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. 19 June 22-28, 2026 P 10.00

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

Imus records dangerous 45.6°C heat index despite rainy conditions

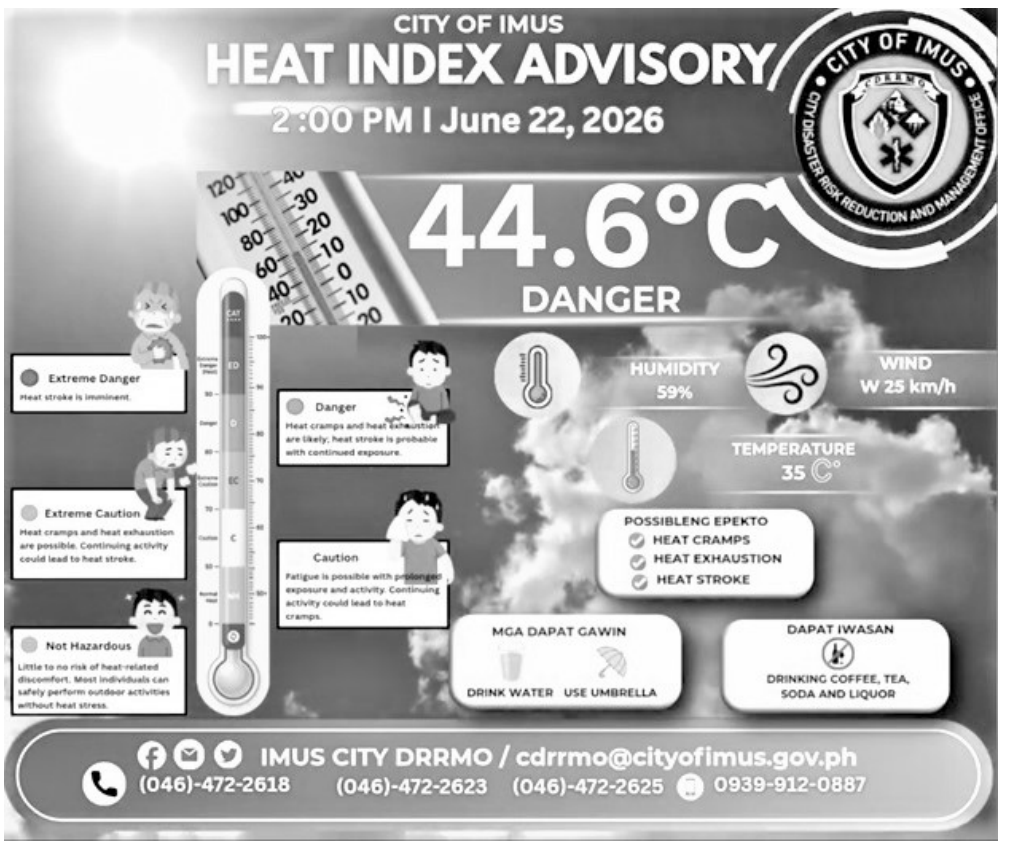
IMUS CITY, Cavite — Residents were advised to remain cautious after Imus City recorded a dangerous heat index of 45.6 degrees Celsius on June 22 despite rainy weather conditions affecting parts of the country.

The Imus City Disaster Risk Reduction and Management Office (DRRMO) issued a public advisory warning residents about the risks of prolonged exposure to extreme heat, which could lead to heat cramps, heat exhaustion, and heat stroke.

Data from the DRRMO showed that the city's heat index reached 40°C at 10:50 a.m. before climbing to 45.6°C at noon, placing it under the "danger" category based on standards set by the Philippine Atmospheric, Geophysical and Astronomical Services Administration (PAGASA).

The heat index reflects the temperature felt by the human body by combining actual air temperature and relative humidity. PAGASA classifies heat index levels between 42°C and 51°C as dangerous due to the increased risk of heat-related illnesses during prolonged exposure.

Although PAGASA suspended the regular release of heat index forecasts on June 5 to focus on weather advisories during the rainy season, the agency continued urging the public to monitor official updates and observe safety measures in areas still experiencing extreme heat.



P680,000 worth of shabu seized in Bacoor, 2 suspects arrested

Agents of the Philippine Drug Enforcement Agency (PDEA) and police officers arrested two suspected large-scale illegal drug traffickers on June 16 and seized P680,000 worth of shabu (crystal meth) in Bacoor City, Cavite.

PDEA Region 4A said its agents, together with personnel from the Cavite Maritime Police Station, arrested the suspects during a buy-bust operation in Barangay Mambog.

The two suspects, both residents of the area, were found carrying 100 grams of suspected shabu valued at around P680,000, based on an estimated price of P6,000 per gram. Authorities also recovered a mobile phone that will be examined for possible records related to illegal drug transactions.

The suspects remain in custody and will face formal charges for violating the Comprehensive Dangerous Drugs Act of 2002.

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.

