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American Neurological Association Spotlights Key Abstracts to Be Presented at ANA2022, Oct. 22–25 in Chicago

Cutting-edge research under embargo until October 14.

(Mount Laurel, NJ, October 7, 2022)—The American Neurological Association’s Annual Meeting covers the latest research and innovations across all areas of neurology. In addition to major symposia on key interdisciplinary topics, the [2022 ANA Annual Meeting](#), taking place October 22–25 at the Hyatt Regency Chicago, features hundreds of presentations on breaking research from academic neurologists and neuroscientists nationwide. This year’s groundbreaking research includes demonstrated links between environmental exposures and Alzheimer’s disease, disparities in stroke deaths, why nicotine may reduce Parkinson’s risk, and cardio training as therapy for movement disorders.

The ANA Annual Meeting convenes the top academic neurologists in the United States, international neurology leaders, trainees, and students around key developments in neurology and neuroscience. Plenary sessions will focus on issues including environmental exposures and neurologic disease, equity and disparities in neurology, and pathologic interactions between the nervous system and other organ systems. Members of the media are welcome to attend the full meeting ([preview the full advance program](#) and the [at-a-glance meeting schedule](#)). **You can [register and obtain press credentials here](#).**

The meeting will feature a **“Highlights of the Meeting” media roundtable on Tuesday, October 25, 2022 from 11:00 am to 12:30 p.m. (U.S. Central Time)**, during which the chairs of the principal symposia will present highlights, discuss the relevance of the work, and answer questions.

A complete list of abstracts will be published in [Annals of Neurology](#) Friday, October 14 and will be available to the media. Here are some top examples* (under embargo until October 14).

MOVEMENT DISORDERS: Aerobic exercise may help more than balance training to hold off symptoms of cerebellar ataxia

Scott Barbuto, MD, PhD, Columbia University Medical Center. "Home Aerobic Versus Balance Training In Cerebellar Ataxias."

Currently, no medications exist to combat spinocerebellar ataxias, debilitating neurodegenerative diseases that cause loss of balance and coordination. Physicians recommend balance training to improve symptoms, but a new study suggests that rigorous aerobic exercise may provide greater benefit for adults with cerebellar ataxia. Patients were able to safely undergo six months of aerobic training five times a week. Their ataxia symptoms improved significantly, by an average of 1.9 points on the Scale for Assessment and Rating of Ataxia, compared with control patients who did balance training (who saw improvement of 0.6 SARA points). Although some balance-specific measures showed better results with balance training and studies of less-rigorous aerobic training showed little benefit, intense aerobic activity appears to be a promising therapeutic avenue for ataxia, and demonstrates important connections between nervous system health and aerobic activity.

STROKE: Death rates are decreasing, but race and sex disparities remain

Daniel Oh, MD, University of Southern California. "Trends in Stroke Mortality by Race/Ethnicity and Sex in the U.S. 2000–2019."

Stroke is a leading cause of death in the United States. This study examined 20-year stroke mortality rate disparities and trends among racial and ethnic groups (White, Black, Asian/Pacific Islander, and Hispanic) and between men and women, particularly with regard to stroke as one of multiple causes of death. Black men and Black women had the highest death rates of any of the examined groups (66.4 and 56.5 stroke deaths per 1,000 individuals, respectively). Among Black, Asian/Pacific Islander and Hispanic populations, stroke rates were lower for women than for men. White men and women had similar death rates (41.7 and 41.5 respectively). The lowest stroke mortality rates were among Hispanic and Asian/Pacific Islander women (32.9 and 34.6 per 1,000 individuals) followed by Hispanic and Asian/Pacific Islander men (37.9 and 38.7 deaths per 1,000 individuals). Overall, stroke mortality rates decreased for all groups, with the steepest decreases among women (across racial and ethnic groups) and Black people, and the slowest decline in stroke rates among Hispanic women. Understanding these trends will help better target public health interventions and assess their success.

MULTIPLE SYSTEM ATROPHY: Identifying cells that accelerate disease progression

Jun-ichi Kira, MD, PhD, International University of Health and Welfare, Fukuoka, Japan. "Synucleinopathy-Associated Microglia Uncovered by a Novel Multiple System Atrophy-Cerebellar Type (MSA-c) Mouse Model."

There is currently no cure for the rare neurodegenerative disorder multiple system atrophy (MSA), and its rarity has made it difficult to understand how the disease progresses. Now a research team has created a successful mouse model of aggressive cerebellar-type MSA and identified new populations of glial cells in the brain that exacerbate disease progression. MSA,

similar to Parkinson's disease, affects the motor system as well as the autonomic nervous system including blood pressure control, eventually leading to death. It is characterized by abnormal accumulations of the protein α -synuclein (α -syn) in a type of central nervous system cell called oligodendrocytes. Researchers bred transgenic mice which expressed the ultra-accumulative A53T mutation of human α -syn when triggered by removal of doxycycline from their diet. The mice showed disease progression similar to humans. Disease symptoms could be fully reversed with the reintroduction of doxycycline to the diet after 23 weeks (soon after development of symptoms) but recovery was only partial if doxycycline was reintroduced after 27 weeks. In addition to cells already known to be involved in disease progression, the researchers identified a cluster of microglial cells that, in transgenic mice, highly expressed the genes for proteins *Sdc4*, *Tgm2*, *Tlr2*, *Arg1* and inflammatory cytokines involved in TNF signaling, NF-kappa B signaling, cytokine-cytokine receptor interaction, Toll-like receptor signaling, and chemokine signaling. A successful mouse model means more research can be performed in the absence of many human patients, and the newly identified factors in disease progression may provide future therapeutic avenues.

