELICIO PEREÑA** and **EUSEBIA AURE**.

Any person adversely affected by said petition may file his written opposition with this office not later than **August 31, 2026**.

(Sgd.) **NOREN A. PEROLA**
Municipal Civil Registrar-OIC

Publication : DIYARYO KABITENYO News Publishing Service
Dates : August 17 & 24, 2026



Republic of the Philippines
Province of Cavite
MUNICIPALITY OF TANZA
LOCAL CIVIL REGISTRY OFFICE

Publication Notice
R.A.10172

NOTICE TO THE PUBLIC

Date: August 13, 2026

CCE - 0162-2026 RA 10172

In compliance with the publication requirement and pursuant to OCRG Memorandum Circular No. 2013-1 Guidelines in the Implementation of the Administrative Order No.1, Series of 2012 (IRR on R.A. 10172), Notice is hereby served to the public that **JENNYLYN MUSA ARELLANO**, has filed with this Office, a petition for **CORRECTION OF ENTRY IN CHILD'S SEX** from **"MALE"** to **"FEMALE"**, in the Certificate of Live Birth of **JENNYLYN MUSA ARELLANO**, who was born on 12 August 1996 at Tanza, Cavite of parents **JOCELYN A. MUSA & NARCISO O. ARELLANO, JR.**

Any person adversely affected by said petition may file his/her written opposition with this Office not later than **August 27, 2026**.

(Sgd.) **OFELIA U. ARGUSON**
OIC-Municipal Civil Registrar

Publication : DIYARYO KABITENYO News Publishing Service
Dates : August 17 & 24, 2026

Publication Notice
R.A. 10172



Republic of the Philippines
Local Civil Registry Office
Province of Cavite
Municipality of Naic



OFFICE OF THE MUNICIPAL CIVIL REGISTRAR

NOTICE TO THE PUBLIC

CFN-0006-2026

In compliance with the publication requirement and pursuant to OCRG Memorandum Circular No. 2013-1 Guidelines in the Implementation of the Administrative Order No. 1, Series of 2012, (IRR on R.A 10172) Notice is hereby served to the public that **ALEXANDER MARTINEZ PEÑA** has filed with this Office a petition for correction for **CHANGE OF FIRST NAME** from **"ALFREDO"** to **"ALEXANDER"** in his Certificate of Live Birth and whose parents are **Benedicto Peña** and **Julita Martinez**.

Any person adversely affected by said petition may file his written opposition with this Office not later than **September 7, 2026**.

(Sgd.) **MARGARITA A. CATIBAYAN**
Municipal Civil Registrar

Publication : DIYARYO KABITENYO News Publishing Service
Dates : August 24 & 31, 2026

Dietary yeast beta-glucan reverses immune defects caused by obesity

Researchers at Trinity College Dublin and University College Dublin (UCD) have discovered that a yeast-based dietary supplement can strengthen immune cells responsible for fighting cancer in mice, potentially counteracting the immune dysfunction caused by obesity.

Published in Cell Reports, the study was led by Frederick Sheedy, Associate Professor in Immunology in Trinity's School of Biochemistry and Immunology, and Helen Roche, Professor in Nutrigenomics at UCD. Dr. Anna Ledwith, the study's first author and a postdoctoral researcher in Prof. Roche's group, explained the study's core objectives: "We wanted to investigate whether a common dietary supplement, yeast beta-glucan, could reprogram early-stage immune cells in the bone marrow to produce long-lasting, enhanced anti-tumor immune responses. Mice were fed a standard or high-fat diet supplemented with yeast beta-glucan for 4-12 weeks, and then their immune system was challenged by different types of cancer cells:

colorectal, skin, and breast cancer.

The study also tested whether yeast supplementation could overcome immune dysfunction caused by obesity and whether protective effects persist after weight loss."

The team aimed to determine if consuming yeast beta-glucan could induce lasting "trained immunity" by altering bone marrow stem cells, a mechanism previously only achievable through injections. Highlighting the breakthrough, Prof. Roche stated: "This is the first demonstration that dietary delivery of yeast beta-glucan is sufficient to induce trained

immunity through reprogramming of bone marrow stem cells.

Previous research required injections. Crucially, this dietary intervention restores anti-tumor innate immunity in obese mice and reverses long-term immune memory defects that persist even after weight loss, a major unmet clinical challenge."

This finding is particularly significant because obesity disrupts immune function, increasing susceptibility to infections and cancer, and these negative effects often persist even after weight loss.