The hidden link: How Diabetes and Dementia are more connected than you think

For years, doctors have warned that diabetes is tough on the heart and kidneys. But a growing body of research reveals a startling truth: the brain is also at risk. New studies show that the two conditions share a complex, two-way relationship, with blood sugar issues potentially accelerating cognitive decline, and brain changes potentially raising blood sugar levels. The Risk is Real People living with diabetes are about 60% more likely to develop dementia than those without the condition. The risk jumps even higher for those who experience frequent low blood sugar episodes, which are linked to a 50% increase in cognitive decline.

It's Not Just a Body Problem Insulin resistance, the hallmark of type 2 diabetes, doesn't stop at the liver or muscles. It affects the brain, too. In Alzheimer's patients, brain cells often struggle to respond to insulin, making it difficult to use glucose for energy. This "starvation" of brain cells contributes significantly to memory loss.

The "Type 3 Diabetes" Theory The brain uses a massive amount of the body's energy despite being small. In dementia, brain cells lose the ability to process glucose properly. Because of this overlap with insulin issues, some researchers have unofficially dubbed Alzheimer's "Type 3 Diabetes."

The Connection Goes Both Ways The relationship isn't one-sided. People with Alzheimer's often show higher fasting blood glucose levels, even before a diabetes diagnosis. Furthermore, the APOE4 gene, the strongest genetic risk factor for Alzheimer's, appears to reduce insulin sensitivity, effectively trapping insulin receptors and preventing them from working.

Blood Vessels Take the Hit Diabetes damages blood vessels throughout the body, and the brain is no exception. High or fluctuating blood sugar can injure the delicate vessels in the brain, reducing oxygen flow and allowing harmful substances to enter. This leads to inflammation and reduced blood flow, both of which are major drivers of dementia.

A Drug Born from Mistakes Memantine, a common drug used to treat moderate to severe Alzheimer's, was actually originally developed as a diabetes medication. While it failed to control blood sugar, researchers discovered it helped brain function. This serendipitous finding highlights how diabetes research may hold the key to treating brain disorders. The Power of Metformin Metformin, the most widely prescribed diabetes drug, may do more than just lower blood sugar. It can cross into the brain to reduce inflammation. Studies suggest that diabetics taking Metformin have a lower risk of dementia, and that risk rises again if they stop taking it. Clinical trials are now testing whether it can help people without diabetes.

Weight-Loss Drugs Show Promise Newer drugs like semaglutide (known as Ozempic or Wegovy), which help lower blood sugar and aid weight loss, are showing promising results. Records indicate they may be more effective than Metformin at reducing dementia risk. Major trials are currently underway to test oral versions of these drugs in people with early-stage Alzheimer's.

Nasal Insulin: A Potential Future? Because insulin resistance in the brain is a core issue, researchers are testing insulin sprays delivered through the nose. This method aims to send insulin directly to the brain without spiking blood sugar elsewhere. Early small studies suggest it may help memory and reduce brain shrinkage, though long-term safety is still being tested.

A New Contender: SGLT2 Inhibitors Perhaps most exciting is the emerging evidence for SGLT2 inhibitors, a class of diabetes pills that lower blood sugar by removing it through urine. Recent data suggests these drugs might be superior to GLP-1 drugs at lowering the risk of both Alzheimer's and vascular dementia, likely by reducing brain inflammation.

The Bottom Line Managing diabetes is now about more than just protecting your heart and kidneys; it is a vital strategy for preserving your brain. While questions remain about whether these drugs can help people without diabetes, the breakthroughs in diabetes medication are offering new hope for fighting cognitive decline. With over 50 different diabetes medicines now available, the "side effect" of better brain health may be one of the most significant benefits of all.

REPUBLIC OF THE PHILIPPINES
FOURTH JUDICIAL REGION
REGIONAL TRIAL COURT
OFFICE OF THE CLERK OF COURT
CITY OF IMUS, CAVITE

BDO UNIBANK, INC.,
Petitioner-Mortgagee,

EXTRA JUDICIAL FORECLOSURE OF REAL ESTATE MORTGAGE UNDER ACT 3135 AS AMENDED BY ACT 4118

FC Case No. 19793-26

-Versus-

MARITA R. DELA CRUZ
Respondent-Mortgagors.
x----------x

NOTICE OF EXTRA-JUDICIAL SALE

Upon extra-judicial petition for sale under Act No. 3135 as amended by Act 4118 filed by the **BDO UNIBANK, INC** mortgagee, with principal office address at **33rd floor, BDO Corporate Center Ortigas, No 12 ADB Avenue, Mandaluyong City against MARITA R. DELA CRUZ represented by her Attorney-in-fact SHERYL SABLAN VILLANUEVA,** mortgagors, with residential and postal address at **#1081 Roxas Street, Sampaloc, East Manila/or Lot 3 Block 2, Grandiose South, Brgy. Bucandala V, Imus, Cavite** to satisfy the mortgage indebtedness which as of **April 16, 2026** amounts to **ONE MILLION FIVE HUNDRED SEVENTEEN THOUSAND ONE HUNDRED SEVENTY TWO & 94/100 (P1,517,172.94)** Philippine Currency, including interest and penalty charges but excluding attorney's fees, sheriff's fees, and all other charges incidental to this foreclosure and sale, the undersigned Sheriff IV will sell at public auction on **July 21, 2026**, at 10:00 a.m. or soon thereafter at the main entrance of the Office of the Clerk of Court, RTC-Imus, Bulwagan Ng Katarungan, Aguinaldo Highway, Imus City, Cavite, to the highest bidder for CASH and in Philippine Currency, the following properties with all the improvements therein, to wit:

Transfer Certificate of Title
No. 057-2018013624

IT IS HEREBY CERTIFIED *that certain land situated in* BARANGAY OF BUCANDALA - V, MUNICIPALITY OF IMUS, PROVINCE OF CAVITE, ISLAND OF LUZON, *bounded and described as follows:*

A PARCEL OF LAND (LOT 3, BLOCK 2 OF THE SUBDIVISION PLAN PSD-04-227178, BEING A PORTION OF LOT 2400-A, PSD-63558, L.R.C. RECORD NO. 8843), SITUATED IN BRGY. BUCANDALA V, MUN. OF IMUS, PROVINCE OF CAVITE. BOUNDED ON THE SW., ALONG LINE 1-2 BY ROAD LOT 4 (8.00 M. WIDE), OF THE SUBDIVISION PLAN; ON THE NW., ALONG LINE 2-3 BY LOT 2, BLK. 2, BOTH OF THE SUBDIVISION PLAN; ON THE NE., ALONG LINE 4-1 BY LOT 2400-B, PSD-63558. BEGINNING AT A POINT MARKED "1" ON PLAN BEING, S. 52 DEG. 01' E., 200.80 M. FROM MON. NO. 110, IMUS ESTATE. THENCE N. 11 DEG. 50' W., 8.00 M. TO POINT 2; THENCE N. 78 DEG. 10' E., 9.00 M. TO POINT 3; THENCE S. 11 DEG. 50' E., 8.00 M. TO POINT 4; THENCE S. 78 DEG. 10' W., 9.00 M. TO THE POINT OF BEGINNING, CONTAINING AN AREA OF SEVENTY TWO (72) SQUARE METERS. ALL POINTS REFERRED TO ARE INDICATED ON PLAN AND ARE MARKED ON THE GROUND BY OLD PS CYL. CONC. MONS. 15 X 60CM; BEARINGS; TRUE; DATE OF ORIGINAL SURVEY, JULY 27, 1905-JUNE 11, 1908; AND THAT OF THE SUBDIVISION SURVEY, JUNE 12 TO 19, 2012; AND WAS APPROVED ON DECEMBER 12, 2012.

is registered in accordance with the provision of the Property Registration Decree in the name of
Owner: MARITA R. DE LA CRUZ, OF LEGAL AGE, FILIPINO, SINGLE.
Address: #1081 ROXAS ST., SAMPALOC, MANILA.

as owner thereof in fee simple, subject to such of the encumbrances mentioned in Section 44 of said Decree as may be subsisting.

All sealed bid must be submitted to the undersigned on the above stated time and date.

In the event the public auction should not take place on the said date, it shall be held on **July 28, 2026** at 10 a.m. without prior notice.

Prospective bidders/buyers are hereby enjoined to investigate for themselves the titles to the said properties and encumbrances thereon if any there be.

Imus City, Cavite, Philippines, May 14, 2026.

(Sgd.) **AVREIL JOHN L. DAVID**
Sheriff IV

APPROVED:

(Sgd.) **ARMIE A. FRANCISCO**
Clerk of Court VI

COPY FURNISHED:

BDO UNIBANK INC.
C/O JACEL DL. FLORENCIO
33rd floor BDO Corporate Center Ortigas
No. 12 ADB Avenue, Mandaluyong City

MARITA R. DELA CRUZ,
#1081 Roxas Street, Sampaloc, East Manila /or
Lot 3 Block 2. Grandiose South, Brgy. Bucandala V, Imus, Cavite

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 8, 15 & 22, 2026

This DNA repair gene went rogue and exposed a cancer weakness

Tumor suppressor genes are often viewed as the body's built-in defense system against cancer. They produce proteins that help maintain and repair DNA, reducing the chances that harmful mutations will accumulate. When these genes stop working properly or are present at low levels, cancer risk can rise. But new research suggests that having too much of one DNA repair protein can also be a problem.

Researchers at Penn State College of Medicine found that excessive activity of the gene EXO1 can damage DNA rather than protect it. Instead of repairing genetic material, too much EXO1 can break down DNA and destabilize the genome, a key feature of cancer.