PARKINSON'S DISEASE: Uncovering why nicotine may be protective

Abby Olsen, MD, PhD, University of Pittsburgh. "Nicotine-mediated Rescue Of Alpha-synuclein Toxicity Requires Synaptic Vesicle Glycoprotein 2c."

Parkinson's disease is caused by a combination of genetic and environmental factors, one curious example being that people who smoke appear to be less likely to develop Parkinson's. This may be due to interactions between nicotine and genetic variations in synaptic-vesicle glycoprotein 2C (SV2C). Parkinson's is characterized by aggregation of α -synuclein protein and loss of dopaminergic neurons in the part of the brain known as the substantia nigra. Researchers were able to duplicate Parkinson's symptoms in *Drosophila* flies engineered to express human α -synuclein, and found that dosing them with nicotine improved their locomotion and dopamine receptor counts and reduced α -synuclein aggregation. However, this was only true for flies that expressed the fruit fly equivalent of SV2C—other flies showed no improvement. So far, human trials of nicotine as a therapeutic or preventative for Parkinson's show little success. This study suggests that the benefit of nicotine may apply to those with a specific genetic makeup, which could enable more targeted studies or more focused therapies. In addition, creation of a successful *Drosophila* model will allow researchers to study the mechanics of gene-environment interactions in Parkinson's disease in more depth.

VERTIGO: Remote diagnosis by experts using video-oculography can help rule out benign causes of dizziness and vertigo

Shervin Badihian, MD, Johns Hopkins University School of Medicine. "Remote Expert Diagnosis by Video-Oculography is More Accurate Than In-Person ED Diagnosis in Acute Vertigo and Dizziness—Preliminary Results of the AVERT Trial."

Vertigo can be due to relatively benign conditions like vestibular neuritis or benign paroxysmal positional vertigo (BPPV), but it can also be a symptom of dangerous conditions such as stroke. Being able to accurately diagnose the less dangerous causes in the emergency room may reduce the need for cost-intensive diagnostics like MRI, while helping identify patients who need more careful monitoring and follow-up tests. Video-oculography (VOG), which uses video for detailed

measurement of eye movement, is commonly used for diagnosis in specialty clinics but not in emergency departments; in connection with expert telehealth consultations, it could allow for better diagnosis of vertigo in EDs. In this study, an expert oto-neurologist reviewed VOG recordings taken from 130 vertigo patients at tertiary-care emergency departments in combination with a brief summary from the ED clinician. The expert consult using VOG was much more accurate than emergency department assessments at diagnosing BPPV (69.8% accuracy vs. 9.3% accuracy) and vestibular neuritis (83.3% accuracy vs. 20.8% accuracy). Although this assessment was not significantly more accurate in detecting stroke, likely due to small numbers, it could prove to be a valuable tool for patient triage in cases of vertigo in the emergency room.

ALZHEIMER'S DISEASE: Making connections between air pollution and neurodegeneration

Hendrik Greve, PhD, Indiana University School of Medicine. "Ozone Air Pollution and Alzheimer's Disease: An Emerging Role for HMGB1 in the Lung-Brain Axis."

Exposure to urban air pollutants such as ozone (O₃) is increasingly linked with Alzheimer's disease; yet because ozone cannot travel from the lungs to the brain, the mechanism by which it contributes to development of Alzheimer's has been poorly understood. The hypothesis is that an immune response in the lungs leads to downstream changes in gene expression in brain cells. In a mouse model of Alzheimer's disease, mice exposed to high levels of ozone over time showed increased levels of the protein HMGB1 in the blood, and their brains showed increases in deposition of disease-associated amyloid plaques, decreases in plaque-associated microglial cells, and increased expression of genes responsible for neuroinflammation. Further study revealed that HMGB1 in the blood is responsible for regulating the neuroimmune response that mediates development of Alzheimer's disease. Although there was no evidence that lung tissue inflammation was involved, this is a key piece in the puzzle demonstrating that the peripheral immune system plays a vital role in the brain's immune responses and is likely the avenue by which ozone exposure leads to risk factors for Alzheimer's disease.

*Note that only first authors are listed above.

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About the American Neurological Association (ANA)

From advances in stroke and dementia to movement disorders and epilepsy, the [American Neurological Association](#) has been the vanguard of research since 1875 as the premier professional society of academic neurologists and neuroscientists devoted to understanding and treating diseases of the nervous system. Its monthly *Annals of Neurology* is among the world's most prestigious medical journals, and the ANA's *Annals of Clinical and Translational Neurology* is an online-only, open access journal providing rapid dissemination of high-quality, peer-reviewed research related to all areas of neurology. The acclaimed [ANA Annual Meeting](#) draws faculty and trainees from the top academic departments across the U.S. and abroad for groundbreaking research, networking, and career development. For more information, visit www.myana.org or @TheNewANA1.

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