While yeast beta-glucan is already a

commercially available supplement with a strong safety record, its potential to reverse these deep-seated immune memory defects in obese mice offers a promising avenue for future human trials. Looking ahead, Professor Sheedy noted: "This research paves the way for dietary intervention studies in people living with obesity, chronic infections and other immunocompromised populations.

The yeast beta-glucan used, WellmuneTM, from Kerry Group, is already food-grade and commercially available, facilitating

rapid clinical trials. Ultimately, a simple dietary supplement could help boost the immune system's cancer-fighting ability, complementing existing treatments such as chemotherapy and immunotherapy, with the potential to improve vaccine responses and resistance to infection."

As cancer remains a leading cause of death globally, the ability to safely restore immune function through an accessible dietary supplement could offer a vital complement to current therapies, though future studies will be required to confirm these benefits in humans.

Gut bacteria leave lasting molecular mark to fight inflammation

New Northwestern Medicine research published in Nature Communications reveals that a substance produced when gut bacteria digest dietary fiber may leave a durable molecular mark on the cells lining the intestine, promoting immune tolerance and potentially protecting against inflammatory bowel disease-like conditions even after the metabolite is no longer present.

While short-chain fatty acids like butyrate are known for their anti-inflammatory effects, Yu noted a key unresolved question: "A large proportion of butyrate in the gut is rapidly absorbed and metabolized by intestinal epithelial cells (IECs), which limits the amount of free butyrate that can directly reach underlying immune cells," prompting the inquiry into whether butyrate acts through IECs to regulate immunity.

Experiments showed that mice treated with butyrate retained elevated levels of the anti-inflammatory cytokine IL-10 in CD4+ T-cells two weeks after treatment ceased and

were more resistant to chemically induced colitis, with protection depending on IL-10 signaling. Importantly, this lasting effect did not rely on changes to the gut microbiome, as butyrate still created a persistent immune-regulating environment in germ-free mice.

Yu explained, "We found that oral butyrate treatment induces a sustained immunoregulatory response in the intestine, characterized by increased IL-10 production in CD4+ T-cells and protection from intestinal inflammation even after butyrate treatment is stopped," adding that this occurred even without continuous microbial

stimulation. Further investigation identified that butyrate-treated IECs secrete factors inducing IL-10 production, with metabolomic analysis pointing to N1-acetylspermidine as a key candidate.

Yu detailed, "Mechanistically, we found that butyrate acts on IECs and induces sustained transcriptional and epigenetic activation of Sat1, an acetylpolyamine biosynthetic enzyme," which promotes N1-acetylspermidine production to drive IL-10 expression in T-cells.

These findings challenge the view of intestinal epithelial cells as merely transient barriers, suggesting instead they retain a lasting

molecular record of beneficial signals. Yu emphasized, "The significance of this work is that it identifies a mechanism by which a microbiota-derived metabolite can create durable epithelial T-cell crosstalk," noting that the intestinal epithelium can "retain a lasting imprint of a microbial metabolite signal" and that these metabolites may "train" IECs to maintain immune tolerance.

While further research is needed to confirm these mechanisms in humans, Yu stated, "One important next step is to determine how this epithelial metabolic pathway operates in human intestinal disease, especially in patients with inflammatory bowel disease,"

and whether the butyrate-Sat1-N1-acetylspermidine pathway correlates with disease activity. The team also plans to explore other metabolites, as N1-acetylspermidine did not fully explain all observed immune-regulating activity. Long-term, Yu highlighted, "Many studies have focused on how inflammation can leave harmful memory in epithelial cells, but our findings suggest that beneficial microbial metabolites may also establish protective epithelial programs," suggesting that understanding how diet and microbes shape intestinal epithelial memory could open new avenues for restoring immune tolerance in inflammatory bowel disease.

Your blood holds the secret to your vaccine response—AI can decode it

Vaccines prevent serious illness for many people, but the immune protection they produce can differ substantially from one person to another, and new research led by Arizona State University offers clues about what may be behind those differences.

While scientists typically evaluate vaccine responses after vaccination by measuring whether the immune system generated antibodies against the intended target, researchers at ASU and collaborating institutions examined blood samples from more than 4,000 people to take the opposite approach: determining if immune patterns already present in the blood could reveal how someone would respond before receiving a vaccine. Using artificial intelligence to search

for patterns in blood samples taken before and after COVID-19 vaccination, the team analyzed antibodies that recognized 185 antigens, including common viruses and bacteria, along with targets connected to autoimmune diseases.

Joshua LaBaer, executive director of the Biodesign Institute at ASU and director of the Virginia G. Piper Center for Personalized Diagnostics, who led the study, explains, "What our study found is that certain biomarkers, when analyzed with AI, can predict who is likely to respond well to a vaccine, even before they receive it.