The findings, published in Nature Communications, show that EXO1 is overexpressed in 20% to 30% of breast and ovarian cancers, as well as in melanoma, testicular, cervical and hepatobiliary cancers, which occur in the liver, gall bladder and bile duct. The team also discovered that cancer cells with unusually high levels of EXO1 behave much like cells carrying BRCA mutations, which are well known for increasing the risk of hereditary breast and ovarian cancers. Importantly, these BRCA-like behaviors occurred even when no BRCA mutation was present. The researchers found that tumors with elevated EXO1 responded to treatments in ways that closely resembled BRCA-mutant cancers.

"EXO1 doesn't predict cancer risk, but it could potentially serve as a biomarker to help predict which patients are more likely to respond to certain chemotherapy treatments, leading to more personalized therapies," said George-Lucian Moldovan, professor of molecular and precision medicine and senior author on the study. "The same drugs that are reserved for treating BRCA-mutant tumors and that have fewer side effects could potentially be used to treat EXO1 overexpressing tumors, which don't have BRCA mutations. It would expand the applicability of those drugs."

To investigate the role of EXO1, the researchers analyzed tumor data from The Cancer Genome Atlas, a National Cancer Institute cancer genomics program. They found evidence of EXO1 overproduction in multiple cancer types, including tumors of the breast, skin, liver and cervix, consistent with earlier research. Elevated EXO1 levels were especially associated with basal-like breast cancer, an aggressive form of the disease.

The team then performed laboratory experiments using commercially available human cancer cells. Researchers artificially increased EXO1 production in the cells to determine how excess amounts of the protein affected DNA. They also created a disabled version of EXO1 that produced protein but lacked its normal biochemical activity.

AFFIDAVIT OF SELF-ADJUDICATION

NOTICE is hereby given that the estate of the late **TEONILA Y. KOA** who died intestate on November 08, 2024 in Metropolitan Medical Center, Tondo, Manila, consisting of a parcel of land she left at the time of her death, located at Bo. of Buenavista, Gen. Trias City, **Cavite**, covered by Transfer Certificate of Title No. T-208779 containing an area of FIVE HUNDRED NINETY FIVE (595) SQUARE METERS, more or less, has been self-adjudicated by her sole heir YOLANDA YAP KOA on June 4, 2026 at Trece Martirez City, Cavite, Philippines before Notary Public Atty. Cesar N. Santiago and entered in his Notarial Register as Doc. No. 385; Page No. 77; Book No. XXXIX; Series of 2026.

(Sgd.) **Affiant**

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 8, 15 & 22, 2026

Publication Notice
R.A. 10172

Republic of the Philippines
Province of Cavite
Municipality of Maragondon
-oOo-
Office of the Municipal Civil Registrar

NOTICE TO THE PUBLIC

Date: JUNE 9, 2026

RA - 10172
CCE - 0040-2026

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 Name of Document Owner has filed with this Office, a petition for **correction of date of birth** from **"August 5, 1968"** to **"August 4, 1968"** in the certificate of live birth of **FAUSTINO CABILLO ANIT JR.** who was born on **August 5, 1968** at Maragondon, Cavite and whose parents are **FAUSTINO LINEZO ANIT SR.** and **DIONESIA CABELLO**.

Any person adversely affected by said petition may file his written opposition with this Office not later that **June 29, 2026**.

(Sgd.) **HELEN P. UMALI**
Acting Municipal Civil Registrar

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 15 & 22, 2026

Publication Notice
R.A. 10172

Republic of the Philippines
Province of Cavite
Municipality of Maragondon
-oOo-
Office of the Municipal Civil Registrar

NOTICE TO THE PUBLIC

Date: June 9, 2026

RA-10172
CFN-003-2026

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 the Name of Document Owner has filed with this Office, a petition for the **Change of First Name** from **GAVINO** to **SANTIAGO** in the certificate of live birth of **GAVINO MAGTIBAY ANGUE** who was born on October 25, 1963 at **Maragondon, Cavite** and whose parents are **SANTIAGO LOYOLA ANGUE and FELICIDAD PUNONGBAYAN MAGTIBAY**.

Any person adversely affected by said petition may file his written opposition with this Office not later that **June 29, 2026**.

(Sgd.) **HELEN P. UMALI**
Acting Municipal Civil Registrar

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 15 & 22, 2026

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

In the matter of Change of First Name
Certificate of Live Birth of **EDGAR POMPEYO NUESTRO**

CFN #0005-2026 RA 9048

EDGARDO LEPARDO NUESTRO
Petitioner

NOTICE OF PUBLICATION

There is a petition for the Change of First Name of **EDGAR POMPEYO NUESTRO** from **EDGAR POMPEYO** to **EDGARDO** who was born on **JULY 7, 1956** at **MENDEZ, CAVITE** and whose parents are **CANDIDO LEPARDO** and **PEDRO NUESTRO.**

NOTICE IS HEREBY GIVEN that any interested person is cited to notify this office and show cause why the same should not be granted.

Let this Notice be published at least once a week for two (2) consecutive weeks in a newspaper of general circulation as required under Section 5 of Republic Act No. 9048.

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

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 15 & 22, 2026

Scientists discover neurons must break their DNA to build the brain

As the brain develops, newly formed neurons must travel through tightly packed tissue to reach their final destinations in the cerebral cortex, where they become part of the brain's communication network. This journey forces the cells through narrow gaps between fibers and neighboring cells.

A new study published in Nature has revealed an unexpected consequence of that process. Researchers from Kyoto University's Institute for Integrated Cell-Material Sciences (WPI-iCeMS) and collaborating institutions found that migrating neurons routinely experience significant DNA damage. Specifically, the cells develop double-strand breaks, a severe form of DNA damage in which both strands of the DNA double helix are cut.

Although double-strand breaks are typically associated with mutations, cell dysfunction, and even cell death, the researchers discovered that they are a normal part of brain cortex development. In healthy brains, the damage is rapidly repaired before it can cause lasting problems.

"The developing brain appears to have evolved to tolerate and repair the neuronal damage efficiently," says Professor Mineko Kengaku, of WPI-iCeMS, who led the study. "But understanding the limits of that tolerance -- and what happens when repair is incomplete -- brings us closer to understanding a range of neurological conditions."

To investigate how this damage occurs, the researchers recreated the physical challenges faced by developing neurons. They guided neurons through tiny microchannels designed to mimic the confined spaces found in growing brain tissue. Using fluorescent markers, the team observed double-strand DNA breaks appearing as neurons moved through the channels. Once the cells emerged from the other side, the damage gradually disappeared. Most of the breaks were repaired within 24 hours, and the neurons continued functioning normally. The researchers identified the source of the damage as Topoisomerase IIβ, an enzyme that normally helps cells

Turn to page 5

EXTRAJUDICIAL SETTLEMENT OF ESTATE OF THE DECEASED SPOUSES RICARDO HAYAG PAPA, SR. & NELINDA GARCIA PAPA WITH ABSOLUTE SALE

NOTICE is hereby given that the estate of the deceased **Spouses RICARDO HAYAG PAPA, SR. AND NELINDA GARCIA PAPA** who both died intestate at **Imus, Cavite** on September 14, 2007 and on July 7, 2011, respectively, consisting of one (1) parcel of land located at Caloocan City, covered by Transfer Certificate of Title No. T-121890 of the Registry of Deeds for Caloocan City, containing an area of TWO HUNDRED AND FIFTY TWO (252) Square Meters has been adjudicated and extrajudicially settled by and among their heirs in equal share; that, for and in consideration of the amount of ONE MILLION FIVE HUNDRED THOUSAND PESOS (P1,500,000.00), Philippine Currency, they do hereby sell, transfer and convey, the above-mentioned one (1) parcel of land, free from all liens and encumbrances, in favor of JAKE WEASLEY SAGUN and JAMES ANDREW SABADO, both single, of legal age, on May 12, 2026 at Gen. Trias City, Cavite before Notary Public Atty. Chares Marie R. Torres-Elacion and entered in her Notarial Register as Doc. No. 300; Page No. 61; Book No. 1; Series of 2026.

(Sgd.) **All Heirs**

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 15, 22 & 29, 2026

DEED OF EXTRAJUDICIAL SETTLEMENT OF REAL PROPERTY WITH PARTITION

NOTICE is hereby given that the estate of the late **LUIS NARCISO NARTE** and **JUANITA SUAREZ NARTE** who both died intestate on May 11, 2000 at Manila City and on May 31, 2020 at Silang, Cavite, respectively, consisting of three (3) parcels of land situated at Imus, Cavite, General Trias, Cavite, and Guinayangan, Quezon , more particularly described as follows:

REAL PROPERTY 1: IMUS, CAVITE
A parcel of land located in Barangay Bucandala, Imus, Cavite, with an aggregate area of Two Hundred Fifty (250) square meters, more or less, and covered by Transfer Certificate of Title No. T-104857 of the Registry of Deeds for the Province of Cavite;

REAL PROPERTY 2: GENERAL TRIAS, CAVITE
A parcel of land located at Block 13, Lot 13, Barangay Biklatan, General Trias, Cavite, with an aggregate area of Two Hundred (200) square meters, more or less, and covered by Transfer Certificate of Title No. T-361875 of the Registry of Deeds for the Province of Cavite;

REAL PROPERTY 3: GUINAYANGAN, QUEZON
A parcel of land located in the Barangay Dungawan, Guinayangan, Quezon, with an aggregate area of One Hundred Four Thousand Seven Hundred Ninety-Nine (104,799) square meters, more or less, and covered by Transfer Certificate of Title No. T-115144 of the Registry of Deeds for the Province of Quezon,

has been adjudicated and extrajudicially settled by and between their compulsory heirs in the following manner:

1. TO THE HEIR/DAUGHTER: The properties located in Imus, Cavite (Transfer Certificate of Title No. T-104857) and General Trias, Cavite (Transfer Certificate of Title No. T-361875) are hereby adjudicated, transferred, and assigned entirely and exclusively to MARIA LUISA NARTE PAREDES;

2. TO THE HEIR/SON AND HEIR/DAUGHTER (EQUAL SHARE): The property located in Guinayangan, Quezon (Transfer Certificate of Title No. T-115144) shall be divided equally, with fifty percent (50%) undivided share going to JOEL SUAREZ NARTE and fifty percent (50%) undivided share going to MARIA LUISA NARTE PAREDES

on June 10, 2026 at Tanza, Cavite, Philippines before Notary Public Atty. Yvey Rose S. Caringal and entered in her Notarial Register as Doc. No. 425; Page No. 86; Book No. I; Series of 2026.

(Sgd.) **Heir/Son and Jean Luigi Narte Janohan as Attorney-in-fact of Heir/ Daughter)**

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 22, 29 and July 6, 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: June 18, 2026

CCE - 0112-2026 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 **FELY G. DIAZ**, has filed with this Office, a petition for **CORRECTION OF ENTRY IN CHILD'S SEX** from **"FEMALE"** to **"MALE"**, in the Certificate of Live Birth of **JEFF GARCIA DIAZ**, who was born on 5 March 2009 at Tanza, Cavite of parents FELY BALUTE GARCIA & URBANO JR. ABELLERA DIAZ.

Any person adversely affected by said petition may file his/her written opposition with this Office not later than **July 2, 2026**.

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

Publication : DIYARYO KABITENYO News Publishing Service
Dates : June 22 & 29, 2026

(SCIENTISTS...from page 4)

manage stress within DNA. Under ordinary conditions, the enzyme temporarily cuts DNA strands to relieve twisting and tension generated by routine cellular activity before reconnecting them.

The process can be compared to cutting a tangled cable to remove twists and then reconnecting it. However, when neurons are subjected to mechanical stress while squeezing through tight spaces, the enzyme can become trapped midway through the process, leaving sections of DNA broken. The cell then relies on a repair mechanism called non-homologous end joining to reconnect the damaged DNA ends.

The team found that neuronal DNA damage differs from the damage seen in certain cancer cells moving through the same microchannels. In cancer cells, DNA damage tends to occur more randomly and can disrupt normal cellular activity or trigger cell death.

In contrast, the DNA breaks in neurons were concentrated mainly in regions of the genome that are not actively involved in critical gene functions. Because essential genes are largely spared, the cells are able to maintain normal function despite the temporary damage. To explore the consequences of failed repair, the researchers engineered mice whose newly formed cerebellar neurons lacked Ligase 4, an enzyme required for repairing DNA breaks. The mice developed normally and showed no obvious early abnormalities. However, as they reached adulthood, they began to experience mild but gradually worsening balance problems. These symptoms resemble those seen in certain human disorders linked to genome instability that affect the cerebellum.

The findings suggest that DNA breakage and repair may play a larger role in brain biology than previously recognized. Researchers now want to understand whether these early DNA changes contribute to differences between individual neurons and whether they influence neurodevelopmental or neurodegenerative diseases later in life.

"It shifts how we think about the neuronal genome," says Professor Kengaku. "All neurons originate from the same DNA, but DNA damage and repair can introduce small genetic differences between individual neurons through a small mechanical journey. Some of that history may be written into the genome itself."

The study was conducted through a collaboration involving Kyoto University, the University of Tokyo, the University of Osaka, the National University of Singapore, and the Tokyo Metropolitan Institute of Medical Science.

Scientists may have found what really triggers Alzheimer's disease

Researchers at the University of California, Riverside have proposed a new explanation for how Alzheimer's disease may begin. Instead of being driven primarily by plaque buildup in the brain, the disease could start when one protein interferes with the normal function of another inside nerve cells.

For years, Alzheimer's research has largely centered on amyloid beta (a-beta), a protein that forms clumps in the brains of people with the disease. The idea gained support because inherited mutations that increase a-beta levels can cause early onset Alzheimer's.

However, despite thousands of clinical trials designed to remove a-beta, those treatments have largely failed to stop the disease or reverse its progression.

Scientists have also long known that another protein called tau accumulates in the brains of Alzheimer's patients. What has remained uncertain is exactly how tau and a-beta are connected.

"In addition to having dementia, Alzheimer's diagnosis requires both a-beta and tau buildup in the brain," said UCR chemistry professor and study lead author Ryan Julian. "But many labs focus on the role of one and ignore the other."

Published in the Proceedings of the National Academy of Sciences, Nexus, the new study points to a direct interaction between these two proteins.

Tau normally helps stabilize microscopic structures known as microtubules. These tiny tube-like structures act as transportation routes inside nerve cells, carrying essential materials to different parts of the neuron. Without functioning microtubules, neurons struggle to transport the molecules they need to survive and communicate.