This suggests that some people may be more immune-ready than others." The study, which appears in the current issue of the journal *Cell Press Blue*, examined 8,687

samples from 4,089 participants, including healthy volunteers and people with conditions such as HIV, multiple myeloma, solid organ malignancy, autoimmune disease, inflammatory bowel disease, and solid organ transplantation.

While several immunosuppressed groups were more likely to show reduced responses to COVID-19 vaccination, simply placing someone into an immunosuppressed or healthy category did not reliably predict the outcome; some participants with suppressed immune systems still developed strong responses, while about 5% to 6% of healthy participants showed weak vaccine responses.

The analysis identified specific antibodies that were already present before vaccination

as key indicators, noting that higher levels of antibodies targeting common microbes, including *Staphylococcus aureus*, RSV, and human respirovirus 3, were associated with stronger responses to COVID-19 vaccines.

The researchers call these "sentinel" antibodies because they may serve as indicators of a person's underlying immune readiness, noting that these antibodies are not necessarily acting directly against the vaccine target but rather provide information about how prepared the antibody-producing portion of the immune system is to mount a response.

The team also investigated whether the complete antibody fingerprint could provide more predictive information

than a small number of individual biomarkers, utilizing a deep learning model to examine patterns across the entire antibody panel, a method the findings suggest demonstrates one potential advantage of using AI in biomedical research where machine learning systems can search millions of biological data points for subtle relationships that may be difficult to detect using conventional approaches.

This suggests that understanding vaccine readiness may require looking at the immune system as an interconnected whole instead of concentrating on one antibody or one disease, and the research also demonstrates the potential of newer technologies capable of measuring many

antibody responses simultaneously rather than testing whether a person has antibodies against a single pathogen.

If the findings are confirmed in future studies and extended to additional vaccines, the approach could have uses well beyond COVID-19, potentially supporting vaccine research, development, and medical care for people who are especially vulnerable to weak immune responses, as doctors could potentially

use this type of information to identify people who might benefit from additional vaccine doses, more careful follow-up, or other protective strategies, ultimately pointing toward a future in which vaccination decisions could be informed by an individual's own level of immune readiness.

The indigo gap: Why missing light in our homes fuels nearsightedness

Children who lighting. spend excessive time indoors looking at screens and insufficient time outdoors face a rising risk of myopia, or nearsightedness—a condition projected to affect nearly 5 billion people by 2050.

In experiments using tree shrews, whose visual systems share important similarities with humans, researchers found that exposure to indigo light completely prevented the development of nearsightedness.

than on it. This can lead to severe complications like retinal detachment, glaucoma, and macular degeneration later in life. First author Rafael Grytz, PhD, a visual sciences expert at UAB, explains the choice of animal model: "Even though tree shrews look like squirrels, they are a near-primate, with an eye very similar to that of humans, and so are a good model to study the cause of myopia in the human population."

Corresponding author Richard Lang, PhD, director of research in the Division of Ophthalmology at Cincinnati Children's, notes, "The model of myopia we use in the tree shrew is fairly extreme... So, if indigo light can suppress myopia in these tests, then it should also be quite effective in humans."

Myopia occurs when the eye elongates, causing light to focus in front of the retina rather

than on it. This could reduce myopia via the light-sensing receptor opsin 5 (OPN5), this wavelength is blocked by the lenses of tree shrews and humans. Lang clarifies, "Human lenses, and now we know tree shrew lenses, don't transmit a lot of light in the ultra-violet spectrum.

light humans evolved intensive care unit under. Lang states, "We evolved outside in the full-spectrum light provided by our sun. When we live inside, we don't get all the wavelengths the eye needs for normal refractive development, and so we get myopia.

in 2021, lighting products specifically designed to prevent myopia are not yet widely available. The Science of Light Center at Cincinnati Children's is now preparing to test indigo-enriched lighting in daycare centers to determine

New research led by scientists at Cincinnati Children's and the University of Alabama at Birmingham (UAB), published online on August 18, 2026, in Cell Reports Medicine, suggests that indoor lighting may play a critical role in mitigating this risk.

That's the basic message of this paper." While encouraging more outdoor time remains a key strategy, researchers acknowledge the difficulty of reversing indoor-centric lifestyles and propose redesigning indoor lighting to better replicate outdoor spectra.

They basically cut off most wavelengths below 400 nanometers. This finding pushed us to explore slightly longer wavelengths that could still stimulate OPN5. Ultimately, we found that indigo light from 419 to 446 nanometers was the most effective at preventing myopia."

By using tiny spectacles to induce myopia in one eye while using the other as a comparison, the team exposed the animals to various light wavelengths.