The research team noticed that the section of tau responsible for attaching to microtubules closely resembles a-beta in both size and structure. That observation led them to wonder whether a-beta could also bind to microtubules.

To investigate, the scientists attached a fluorescent marker to a-beta. By tracking changes in its movement and light emission, they were able to determine when the protein attached itself to microtubules.

Their experiments revealed that a-beta and tau bind to microtubules with similar strength. As a result, when a-beta accumulates inside neurons, it can potentially push tau out of its normal position.

"Our work shows amyloid beta and tau compete for the same binding sites on microtubules, and that a-beta can prevent tau from functioning correctly," Julian said.

According to the researchers, Alzheimer's may begin when a-beta displaces tau from microtubules. Once that happens, the cell's internal transport network may start to break down.

At the same time, tau may begin behaving abnormally. Without its normal interaction with microtubules, the protein can clump together and move into regions of neurons where it does not normally belong.

This model suggests that the buildup of a-beta and tau may be a consequence of deeper cellular problems rather than the original cause of the disease. The idea could help explain several long-standing puzzles in Alzheimer's research.

For example, plaques made of a-beta often form outside cells. If the key damage occurs when a-beta interferes with tau inside neurons, those external plaques may not directly disrupt tau or the microtubules it supports.

The proposed mechanism also fits with evidence that the brain's natural recycling process becomes less efficient with age.

A process known as autophagy normally removes unwanted proteins, including a-beta, from cells. As autophagy slows in older adults, a-beta may accumulate inside neurons and increasingly compete with tau for access to microtubules.

Additional observations support the theory as well. Some recent studies have reported that lithium may reduce the risk of Alzheimer's disease, while earlier research found that lithium helps stabilize microtubules.

Those findings raise the possibility that protecting microtubules could help counter some of the harmful effects caused by a-beta.

Long-lived families reveal a rare genetic clue to healthy aging

People age differently. Some remain free of major diseases well into old age, while others develop serious health problems much earlier. Understanding why this happens is becoming increasingly important as populations grow older around the world. Although life expectancy has risen dramatically over the past 200 years, the number of years people spend in good health has not increased at the same pace. Researchers have long known that exceptional longevity (longevity) often runs in families and is linked to a later onset of chronic illnesses. However, the genetic factors that help protect these families remain poorly understood.

Most previous studies have focused on the genetics of individual people who live long lives. New research being presented at the annual conference of the European Society of Human Genetics in Gothenburg suggests that studying entire long-lived families may provide a clearer picture of the biological mechanisms that support a longer healthspan. (A person's healthspan is the number of years they live free from chronic disease and cognitive decline).

Studying families offers an important advantage because longevity is influenced by many factors beyond genetics. Socioeconomic status, lifestyle, behavior, and environmental influences all play major roles in determining both lifespan and healthspan. As a result, some people from families with average life expectancy may still live exceptionally long lives, while others from long-lived families may not. Presenting findings from the intergenerational aging study, Mr. Pasquale Putter, a final-year PhD student in Prof. Eline Slagboom's group at Leiden University Medical Center in Leiden, The Netherlands, explained that previous research from the team had already revealed a striking pattern. Middle-aged individuals with long-lived parents developed cardiometabolic diseases an average of 13 years later than their partners whose parents had shorter lifespans. "This made it clear that their longer healthspan was passed down to subsequent generations," he says. To investigate further, researchers analyzed the genomes of 212 groups of long-lived sibships (offspring with the same two parents) participating in the Leiden Longevity Study. The team identified four regions of the genome that appeared likely to contain genes linked to longevity.

"This meant that we could restrict our focus to 350 genes rather than around 20,000," says Mr. Putter. Additional analysis narrowed the search even further, revealing 12 rare protein-altering genetic variants that may contribute to longer and healthier lives. One of those variants was found in the CGAS (cyclic GMP-AMP synthase) gene, which has previously been linked to aging. The variant appeared in two long-lived families included in the study. CGAS helps trigger inflammation when DNA is detected where it does not belong inside a cell. This can happen during viral infections or when cells are damaged. "It is likely that members of these families had only one active copy of the CGAS gene, rather than two, and that this will have reduced the inflammatory response in their bodies, while still being sufficient to clear infections and repair damage, thereby contributing to the protective mechanisms that enable extended healthspan and survival," Mr. Putter says.

The researchers believe this reduced inflammatory response may help protect against some of the damaging effects associated with aging while preserving the body's ability to defend itself. "We hope that taking this family approach will help us to untangle some of the environmental factors from those that are truly genetic, particularly those where rare mutations are involved. We have been surprised by the magnitude of the effect of the CGAS mutation in the in vitro experiments we have carried out to date."The scientists caution that much more work is needed before any implications for human health can be determined. The effects of CGAS depend heavily on context. Completely shutting down the CGAS pathway could make people more vulnerable to infections and cancer. On the other hand, excessive activation of the pathway can lead to chronic inflammation and long-term tissue damage. To better understand how the mutation functions in a living organism, the researchers are moving from in vitro experiments to in vivo studies. They plan to introduce the CGAS mutation into killifish at the Max Planck Institute for the Biology of Ageing in Cologne, Germany. "Killifish are the shortest-lived vertebrates, with a natural lifespan of between three to nine months. Using them as a model will enable us to determine whether the mutation contributes to increased lifespan when compared with control groups, and also to investigate its health effects in tissues," says Mr. Putter.