While previous mouse studies suggested violet light near 380 nanometers

if it can actively reduce myopia rates in children. Lang emphasizes that the goal is not merely increasing brightness but making artificial light biologically complete, concluding, "What's our best option? It is to change the lighting environment inside.

The study examines the effects of indigo light, a short-wavelength visible light abundant in natural sunlight but largely absent from standard white LED

If future clinical studies confirm the findings, indigo-enriched lighting could become a safe, passive, and scalable way to help reduce childhood myopia risk."

Although Cincinnati Children's installed a programmable, full-spectrum lighting system in its neonatal

Engineering tissue-resident natural killer cells to infiltrate solid tumors

Cell therapies harnessing the immune system have transformed treatment for blood and lymphatic cancers, yet solid tumors remain challenging due to their resistance to immune cell entry and their ability to suppress nearby defenses; addressing this, researchers at Stanford Medicine and collaborating institutions developed a strategy that transforms natural killer cells into a specialized tissue-resident form capable of infiltrating and destroying solid tumors. “We show that these tissue-resident natural killer cells infiltrate into the solid tumors much better than conventional natural killer cells.

It was very reproducible, very striking and very

clear,” said John Sunwoo, MD, the Edward C. and Amy H. Sewall Professor in the School of Medicine and senior author of the study published last month in Science Translational Medicine.

Led by co-authors Nina Horowitz, PhD, Imran Mohammad, PhD, and June Ho Shin, PhD, the team found in mouse models that these modified cells slowed various solid tumors, with effects amplified when paired with an antibody treatment that guides immune cells toward cancer.

A key advantage of natural killer cells is their ability to be transferred between individuals without triggering immune reactions, potentially allowing for large-

batch production rather than patient-specific manufacturing; “It

would be almost an off-the-shelf drug,” Sunwoo said. “It could make cell therapy much more accessible to a wider variety of patients.”

While natural killer cells, first identified in the 1970s, are known for rapidly destroying abnormal cells without prior exposure, immunology historically focused on circulating blood cells until recent tools enabled deeper tissue analysis. “For a long time, the study of immunology and disease in humans was concentrated on the blood immune cells,” Sunwoo said. “With the advancement of tools and bioinformatics, we are now starting to look more at what's going on in tissue.

For most immune cells, the tissue is where the action is.” Although tissue-

resident natural killer cells exist in skin, lungs, and other organs, their functions have been unclear due to conflicting studies showing them as either weak suppressors or potent killers. “They may adopt different functions based on certain cues in the microenvironment and in the tissue, and differentiate into a certain kind of sub-population,” Sunwoo said. The team discovered that the signaling protein TGF- β plays a critical role, where the amount and duration of exposure determine cell fate.

“It's a Goldilocks kind of thing where if you give just enough of a TGF- β signal, then the natural killer cells become tissue resident with strong toxic activity against malignant cells.

If you give too

much TGF- β , they're still tissue resident, but they're inhibited and dysfunctional, and they don't kill,” Sunwoo said. “You need it to be presented to the natural killer cells in just the right amount and in just the right manner.” By briefly exposing cells to short-lived tumor cells providing a temporary TGF- β burst and requiring direct physical contact, researchers produced highly effective killers expressing CD39, perforin, and granzyme A, unlike the inhibited variant.

These modified cells successfully infiltrated tumor organoids and, when combined with cetuximab, significantly suppressed tumor growth in mice over a month with no apparent adverse

effects. “Even at day 30, when the other mice were sick, the mice that received the combination seemed very healthy,” Sunwoo said, though he cautioned against extrapolating too much from mice to humans, adding, “This was just proof of concept.”

With a Phase I clinical trial for advanced squamous cell carcinoma potentially beginning by year-end pending FDA approval, and a patented method allowing a single donor's cells to generate about 20 doses in two weeks, the path toward broader accessibility is emerging. “They'll be cryopreserved, so we can make a bunch of doses and give it to different patients,” Sunwoo said. “There would be no delay.”

a subset of these mice developed aversion-resistant alcohol intake, meaning they drank despite the addition of quinine to make it increasingly bitter, and notably, "compared to those who did not experience forced abstinence, these mice drank even larger

Crucially, "we saw activity in the BNST even before we gave abstinent mice access to the bitter alcohol," indicating that "it might be possible to identify people who are at risk of relapsing by screening for BNST activity when someone is given access to alcohol."

will need help," and while FDA-approved treatments exist, "the number of people diagnosed with this condition remains very high," with numbers that "have effectively doubled in the U.S. since 1999," making the development of better identification strategies vital.