"We also intend to follow up on our research by investigating other promising candidate longevity variants that we identified in the Leiden Longevity Study through collaborations with other groups."

DIYARYO KABITENYO
News Publishing Service
Vol. 29 No. 19 June 22-28, 2026 P 10.00

Tubulin prevents toxic brain protein clumps linked to Alzheimer's and Parkinson's

Scientists at Baylor College of Medicine have identified a potential new approach for tackling Alzheimer's and Parkinson's diseases. Both conditions are associated with the buildup of harmful clumps formed by the proteins Tau and alpha synuclein in the brain.

In a study published in Nature Communications, the researchers found that tubulin, a protein that serves as the building block of microtubules, may help prevent these toxic accumulations. Microtubules act as the cell's internal 'railway tracks,' helping transport materials and maintain structure. According to the findings, tubulin can keep Tau and alpha synuclein from forming damaging aggregates and instead encourage them to perform their normal functions inside healthy neurons. "Tau and alpha synuclein are well known for their roles in neurodegenerative diseases like Alzheimer's and Parkinson's. In these conditions, these proteins can misfold, stick together and form harmful aggregates that damage neurons and contribute to memory loss, movement problems and other symptoms," said first author Dr. Lathan Lucas, postdoctoral associate of biochemistry and molecular pharmacology in Dr. Allan Ferreon's lab.

"But Tau and alpha synuclein also fulfill essential functions in healthy neurons - they help maintain cell structure and support communication by interacting with tubulin and contributing to microtubule assembly and stabilization."

Tau and alpha synuclein carry out both their beneficial and harmful activities within tiny cellular droplets known as condensates. Because these droplets are involved in disease-related processes, scientists have considered preventing their formation as a possible treatment strategy. However, condensates also play important roles in normal brain function, raising concerns that eliminating them could disrupt healthy neuronal activity.

"This led us to the following idea: what if instead of preventing the formation of droplets, we created conditions that would drive Tau and alpha synuclein inside the droplets toward their healthy path, discouraging them from taking the disease path?" said Ferreon, associate professor of biochemistry and molecular pharmacology and co-corresponding author of the work.

New analysis suggests vaping likely causes cancer

A major new study led by UNSW Sydney concludes that nicotine-based e-cigarettes are very likely to trigger cancers in the lungs and mouth. Published in the journal Carcinogenesis, this comprehensive review synthesizes international research and brings together specialists from institutions like The University of Queensland, Flinders University, and several major hospitals. By pulling together experts in pharmacy, public health, thoracic surgery, and epidemiology, the team aimed to answer a critical question: can vaping itself cause cancer, even without traditional tobacco?

"To our knowledge, this review is the most definitive determination that those who vape are at increased risk of cancer compared to those who don't," says Professor Stewart.

While vaping has often been studied as a stepping stone to smoking, researchers argue that far less attention has been paid to the possibility that e-cigarettes could cause cancer on their own. This work represents one of the most thorough evaluations yet of whether vaping independently increases cancer risk. The analysis didn't just look at one angle; it combined data from clinical research, animal studies, and lab investigations into chemicals produced by e-cigarettes.

"Considering all the findings -- from clinical monitoring, animal studies and mechanistic data -- e-cigarettes are likely to cause lung cancer and oral cancer," Professor Stewart states. Despite the consistency of the results across different research areas, Professor Stewart admits that the exact number of cancer cases linked to vaping is still unknown. The assessment was qualitative, meaning the team didn't calculate a specific numerical risk or burden. They explain that pinpointing the precise risk will require waiting for longer-term studies to come to fruition.

E-cigarettes hit the market in the early 2000s and arrived in Australia around 2008. Originally marketed as a safer alternative to cigarettes and a tool to help people quit, they have evolved significantly since then. Brightly colored and flavored products have grown in popularity, particularly among younger users. Even with stricter regulations introduced by the Australian Government in 2023—banning disposable and non-therapeutic vapes and restricting sales of therapeutic products to pharmacies—vaping remains common outside of schools, bars, and train stations. "E-cigarettes are known to be a gateway to smoking and hence cancer," says co-author UNSW Associate Professor Freddy Sitas.

Professor Sitas notes that while the gateway effect is well-known, the extent to which vaping causes cancer independently has not been a primary focus in research. "The evidence was remarkably consistent across fields," he says, "It dictated an unequivocal finding now, though human studies that estimate the risk will take decades to accumulate."